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A TEXT-BOOK

OF THE

PRACTICE OF MEDICINE.

FOR STUDENTS AND PRACTITIONERS.

BY

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"A TEXT-BOOK OF PRACTICAL DIAGNOSIS."

SECOND EDITION, REVISED AND ENLARGED.

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PREFACE TO THE SECOND EDITION.

In presenting the second edition of his text-book on the Practice of Medicine the author desires to express his appreciation of the cordial reception of the work since it first appeared, a reception which necessitated its being repeatedly put to press.

The present volume, prepared for the physician and student of medicine, embodies the experience of more than twenty-two years of active hospital and private practice, during which time the author has been constantly teaching the subjects of clinical medicine and therapeutics. With this experience he has attempted to present the facts which the practitioner needs and which the student must thoroughly grasp if he is to be successful in gaining his degree and in practising his art.

In the preparation of many portions of the work careful collections of statistics have been made, and these have not infrequently given results which add materially to our conception of the frequency of certain diseases at different periods of life, of the relative frequency of different symptoms, and the value of certain plans of treatment.

Particular pains have been taken to present methods of treatment clearly and in such a way that they may be put in practice.

Much information that might be included, which deals with subjects which are still uncertain and debatable, has been excluded.

Warm thanks are due to my colleague, Dr. W. M. L. Coplin, Professor of Pathology in the Jefferson Medical College, for valuable suggestions and criticisms, in which he has shown not only a complete grasp of his own department of medical study, but intimate knowledge concerning the latest developments in clinical medicine.

The author also desires to acknowledge the valuable suggestions, made during the preparation of this edition, of Dr. Aller G. Ellis, Demonstrator of Pathology, and Dr. Alfred Gordon, Instructor in Neurology in the Jefferson Medical College.

The fact that the United States has, within the last few years, become possessed of territory in the tropics has greatly increased our interest in the many tropical diseases heretofore scarcely known by practitioners in the temperate zone, and the investigations by surgeons in the Army, Navy, and

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the Public Health and Marine Hospital Service have thrown much light upon these affections. Further than this, the investigations by English physicians have broadened our views very greatly as to tropical disease. As troops returning from tropical service often bring with them manifestations of these diseases, it behooves every practitioner to be able to recognize and treat such conditions. It seems appropriate, therefore, that a modern work on medicine should contain chapters on tropical medicine, the more so as lectures upon this subject are now given in many of the great medical schools.

In the preparation of this second edition great care has been taken to embody the latest views that have received general acceptance as being scientifically and clinically correct, it being constantly borne in mind that the needs of the student and practitioner will be best met by excluding statements open to criticism and presenting facts which will prove useful in practice.

H. A. H.

SPRUCE AND EIGHTEENTH STREETS,
PHILADELPHIA, January, 1907.

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PRACTICE OF MEDICINE.

DISEASES DUE TO A SPECIFIC INFECTION.

TYPHOID FEVER.

Definition.—Typhoid or Enteric fever, sometimes called Autumnal or Gastric fever, is an acute infectious disease due to the entrance into the body of a susceptible individual of the specific bacillus of Eberth, commonly known as *Bacillus typhosus*. The entrance of this organism into the system results, after a period of from one to three weeks in some persons, but not in all, in the development of fever, anorexia, headache, mental heaviness, and more or less severe pain in the bowels, back, and limbs. The tongue is coated, and the bowels are loose or constipated. With these symptoms are developed enlargement of the liver and spleen, and swellings and ulceration of the lymphoid structures of the small and large intestines, and a rose rash on the skin.

History.—Typhoid fever for many years was confused with typhus fever and malarial fever, and its very name means "like typhus." In 1813 it was considered as a separate disease, but this separation was not generally known by the profession until Louis, of Paris (1829), first emphasized a number of its cardinal points. Not until 1837 was the identification complete, when Gerhard, of Philadelphia, published results achieved under the guidance of Louis which proved the malady to be a distinct entity. More than forty years later (1880) Eberth isolated the specific bacillus and proved it to be the sole cause of the disease. Up to that time various causes had been thought to exist, but it had been recognized for many years as a "filth disease," and, therefore, preventable to some degree.

Distribution.—Enteric fever is one of the diseases which may be said to be pandemic, since it is found with some degree of constancy all over the world, its prevalence depending upon the introduction into the body of the specific bacillus usually with water or food.

Etiology.—The cause of this disease, as just stated, is the specific bacillus of Eberth, a short, thick, actively motile bacillus, with rounded ends and flagella, growing readily in ordinary suitable media. It is killed by exposure to 60° C. (140° F.), but it can withstand a freezing temperature for many days. Exposed to sunlight it is slowly killed, but drying, except in very thin layers, does not destroy it. It remains alive for months, and even for years, in clothing and in soil, if the conditions are favorable. It is readily destroyed by the stronger germicides, such as carbolic acid (1:200) and bichloride of mercury (1:2000). The bacillus of Eberth bears a close resemblance to the *Bacillus coli communis*, which is always present in the intestine, and to the so-called paracolon bacillus and the *Bacillus dysenteriæ*.

The second etiological factor in the development of the disease is the mode by which the bacillus gains access to the body. Almost invariably this access is through the mouth, stomach, and intestine, more rarely by inhalation of the bacillus in dust by the lungs. Infection takes place by the mouth in a host of ways, as by infected water, or milk diluted with infected water, or chilled by infected ice; by vegetables and oysters and clams, which, when eaten raw, are often the means of carrying infection. It has recently been proved at Ogdensburg, New York, that infected ice may transmit the organism after it has been stored in an ice-house for at least nine months. In still other instances persons nursing cases of this disease get the finger-tips infected and so, on putting the fingers to the mouth, introduce the organism into the body. Again, it has been proved beyond doubt that flies after lighting upon the discharges of a case of typhoid fever may carry the bacillus to otherwise pure food, and so spread the infection as long as twenty-three days after feeding on infected stools (Ficher). Stokes describes an epidemic in a factory employing 1500 women and 400 men. As many as 200 of the women were ill at one time with typhoid fever, but none of the men fell ill. All the men drank beer at luncheon, whereas all the women used milk. The milk was found to have been infected by flies from a neighboring privy. Cockroaches may also spread the bacillus.

Every great epidemic of the disease has been due to contamination of the water supply. In the Maidstone epidemic in England 1 person in every 17 in the town was infected; while in the Plymouth epidemic in Pennsylvania 1 in every 7 was stricken, for there were 1200 cases in a population of 8000. As only a part of these 8000 persons used the contaminated water, the proportion of actual infection to exposure was far higher than 1 in 7. The influence of a bad and good water supply is shown in Figs. 1, 2 and 3.

In 1888 the use of filtered drinking water was begun in the French army, as a result of which the morbidity of typhoid fever was diminished 49 per cent. in 1890, and the mortality 34 per cent.

Prevention.—From what has just been said it is evident that typhoid fever is an entirely preventable disease, provided that the bacilli as they escape in the feces, the urine, the sputum, and, perhaps, in the sweat, are destroyed as soon as they pass from the patient's body. The destruction of the discharges and so of the bacilli is therefore absolutely essential, and in addition careful antisepsis on the part of the attendant as to personal cleanliness and the protection of the discharges from flies are to be enforced. As careless or ignorant persons do not disinfect the stools, the additional measures of prophylaxis are the boiling of all water that is to be placed in the mouth, and the use of nothing but well-cooked foods, which have not been exposed to flies or dust after cooking. The vessel which receives the discharges of the patient should contain carbolic acid (1:200), corrosive sublimate (1:2000), or chlorinated lime (a heaping teaspoonful to the pint). Formaldehyde solution (40 per cent.) may also be used. If the stool is formed, it should be broken up by stirring it with a rod, so as to expose all the fecal matter to the germicide.

Fig. 1

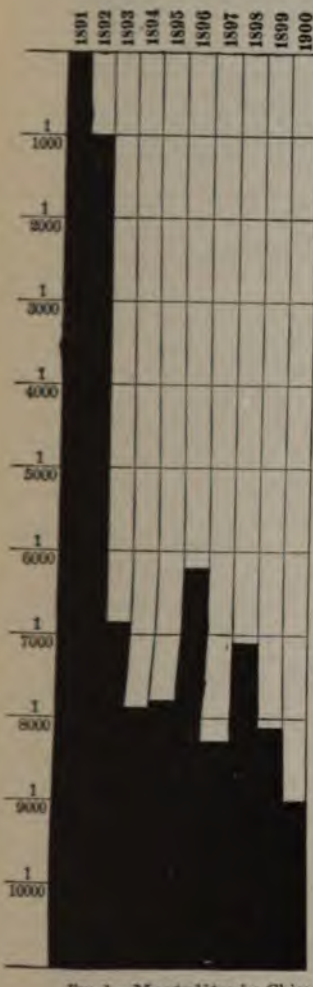

Fig. 2

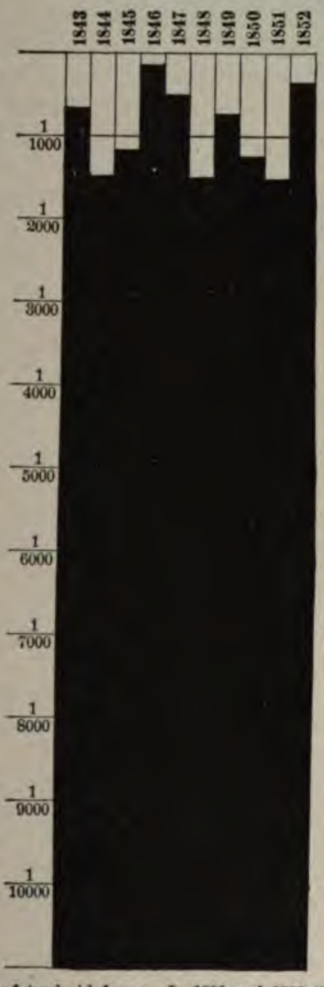

Fig. 3

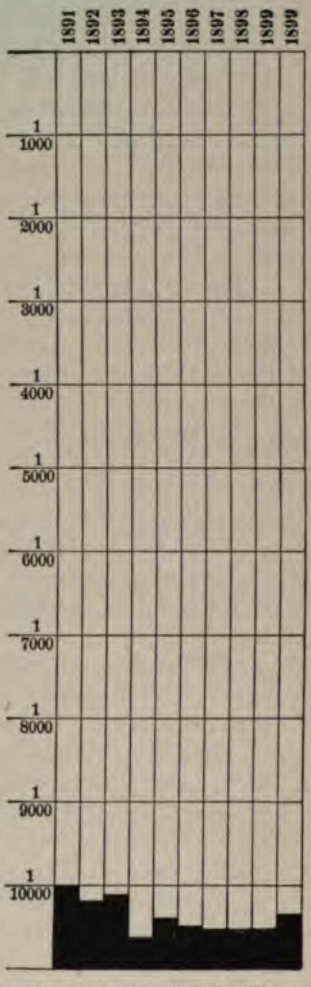

Fig. 1.—Mortality in Chicago of typhoid fever. In 1891 and 1892 the water was contaminated with sewage and the death rate was about 1 to 450 to 1 to 1500. With a change in water supply the mortality has fallen to 1 to 6000 or even 1 to 9000. (Seibert.)

Fig. 2.—Mortality of typhoid fever in Berlin before the supply of drinking water was filtered. In the decade 1843 to 1853 the average yearly mortality was 1 per 900 of inhabitants.

Fig. 3.—Mortality of typhoid fever in Berlin after water was filtered. (Seibert.)

Physicians and nurses are not careful enough about the destruction of the stools, and the average individual is willing to take his chances on the use of unboiled water. Another of the difficulties is that patients may, when no longer kept in the house by the disease, continue to cast off bacilli in the urine or feces which are capable of infecting water supplies. This danger is of great importance, because at each urination or defecation the convalescent patient may produce a new source of infection. Further, it is toward the close of the attack and during convalescence that the urine contains these specific organisms in pure culture and in enormous numbers, and they may remain persistently present, not only for days but for months. The patient should be told of this danger, should be directed to disinfect his discharges, and should receive daily doses of uritone or urotropin to destroy the bacilli in the urine before they are passed in that fluid. If he is also informed that bacilluria is a danger to himself, in that it may result in secondary diseases in his genito-urinary tract, he may be interested enough to aid the physician in arresting the spread of the bacillus by adhering to a plan of careful medication.

All clothing, instruments, bedding, pillows, utensils, bath-tubs and ordinary wash-tubs, which may be contaminated by the discharges of a patient, should be disinfected thoroughly as soon as their function is performed. The hands of the nurses should be repeatedly disinfected.

Another preventive of typhoid fever consists in the injection, or inoculation, of the individual with bouillon containing the toxins of *Bacillus typhosus*, the organisms themselves being destroyed beforehand by heat. Such an injection produces local swelling and some pain, a sense of nausea and depression, and some febrile movement, which symptoms speedily disappear, the patient at the end of twenty-four to thirty-six hours being well again. A fortnight later the individual is injected a second time. It is interesting to note that these injections increase the bacteriolytic power of the blood, cause in some degree the so-called Widal reaction, or agglutination of the *Bacillus typhosus*, to take place when the serum of the injected person is used for this test, and in many hundred cases have probably served to act as a protective agent against infection, although the protection is by no means so complete as that afforded by vaccination against smallpox, nor has it been tried sufficiently widely to place its use upon a similar clinical basis. It has, however, had complete tests in the last few years. Thus, of 29,650 individuals in South Africa, Egypt, Cyprus, India, and Ireland, who were similarly exposed to infection by typhoid fever, 7055 received preventive inoculations. Of this number 333, or 4.72 per cent., contracted the disease; 34, or 1.03 per cent., died. Of the 22,595 who were not inoculated, 2763, or 12.22 per cent., contracted the disease; 507, or 2.7 per cent., died.[1]

It has been shown in the United States Government Laboratories at Washington and in the City Laboratory of Philadelphia that the introduction of so small an amount of sulphate of copper as 1 : 1,000,000, or even 1 : 4,000,000, will destroy the typhoid bacillus in a very few hours, and already this means has been successfully used in large reservoirs for the purification of the water supply of towns. It is said to be efficient, is very cheap, and entirely harmless to human beings who drink the water and to the fish in the water. How the copper acts is not known. If water containing typhoid bacilli is placed in burnished copper vessels for a few hours most of the typhoid germs are also destroyed. More recently, Gage and Clark have thrown doubt on this method, and its value must be considered *sub judice*.

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[1] These statistics are compiled from the reports of Wright, Leishman, Luck, Fawcett, and Burt.

Frequency.—Typhoid fever affects males oftener than females, and occurs most frequently between fifteen and thirty years of age. It may, however, affect infants or aged persons. It occurs more frequently in August, September, and October than any other quarter of the year, but is by no means limited to this period. (See Fig. 4.)

FIG. 4

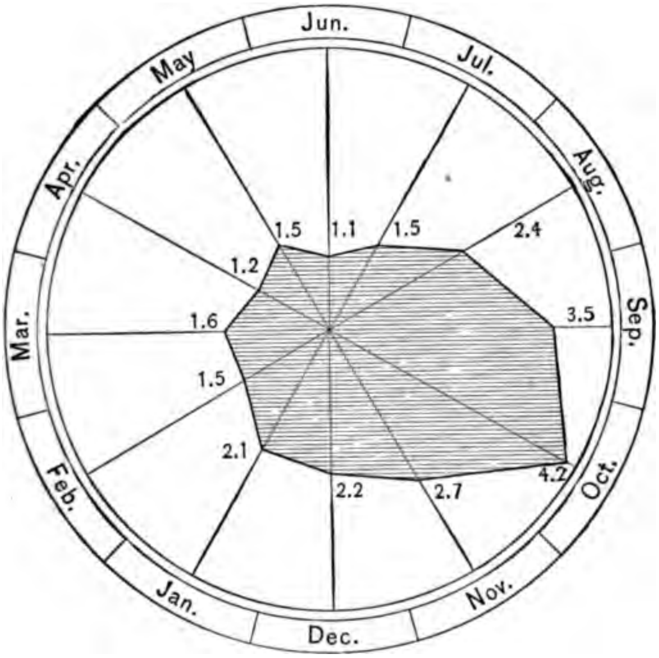
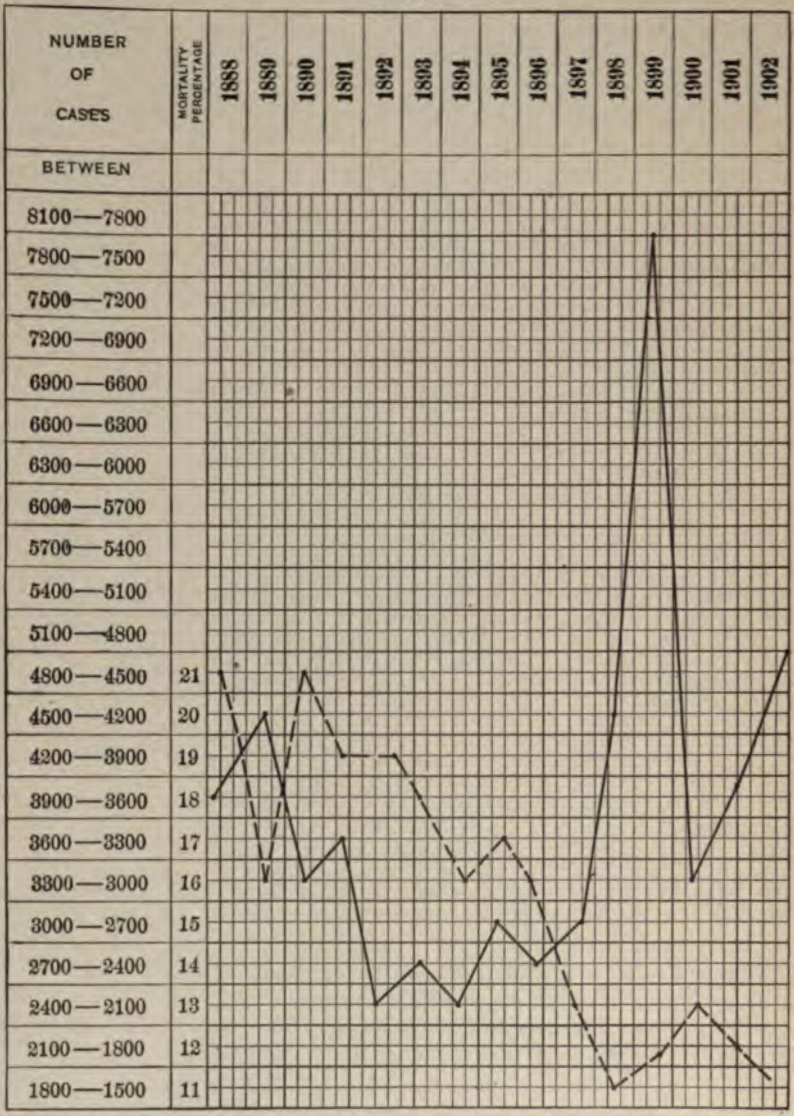


Chart from the United States census, showing the period of the year when the mortality from typhoid fever reaches its maximum.

Typhoid fever is becoming less and less frequent, and less severe all over the world. In Munich the mortality in the decade from 1851 to 1861 ranged from 123 in 100,000 inhabitants to 453 in 100,000 inhabitants, whereas in the years from 1890 to 1897 the mortality was from 57 or 14.8 in 100,000 people to 10 or 2.5 per 100,000; in Vienna it has fallen from 120 per 100,000 to 10 per 100,000; in Dantzig, from 100 per 100,000 to 10.5. In Massachusetts the mortality from typhoid fever in 1901 was less than in any year since 1842. In 33 cities in that State it was only one-fourth of what it was thirty years ago. In Philadelphia a similar decrease is seen in both mortality and morbidity. (See Fig. 5.) The rise in morbidity in 1898 is due to the soldiers returning from the Spanish-American war, for the careless sanitation of large camps always makes it epidemic. Thus, in this war about 1 in every 5 of the soldiers in the United States army became infected, and nearly 87 per cent. of all the deaths in the army

FIG. 5



Showing the decreasing mortality of typhoid fever in Philadelphia. Solid line, morbidity with sharp rise in 1898-99, due to returned soldiers of Spanish war. Broken line, mortality.

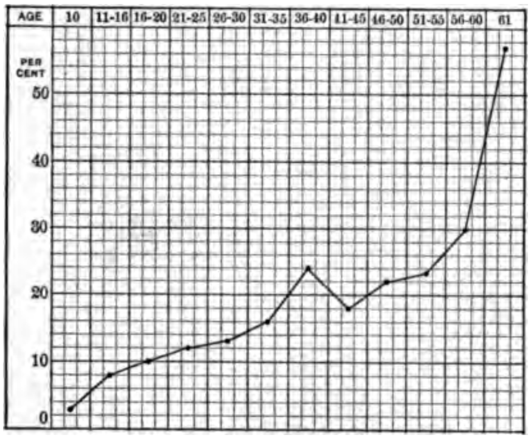
were due to this cause. In Melbourne, Australia, there has also been a decrease in the mortality rate which is very noticeable, being over 50 per cent. This decrease is due chiefly to care in regard to water supplies.

The general mortality rate of the world may be said fifty years ago to have been almost universally 25 per cent., whereas it is now from 15 to 10 per cent.

With advancing years of age the morbidity decreases, but the mortality greatly increases. (See Fig. 6.)

Pathology and Morbid Anatomy.—In studying the morbid anatomy of typhoid fever it must be remembered that it is not, when fully developed, a local infection, restricted to one or more foci from which the *Bacillus typhosus* distributes its toxin through the body. On the contrary, the typhoid infection is practically universal, and the bacillus may be found in varying numbers in every organ of the body, including the bone-marrow and skin. Contrary to general belief, they may not be demonstrable in the intestinal contents in large numbers until the disease is well advanced, and their presence in the stools depends largely upon the intensity of the changes which take place in the intestinal glands. It is true, however, that the agminated glands (Peyer's patches) and the solitary glands of the small bowel are the parts of the body which usually are the seat of the most evident and constant lesions. On the other hand, it is not to be forgotten that cases of undoubted typhoid fever occasionally occur in which no ulceration of the intestinal mucosa takes place.

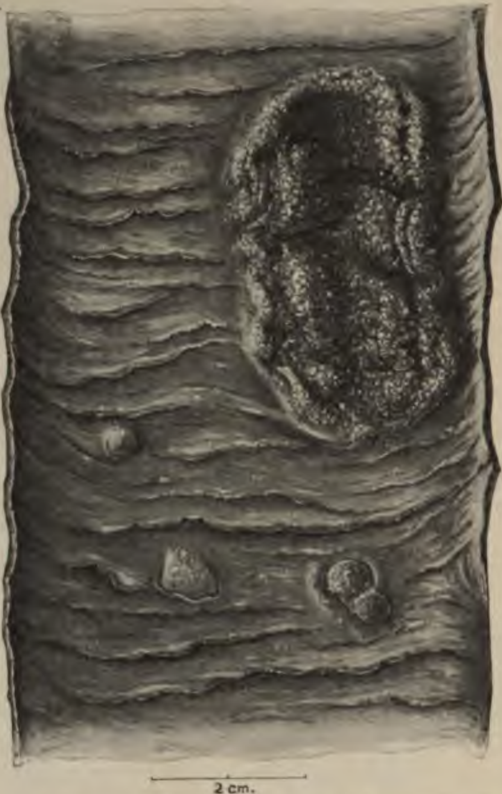
FIG. 6



Showing the increased mortality of typhoid fever with age. (Curschmann.)

The alterations from the normal in the bowel may be discussed under three heads: (1) a diffuse catarrhal inflammation of the intestinal mucosa of varying severity, but usually resulting in desquamation of epithelium; (2) hyperæmia, swelling, endothelial hyperplasia, necrosis, and finally ulceration of the agminated glands or Peyer's patches; and (3) a similar change in the so-called solitary lymph follicles of the intestine, although the changes in the agminated glands are distinctly the more conspicuous. These changes begin in the very earliest stages of onset, and do not wait until the symptoms of the disease are well developed. If by some accident the patient comes to autopsy at this time, the intestinal mucosa will not only be found inflamed, but in addition the lymphoid structures just named will also be found swollen and reddened by hyperæmia. Their edges are not well defined, and the entire gland is hyperplastic and spongy. A little later in the progress of the disease these areas become less red in hue and begin to look somewhat gray in color; they are firmer and project above the surrounding mucous membrane to a marked degree, so that they extend well into the lumen of the bowel.

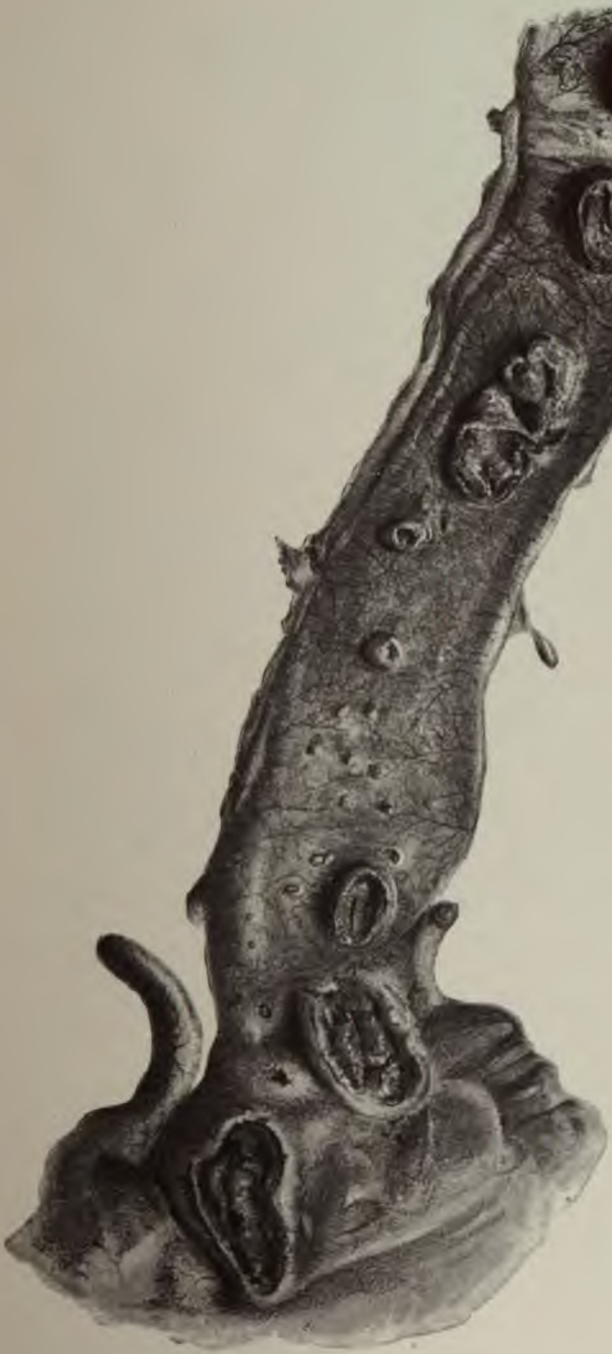
FIG. 7



Ulceration of a Peyer patch in typhoid fever, with associated swelling of solitary glands.

Sometimes the hyperplasia within the gland is so great that its edges overhang the surrounding tissue. Still later the lymphatic tissue may become so infiltrated that neighboring patches of glandular tissue coalesce, and when they reach the stage of necrosis result in large areas of slough, so that the entire thickness of the intestinal wall may be involved (Fig. 7). The presence of so severe an ulcerative process naturally results in deep infiltration of the bowel wall, and the inflammatory condition may extend to the peritoneal coat, so that this serous membrane is reddened or even grayish

PLATE I.



Showing Typhoid Ulcers in Small Bowel and near the Appendix. (Kast and Rumpler.)

from exuded lymph. The severest forms of ulceration usually take place in the lower part of the ileum.

While ulceration of the tissues composing Peyer's patches is the usual result of this infection, necrosis does not always ensue. The gland may become red and swollen and the inflammatory process go no farther, proceeding from this state to that of resolution and healing. Not infrequently this agminated patch is not equally affected in all its parts, and this gives it an uneven appearance, which is emphasized when the portions which are most affected ulcerate, so that small ulcers are dotted over the surface of the swelling, which, if the process is severe, finally coalesce. In severe types of the disease the process is so well diffused that a huge slough forms which, when it drops off, leaves a swollen, ulcerated surface, the excavation being usually very deep. It is this type of necrosis that results in perforation, the opening in the bowel wall being usually found at a point directly opposite the mesenteric attachment. Rarely the perforation takes place between the layers of the mesentery and causes a retroperitoneal abscess. Harte states that in 140 cases out of 190 the perforation occurred in the small bowel within twelve inches of the cæcum. If the patient survives the severer periods of the disease, the swelling of the glandular tissue gradually diminishes, granulations develop, new connective tissue largely takes the place once occupied by the gland, and the ordinary intestinal epithelium covers the exposed area. While it is true that the solitary glands are rarely so markedly affected as the agminated glands, they may suffer much more severely and be found diseased over a larger area than are the glands of Peyer.

The number of ulcers in the bowel in typhoid fever varies greatly. Usually they are limited in number, but occasionally they cover very large areas. They may be more numerous in the cæcum than elsewhere in the colon. Out of 577 autopsies upon cases of this disease in Hamburg and in Leipzig, the cæcum was ulcerated in 510, or 88.39 per cent.; the cæcum and appendix in 247, or 42.81 per cent.; the colon in 184 cases, or 31.89 per cent.; the jejunum in 41 cases, or 7.10 per cent.; the rectum in 12 cases, or 2.08 per cent. The percentage of cæcal lesions, in these statistics of Curschmann, just given, is much higher than is generally noted; 40 per cent. is more nearly correct. As already stated, the lower part of the small bowel is the area chiefly affected.

Next to the changes in the intestine the most noteworthy alterations may be said to take place in the *lymph nodes of the mesentery*, which lie between the intestinal lesion and the general system. These tissues go through a similar process of hyperæmia, swelling, and endothelial proliferation, which usually falls short of extensive necrosis. Small necrotic patches are not infrequent. More rarely large foci of softening or even suppuration may occur in these nodes, and as recovery takes place small septic areas are gradually walled off by lymph, become encysted, or are absorbed. Rupture of enlarged mesenteric nodes has been observed.

The *spleen*, in addition to its swelling, which begins early and lasts for the first three weeks or more of the illness, is full and tense, and of a darker hue than normal. Later, as the attack wanes, it becomes soft and darker in hue. The splenic *blood sinuses* are distended by erythrocytes, the endothelial cells proliferate, and the pulp here and there becomes the seat of small areas of coagulation necrosis. The splenic lesions may also consist in infarction and rupture, but the latter accident is very rare.

Until a few years ago the presence of the typhoid bacillus in the *blood* was unknown, but we now know that this organism is present in this part of the body with great constancy during an attack of typhoid fever. It is usually present as early as the fifth day and persists until the close of the third week, or even longer than this. Rosenberger has recently collected 535 cases of typhoid fever in which the blood was examined for the bacillus. It was found in 80 per cent. of these cases. The examination of the blood has therefore become of great importance in the early diagnosis of the disease. (See Diagnosis.) The bacillus probably gains access to the blood through the mesenteric glands.

The *liver* is usually somewhat swollen, but the changes in its appearance are not peculiar to this disease. The hepatic cells manifest more or less cloudy swelling, and areas of coagulation necrosis containing endothelial cells are present. The cells lining the bile-ducts may be swollen, granular, and, in some cases, undergo a process of desquamation. Abscess of the liver may develop or gallstones may by their presence aid in the production of a cholecystitis, but more commonly typhoid fever probably induces gallstones. (See Complications.)

The *heart muscle* nearly always suffers from typhoid infection in direct proportion to the severity of the toxæmia present. The myocardium is granular and may suffer from fatty or hyaline changes. Very rarely the endocardium becomes affected and the specific bacillus has been obtained from vegetations on the valves.

The *kidneys* show no typical changes. They usually show cloudy swelling, and even an acute nephritis may be present. Sometimes as the result of a terminal infection multiple abscesses may form in the kidneys and a croupous exudate in the pelvis of these organs may develop.

Reference is made elsewhere to the *lesions of the respiratory tract* which may complicate the course of the malady, such as laryngeal perichondritis, ulcerative laryngitis, hypostatic congestion, pneumonia in both its forms, pulmonary infarction, simple pleurisy, and empyema. (See Complications.)

An *endarteritis* (which may be a thromboendarteritis) has been shown to occur in a small percentage of cases, and it is reasonable to assume that the thrombotic processes occasionally observed in the veins depend upon a similar involvement of the lining membrane of these vessels.

Longcope has shown that the lesions in the bone-marrow closely resemble the changes in the lymphoid tissues of the mesentery and of the bowel. There are present many lymphoid cells, large phagocytes and foci of necrosis.

A very important factor to be recalled in the study of the pathology of typhoid fever is the presence of additional infecting micro-organisms which aid the *Bacillus typhosus* in producing severe lesions and often are equally responsible for a fatal termination. This view is, however, based on post-mortem findings, and is not supported by the results of ante-mortem examinations of the blood, for of 150 cases of typhoid fever in which the blood was examined during life Cole found but one in which mixed infection was present; the case was one of staphylococcæmia, with multiple boils, and terminated in death.

Incubation.—The period of incubation of the infection by typhoid fever is generally stated to be from one to three weeks. That the period of incubation may be much shorter than this would seem to be proved by the case reported by Duflocq and Voisin of a girl nineteen years of age who deliberately swallowed a virulent culture of the typhoid bacillus with the intention of committing suicide. She began to feel ill on the third day, had fever on the fourth day, rose spots on the fifth day, and the Widal reaction appeared on the sixth day.

Symptoms.—Typhoid fever usually begins with a sense of wretchedness and general illness, no particular symptom being especially well marked, unless it be more or less severe *frontal headache* and aching in the back and limbs. The *facial expression* very early in typhoid fever usually becomes listless and later stupid and heavy, and the patient is often a little deaf because his mental state is benumbed rather than because there is any actual trouble with the auditory apparatus. Not infrequently there may be a considerable amount of cough without expectoration, and there may be exaggeration of the sounds of bronchial breathing on auscultation.

The *tongue* is somewhat coated, and very early its edges become clean and red, while the central coating remains. This appearance of the tongue is very characteristic, even in mild cases.

Headache, thirst, and *sleeplessness* are usually prominent symptoms during the first week. A *mild fever* develops simultaneously and *nose-bleed* may occur repeatedly. Usually the liver and spleen become swollen toward the end of the first week, and the belly becomes somewhat tumid and tender.

The characteristic *enlargement of the spleen* in enteric fever may be undemonstrable because of the distention of the stomach and intestine with gas; but while the presence of an enlarged spleen is of some importance in reaching a diagnosis of typhoid fever, inability to discover any increase in its size does not negative the diagnosis of typhoid fever in any degree.

An undue amount of *gurgling* can be felt and heard in the right iliac fossa.

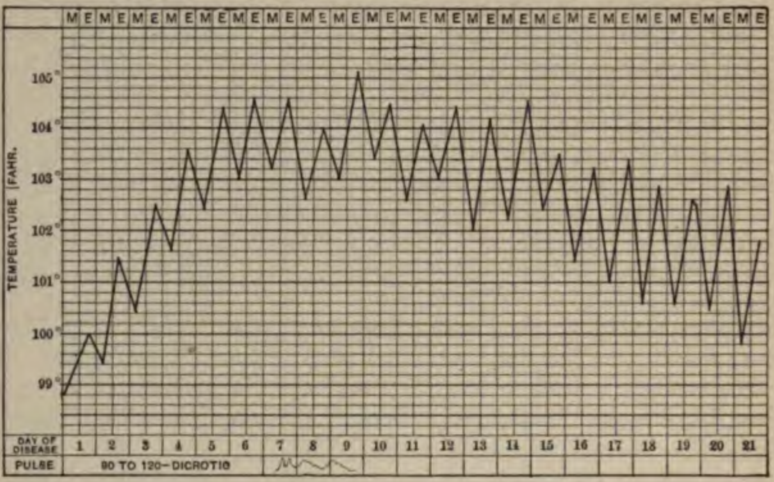
Constipation is usual in the first week, but *diarrhœa* may be marked, and if loose the stools may be brownish, but later resemble okra-soup or pea-soup.

The *temperature* in typhoid fever during the first week rises step by step. Each morning it is higher than on the previous morning, and each evening higher than on the night before, although the morning temperature is often lower than that of the preceding evening. (See Fig. 8.) Usually by the end of this week it reaches in the morning 102° or 103°, and at night 103° to 104°, and remains at this level until the fourteenth or twenty-first day.

The *pulse* is more *rapid* than normal, ranging from 90 to 100 beats per minute, and it is usually soft and compressible; the pulse of debility, not of vigor. Often the pulse is a little dicrotic. As the disease progresses the pulse rate usually increases to about 110, but the pulse force distinctly diminishes.

At about the *seventh* to the *ninth day* a very important diagnostic sign first makes its appearance, namely, the so-called *rose spots*, which usually develop on the skin of the abdomen and chest, sometimes on the back, and more rarely on the limbs. These spots are small, faint macules, usually scanty in number, which lose their color momentarily when pressed upon or when the skin on which they exist is stretched between the finger and thumb of the physician. As a rule these spots are isolated, but very rarely they may be so profuse as to produce the appearance of an ordinary rash.

Fig. 8



Course of typhoid fever. (Modified from Musser.)

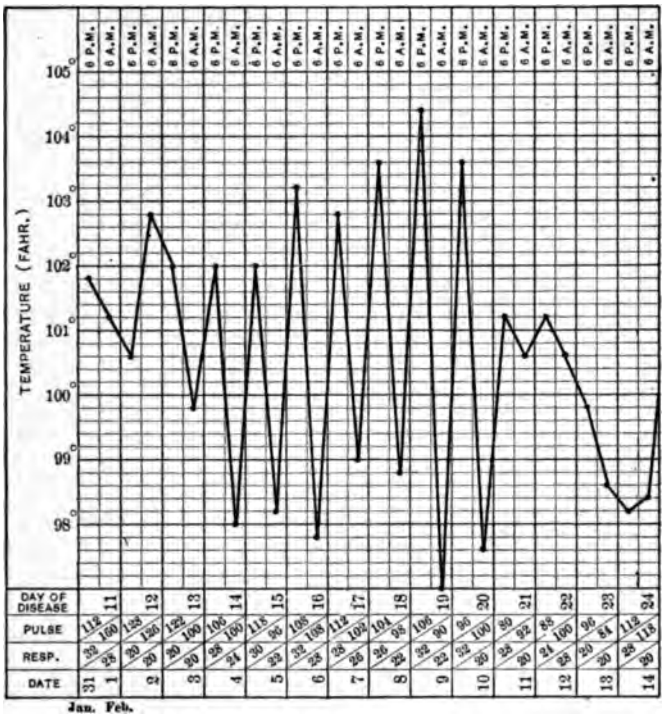
The tongue becomes dry and it may be fissured, the mental stupor increases, diarrhœa is also active, and the moderate tympanites of the earlier days becomes more marked. If the patient has received little care, or if the case is essentially severe, his condition is manifestly one of profound toxæmia, and by the *end of the second week* he is evidently at the very acme of his infection. Death not infrequently takes place during this period as a result of profound toxæmia, hemorrhage, perforation, or pulmonary complications.

Because of the toxæmia *delirium* may be marked, and it is usually of the low muttering type, the patient seems to be in a semi-stupor, the teeth are covered with sordes, and the tongue is foul and dry.

These symptoms gradually carry the patient into his *third week*, with increasing *diarrhœa, greater tympanites, deeper stupor*, and more manifest signs of *profound toxæmia*, with muscular tremors or true *subsultus tendinum*. *Emaciation* by this time is marked and the skin dry and harsh. The heart is feeble, its sounds distant and muffled, and myocardial degeneration is manifestly advanced. To the possibility of the appearance of the fatal complications named in the succeeding pages as appearing at the end of the second week are added at this time still greater danger of pulmonary hypostatic congestion and pneumonia. The patient may be so profoundly poisoned by the toxic products of the disease that he seems almost moribund.

If the pathological process is not so severe that recovery is impossible, the first sign of the ending of the malady may develop at any time between the *fourteenth* and *twenty-eighth day*, according to the severity of the disease. This consists in a slight modification of the temperature range and the development of a low morning temperature with a well-maintained high evening temperature, so that the daily range may amount to from 2° to 3°. This is called the "*period of steep curves,*" and the appearance of these steep curves at this time in the course of the disease is usually a promise of approaching convalescence. An equally good description of this period is that of Murchison, who called it the "stage of changing fortunes," or that of Wunderlich, who described it as the "period of ambiguity."

FIG. 9



Part of a chart showing the period of steep curves from the fourteenth to the twentieth day of an attack of typhoid fever.

The last stage of the acute febrile period having been reached, the temperature falls to normal during the next few days by lysis, and then may be subnormal until convalescence is well established, the patient being wasted and feeble, but usually ravenously hungry.

ATYPICAL FORMS.—While the train of symptoms just described may be considered typical of an attack of typhoid fever occurring under conditions favorable for its full development, it is often so modified by various causes that a large proportion of cases do not present many of the most prominent and diagnostic symptoms, but, in their place, manifestations so at variance with those of ordinary cases as to greatly perplex the physician. Thus, very marked variations in onset may occur and completely mislead the medical attendant if he be not on his guard. In some cases instead of manifesting itself gradually the disease has a *sudden onset* with a *sharp chill* followed, it may be, by a profuse sweat and a continued fever. This variation is perhaps most apt to occur in children. Headache may be so severe in the beginning as to rouse the suspicion of meningeal inflammation, and active delirium may be an early symptom, being severe enough to be maniacal in type. In other instances a pneumonia is the earliest sign of the malady, while in still others a *severe choleraic diarrhœa* may begin the illness. It is also important to recall the fact that well-developed signs of *appendicitis* may appear, due to the swelling of the lymphoid tissues of the intestine and appendix by reason of the infection. This has frequently resulted in enthusiastic surgeons removing the appendix only to find it slightly diseased as part of the general lymphatic change, the speedy appearance of the rose rash and persistent temperature soon showing the true character of the case. Rarely a severe attack of vomiting begins the illness, and still more rarely *acute renal disease*, nephro-typhoid, or the *fièvre typhöide à forme rénale* of the French observers, develops.

Although *diarrhœa* was correctly considered at one time to be one of the most constant symptoms of enteric fever it is now absent in more than half the cases during the whole course of the disease, and *splenic enlargement* in many instances is too slight to be discovered, so that it is to be borne in mind that while these two symptoms possess a positive diagnostic value when present, their absence in no way contradicts the diagnosis of typhoid fever.

In other instances the course of the fever greatly varies from that just described. It may rise very abruptly, and it may end equally suddenly, the lysis being completed in twenty hours. Sometimes the morning temperature is the higher of the two, although this is rare. The regular course of the temperature may also be greatly altered by intercurrent chills. (See Chills.) Very rarely, strange as it may seem, no febrile movement is present at any time in the course of the malady.

The most important variations from what may be called the normal course of the temperature in the second and third week of this disease are those produced by free hemorrhage from an intestinal ulcer and by perforation of the bowel. A sudden fall of several degrees should always arouse suspicion of one of these accidents, for the drop in the fever may be noted before any of the other signs of hemorrhage or perforation manifest themselves.

Marked rises and falls of temperature are also often seen in patients who are markedly anæmic as the result of hemorrhage. Abortion also causes a marked fall of the fever.

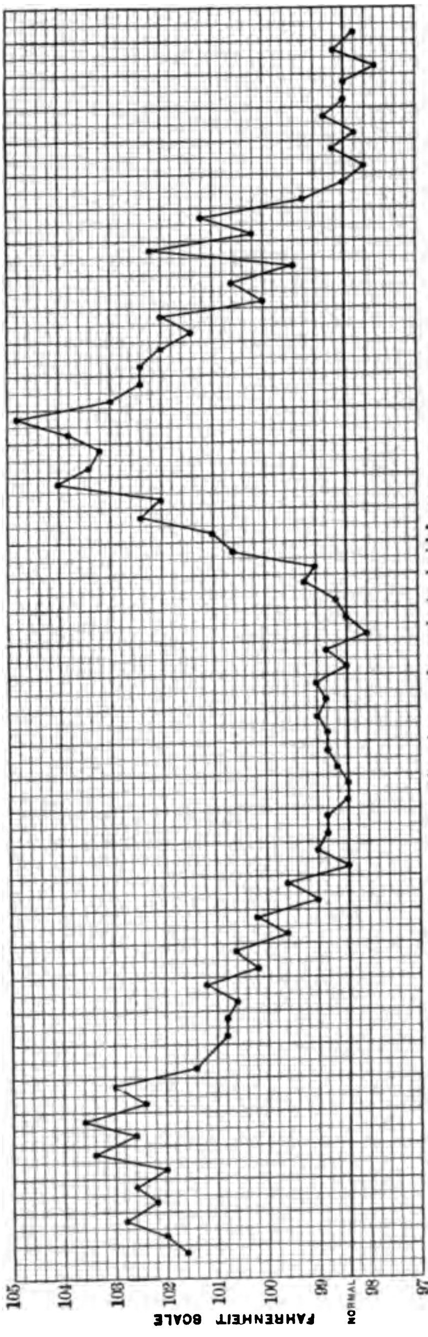


Fig. 10

Showing a relapse in typhoid fever.

The course of the temperature may resemble that of remittent malarial fever, and it has frequently misled physicians into the belief that malarial infection and not typhoid infection was present. (See Chills.) Infectious complications of the disease such as otitis media, phlebitis, furunculosis, meningitis, and erysipelas may also cause sudden variations in temperature. And in cases which have been gravely ill it not rarely happens that fever continues after the typhoid infection has run its course because of post-typhoidal septicæmia—that is, a multiple infection due to the presence of pyogenic organisms, which have found a favorable field for growth in a patient whose vitality has been impaired by the specific fever.

As the stage of convalescence approaches, or when it is reached, a sharp return of active febrile movement may come on for a day or two, the temperature being as high or higher than ever before. It then returns to its ordinary level. This is called a *recrudescence*, and possesses no grave significance. It often follows mental excitement and the taking of improper or too much food. When this rise of temperature persists, it usually is indicative of some complicating malady, or of a relapse called an "intercurrent relapse" if it takes place during the continuance of the primary febrile period. (See Fig. 10.) After the fever has disappeared there may be a prolonged continuance of a slight evening rise of temperature as the result of nervous irritability and anæmia, or it depends upon the abuse of strychnine, with the mistaken idea that it is a valuable heart tonic at this time. In other cases a subnormal temperature for the entire twenty-four hours may persist for days. This is of no importance save that it indicates that the patient is feeble and needs good feeding and fresh air. The other variations met with depend upon the age of the patient. Old persons often have an irregular febrile movement, and children may have marked rises and falls of temperature which do not necessarily indicate any complications.

Persistence of distinct febrile movement after the fourth week in any case of typhoid fever in which a relapse has not occurred nearly always means a complicating or secondary infection. The number of cases of rapid tuberculosis called typhoid fever, until the persistent loss of flesh and fever forces the correct diagnosis upon the physician, is by no means small. The possibility of ulcerative endocarditis, cholecystitis with ulceration, with or without impacted gallstones, and septic infection due to suppuration as causes of fever are to be borne in mind and their presence carefully looked for. (See Complications.)

That a patient with this disease may suffer not only from the infection due to the bacilli of Eberth, but from multiple infections by other organisms which aid in decreasing his vital resistance should be borne in mind.

Closely associated with the study of the temperature is that of *chills*. They may usher in an acute complicating inflammatory process, or be entirely without such significance. Sometimes they occur in cases which suffer from constipation, apparently as a result of the absorption of fecal poisons. (See Fig. 11.) In other cases they are due to a true coincident malarial infection, but it is a noteworthy fact that during the course of typhoid fever, even if the patient is also suffering from malarial infection, the latter usually remains in abeyance until the former has about run its course. It is better for the physician to regard such chills as being an indication of some acute complication than to consider them as malarial, unless he can prove the existence of the last possibility by finding malarial organisms in the blood.

The *skin* is sometimes covered by a *fugacious scarlatiniform* rash in the early stages. In certain cases it desquamates in large flakes or in fine, branny scales, the latter appearing oftenest in those who have been actively bathed and rubbed.

Very commonly if sweating takes place, *sudamina*, or tiny sweat drops retained beneath the superficial epiderm are found on the abdomen, chest, or limbs. Herpes about the mouth is very rare in typhoid fever, but it does occur, notwithstanding the denial of this fact by some observers.

Under the name of *tache bleuâtre* or peliomata, faint blue or steel-gray spots of fairly good size are sometimes met with. They are not due to the disease, but are found only in those who are infested with lice. The so-called *tache cerebrale* is not characteristic of this disease, but is sometimes seen during its course, and consists in a red line with white borders produced by drawing the finger-nail over the skin. It is probably due to vasomotor palsy of the cutaneous vessels.

Of the deeper lesions of the skin, we meet with *bed-sores*, which rarely occur in cases seen from the first and which receive proper care. They appear usually over the sacrum. Cases of *superficial gangrene* of the skin have been reported by Stahl and the author. *Erysipelas* occurs, usually of the

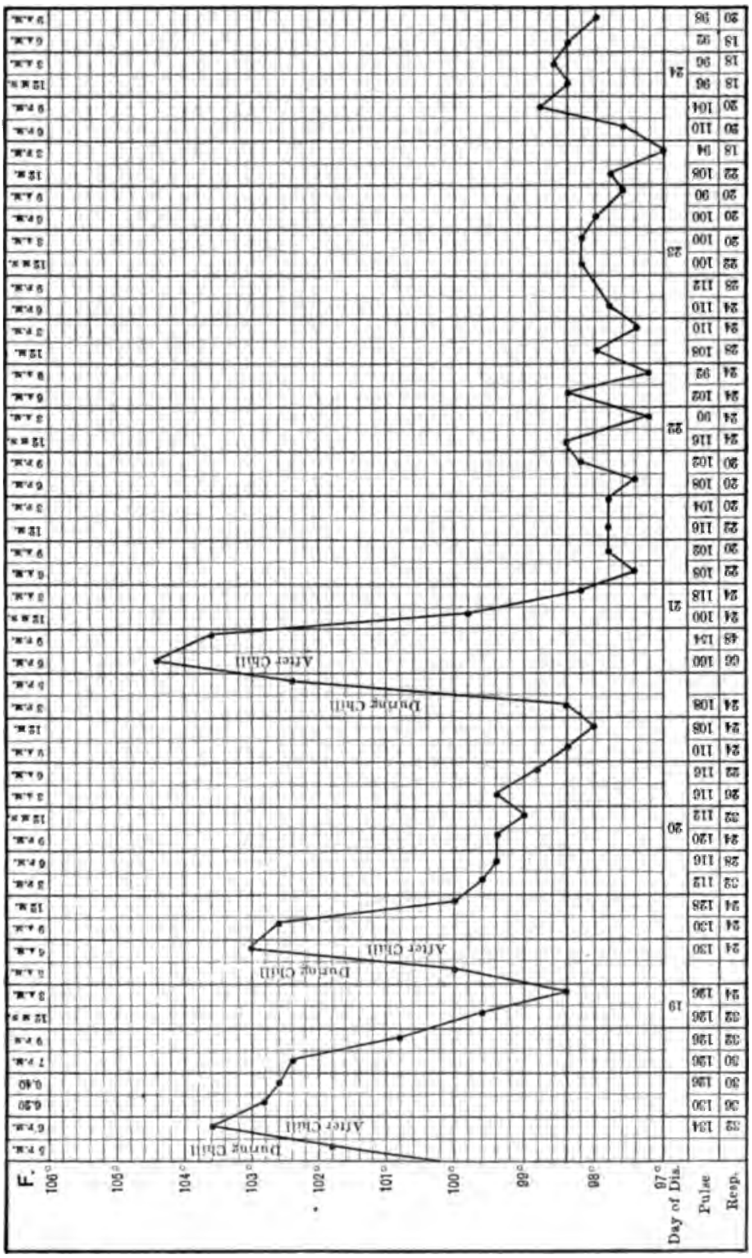


FIG. 11

Toward the end of this case of typhoid fever severe chills developed on the eighteenth, nineteenth, and twentieth days, and the fever ended by crisis.

face, by reason of infection through fissures in the buccal or nasal mucous membrane. Sometimes *erysipelas migrans* develops. In very malignant cases *petechiæ* may be present.

Patients suffering from typhoid fever rarely suffer from other eruptive diseases, but instances of scarlet fever, chicken-pox, and measles occurring as complications are on record. In women in particular the hair often falls out freely during or after an attack. Boils are by no means rare lesions, and even carbuncles may develop as a result of multiple infection.

The *blood* in typhoid fever suffers from an increasing degree of *anæmia* in respect to the number of the red cells and of their richness in hæmoglobin. Indeed, the color-index is more markedly lowered than the corpuscular count. When an inadequate supply of liquids has been allowed the resulting concentration of the blood may produce an apparent corpuscular richness not actually present.

The bacteriolytic power of the blood in severe cases is probably always diminished. The leukocytes are slightly decreased in number, the large mononuclear and transitional cells are relatively increased, and, according to Thayer, the polymorphonuclear cells are decreased. Cabot asserts that a leukocytosis, non-inflammatory in origin, sometimes occurs.

Complications and Sequelæ. CIRCULATORY COMPLICATIONS.—The *heart*, as already stated, is weakened, and if severely affected may develop embryocardia or fetal heart sounds. There are few, if any, diseases which do not have special predilection for the heart muscle or its valves, which so greatly interfere with this organ as does typhoid fever. A pulse rate above 125 is ominous, and one of 130 or 140 dangerous. The danger is usually in direct proportion to the feebleness of the first sound of the heart. When the cardiac sounds are those of the fœtus in utero (embryocardia), the prognosis is grave. A very rapid pulse and irritable cardiac action are sometimes seen in cases in which strychnine has been used to excess with the idea that it is a stimulant. A soft systolic murmur is occasionally audible, which may be hæmic in origin or due to relative insufficiency of the mitral valves. Rarely it may be due to endocarditis or pericarditis, but pericarditis is a very rare complication of typhoid fever. Gaudy and Gourand state that pericarditis arising during the course of typhoid fever occurs in two forms, namely, the fibrinous, which is characterized by an abundant pseudomembranous exudation with only slight serous effusion, and the fibrinopurulent form, in which a considerable effusion may occur. Pericarditis may exist alone or may occur in connection with endocarditis, myocarditis, pleuritis, or pulmonary complications. As a rule, it develops very slowly and may remain latent, so that only most careful auscultation over the præcordial region will reveal the presence of friction fremitus, and later careful percussion may be required to distinguish an effusion. The pathogenesis of this complication is obscure. The purulent form when it occurs may be due to secondary infection, although the fibrinous variety is probably due to a direct infection with the Eberth bacillus. Typhoid fever complicated by purulent pericarditis is always fatal, but the existence of the serofibrinous pericarditis influences prognosis slightly if at all, unless the effusion be profuse.

Sudden cardiac failure may occur as the result of myocarditis, or of embolism or thrombosis of the coronary arteries, from heart clot, thrombosis of the cavæ and pulmonary veins or from pericarditis with effusion. Sometimes the cardiac failure is gradual when due to these causes.

So far as the *bloodvessels* are concerned the most common lesion is *phlebitis*, which usually affects the veins of the left leg, especially the femoral vein. The frequency of involvement of the veins in the left leg depends upon the pressure exercised by the right common iliac artery upon the left common iliac vein, which tends to obstruct the flow of blood. Sometimes the tendency to the formation of a thrombus is greatly increased by a local infection of the endothelial lining of the vessel, and it is not uncommon for a severe chill or chills to mark the onset of the lesion. Wright and Knapp have recently shown that the tendency to the formation of a thrombus in typhoid fever is augmented by the increase of calcium in the blood. When milk is the exclusive diet the rise in the proportion of calcium oxide supplied to the body is very noteworthy. They also recommend that for the prevention of this state the physician add 20 to 40 grains of citrate of soda to each pint of milk taken by the patient in order to decalcify it.

Thromboses of extraordinary size and number may form and extend from the femoral vein to the vena cava. When venous plugging seriously interferes with the circulation, the gangrene which results is usually moist, but in the vast majority of cases of phlebitis of the leg partial recovery takes place, although varicosity of the veins of the limb may persist after convalescence is completed. The rarity of plugging of the veins of the upper extremity is remarkable.

Arterial thrombosis is much more rare than is *venous thrombosis*. This complication usually develops after the second week of the fever, and is manifested by pain and tenderness along the course of the vessel affected. Usually the leg is the limb involved. After a temporary increase in the force of pulsation in the affected vessel the pulse becomes small and may be lost. The part becomes cold and discolored, and finally gangrene ensues. In other cases, in which the vessel which is involved is small, recovery takes place by the establishment of a collateral circulation. Even in the mild cases the patient suffers afterward from fatigue in the affected limb on exertion, and intermittent claudication may develop. The condition is due to an arteritis.

Thayer has published statistics which seem to indicate that typhoid fever is prone to produce early senile changes in the bloodvessels in after years.

COMPLICATIONS IN THE ALIMENTARY CANAL.—The complications in the upper digestive tract are pharyngitis, which is rarely severe enough to cause much discomfort, and œsophagitis, which is still more rare, although several observers have recorded *ulceration of the œsophagus*. Inflammation of the parotid gland is a rare complication of typhoid fever, and usually occurs about the third week in cases of severe infection. This inflammatory state may be due to infection of the gland from the mouth by ordinary pus organisms, or more rarely be due to the specific bacillus. Rarely parotitis occurs in the first week. In the only case the author has seen in which this complication developed at this time there was no pain or redness, and the swelling disappeared in about ten days. It was also bilateral. In advanced typhoid fever it is usually bilateral; is often followed by ugly sloughing, and is a very dangerous complication.

The *stomach* in typhoid fever is rarely much affected. Digestion in this viscus is, as a rule, feeble because in all fevers there is a lack of gastric secretion, and this is particularly true of typhoid fever. Vomiting may come on usually as a result of indiscretions in food and medicine. Sometimes, however, late in the disease a persistent, pernicious vomiting develops which only ends with exhaustion and death. A few cases of gastric ulcer occurring in typhoid fever are recorded.

When there are more than three or four stools a day diarrhœa is to be considered excessive. When a far greater number occur, it is usually the result of improper feeding. The stools are thin and resemble pea-soup save that they are apt to be a little more yellow. They are alkaline in reaction, offensive, and may contain particles of undigested food, as curds of milk, and also small shreds of lymphoid tissue from the sloughs of the bowel. The specific bacillus usually is not to be found in the stools until about the seventh or tenth day. The significance of active diarrhœa as to the gravity of the case has been much discussed, some believing that it is a sign of a severe infection. The real significance is not of severity of infection, but of severity of intestinal involvement, catarrhal or ulcerative, although in some cases even the latter state does not provoke active diarrhœa. General diffuse pain in the bowels is often present early in the disease, but is apt to disappear later.

Hemorrhage from the bowel in typhoid fever is one of the inevitable complications in a certain percentage of cases, and usually takes place after the second week of the disease. Very rarely slight loss of blood may occur in the first week. Proper treatment of the patient all through his attack may diminish toxæmia and prevent a fatal terminal infection, but no form of treatment so far devised has materially diminished the frequency of hemorrhage or the mortality from this cause, although the frequency of the occurrence and mortal effects vary greatly in different epidemics. The general average of its occurrence may be placed at 5 per cent. In 52,196 cases of typhoid fever collected from several series of cases reported by French and German physicians, and from the official reports of hospitals in the United States and Canada, England and Ireland, Germany, Austria, South Africa, and Australia, hemorrhage is stated to have occurred in 2725 cases, which gives a percentage of 5.22. The mortality in persons suffering from it is about 35 to 50 per cent., although in 271 cases of intestinal hemorrhage complicating typhoid fever, collected from the official reports of hospitals in the United States, Canada, England, and Germany, 71 cases proved fatal, which gives a percentage of 26.2. Hemorrhages usually arise from ulcers in the small intestine and are very rare in children. The symptoms consist of sudden fall in the temperature and it may be in the pulse rate, but this primary decrease is usually followed by a more rapid pulse than existed before the accident occurred. A diagnosis of hemorrhage is to be reached not only by the observance of the symptoms just described, but in addition by the presence of blood in the stools and by examining the blood to discover a paucity of hæmoglobin. The gravity of a hemorrhage depends upon the relation of the quantity of blood lost to the vitality of the patient and the frequency with which the bleeding occurs. Thus a fairly profuse hemorrhage in a strong patient may be followed by no severe symptoms, whereas repeated small hemorrhages may greatly exhaust the most lusty individual. When the patient is at the end of a long and severe attack of the fever, even a comparatively small hemorrhage may be fatal. The existence of small losses of blood not sufficient in size to be manifest to the eye when the stools are examined, may be discovered by the tests for occult blood named in the Article on Gastric Ulcer.

Perforation of the bowel, the most serious of all the complications of this disease that is commonly met with, has no relation to the severity of the general symptoms, for it occurs as often in mild as in severe cases. Indeed, in nearly 50 per cent. of recorded cases this accident occurred in mild cases. When perforation occurs the symptoms are apt to be ushered in by agonizing pain, usually felt in the appendicular region, which may be severe enough to rouse the patient from a considerable degree of stupor. If the patient is not too apathetic the pain is often described as being in the lower zone of the belly near the median line, and most commonly slightly to the right. The belly-wall is sensitive to palpation, speedily becomes tense and tympanitic, and all the symptoms of a general diffuse peritonitis may quickly ensue. The pain may, however, not be persistent, but pass away or become modified, as the peritoneal condition resulting from the escape of fecal matter into its cavity becomes more and more septic. The pulse becomes rapid and running, and collapse may speedily assert itself. When this occurs, death speedily comes on, the patient dying in a few hours, or, again, he may rally and survive for several days. Early death is, however, the more common result. Thus in the collection of 34 cases made by Fitz, of Boston, 37.3 per cent. died on the first day, 29.5 per cent. on the second, and 83.4 per cent. in the first week. During the second week 9 died, in the third week 4 died, and 2 other cases lived thirty and thirty-eight days, respectively. If collapse does not ensue, the rally of the system results in a rise of the temperature to a point higher than before the accident, and this movement is often accompanied by chills and rigors. Usually by the second or third day the peritoneal symptoms become more and more marked, the condition of the patient more and more asthenic and depressed, and death results by the fourth day from a general peritonitis with toxæmia from the absorption of toxic materials. In other cases the onset of the perforation is insidious; the belly before the perforation may have been moderately tympanitic, but now becomes intensely hard and rigid; the pain, which in some cases is so severe, does not develop, but the great fall in fever followed by a rise, and this again by rigors, it may be, give evidence of the grave accident which has occurred. The pulse becomes increasingly rapid and running, and the respiration more and more costal and less and less diaphragmatic, until the patient sinks out of life, without much, if any, suffering, in generally the same manner as one sees death come to a case of diffuse septic peritonitis due to a pyosalpinx or to septic appendicitis. In such cases the perforation is usually very small, and is so surrounded by adhesions that the escape of the intestinal contents is very gradual and insidious, infecting the peritoneum without the escaping fluid being copious enough at any one time to produce great pain or reaction.

In this connection it is important to note that a sudden fall in temperature is not a symptom necessary to the diagnosis of intestinal perforation. On the contrary, there are many cases on record in which a rise of temperature has followed this accident.

The diagnosis of perforation is to be reached by the following signs in addition to those just given: The hand of the physician, when lightly placed upon the abdominal wall, not only develops the fact that it is hypersensitive, but that its muscles are unduly tense. If the perforation has occurred very recently the rather swollen and tumid belly may be slightly scaphoid. Percussion may indicate the presence of gas in the peritoneal cavity, and the liver may be pushed away from the abdominal wall in such a manner that the ordinary area of liver dulness is largely decreased. Percussion of the right hypochondrium is, therefore, an essential procedure in the physical diagnosis of these cases. A fallacy underlying this test is the possibility of a portion of the colon, when greatly distended with gas, slipping up between the liver and the belly wall, and thus giving resonance; but this is a rare occurrence.

In some cases, however, as intimated, the symptoms are so insidious that the absence of this sign does not negative the diagnosis of perforation. Indeed a positive diagnosis may not be possible, and cases are sometimes met with in which the perforation has not been suspected, and is found only at the autopsy.

The diagnosis of peritonitis due to perforation is aided, but not confirmed, if an examination of the blood reveals a leukocytosis of polymorphonuclear cells.

There are several conditions causing pain which must be carefully excluded before the physician can arrive at the diagnosis of perforation, even if the symptoms and signs just described are present. These are diaphragmatic pleurisy, pneumonia of the bases, appendicitis, iliac thrombosis, and intestinal obstruction. Further than this, peritonitis may develop from extension of the inflammatory process in the bowel or by reason of the migration of micro-organisms through those parts of the bowel wall which have been impaired by the ulcerative process. In such cases the pain, swelling, and diaphragmatic paralysis may all be present without being due to perforation, and so closely may the symptoms of perforation be aped that operation has been performed, with the discovery that no perforation had occurred; thus in a case under the care of Herringham, nothing was found at the section and the patient recovered. Perforation may also be simulated by suppuration and rupture of a swollen mesenteric gland. Other causes of peritonitis are necrosis of the mesenteric glands, infarction of the spleen, or the development of abscess in an ovary or Fallopian tube. Very rarely peritonitis arises from cholecystitis or cholangitis, with or without gallstones. Liebermeister has recorded two cases in which rupture of the gall-bladder with escape of gallstones into the abdominal cavity took place. An ulcer in the appendix may perforate or an intercurrent appendicitis may complicate the case.

The percentage of frequency of occurrence of perforation is generally stated to be about 2.2, but in 30,966 cases of typhoid fever collected from several series of cases reported by French and German physicians, and from the official reports of hospitals in the United States and Canada, England and Ireland, Germany, Austria, South Africa, and Australia, perforation is stated to have occurred in 1144 cases, which gives a percentage of 3.69. The percentage of its mortality, when surgical interference is not resorted to at the most favorable time, is 90 to 95 per cent., and with operative interference it may be as high as 81 per cent. (See Treatment.)

Perforation is very much more frequently seen in men than in women. Fitz in 444 cases found 71 per cent. in men and 29 per cent. in women. In 21 cases of perforation in Basle, 15 were men and 6 were women; and Griesinger in 14 cases had 10 men and 4 women. Murchison also found in 24 cases 16 men and 8 women, although the general mortality of the disease among women was slightly higher than among men. So, too, Bristowe, of London, met with this accident in men in 11 of 15 cases, and, again, Nacke collected 106 perforation cases, of which 72 were in men and 34 were in women.

Perforation is responsible for a large proportion of the deaths which occur from typhoid fever. Out of 1721 cases which came to autopsy the percentage of deaths due to perforation was 11.3, according to Murchison. According to Hölscher, it was found in 2000 Munich cases 114 times (5.7 per cent.), and in 20 out of 80 of his cases which ended in death. In 4680 cases tabulated by different writers, Fitz found the proportion to be 6.58 per cent., which agrees with Hölscher's statistics. Hoffman found that out of 250 deaths in typhoid fever 20 were due to perforation.

Perforation takes place most commonly in the third and fourth weeks of the malady, but is by no means rare in the second week. It occurs most commonly in patients between twenty and thirty years of age. Elsberg has reported a case of a child of three and a half years who suffered from this accident, but whose life was saved by abdominal section.

The relation of typhoid fever to *appendicitis* is one of great interest. It has been thought by some that appendicitis arising in typhoid fever was a mere coincidence; by others, that its origin depended upon a general infectious process; and, again, by others, that it was due to the direct infection of the appendix with the bacillus of Eberth. Probably all these views hold true in individual cases. The richness of the appendix in lymphoid tissue, and the fact that typhoid fever is particularly prone to attack such tissues, renders this organ peculiarly susceptible on theoretical grounds. That this view is correct is proved by the research of Hopfenhausen, who collected the appendices obtained from 30 cases of typhoid fever and studied them under Stilling in the University of Lausanne. She concludes that moderate changes in the appendix may be found in nearly all cases of this disease, that it is most marked in the earlier stages of the malady, and consists chiefly in cellular infiltration, specific lesions being rare and not sufficient to produce the more severe forms of appendicular disease.

True appendicitis complicating typhoid fever, in the sense of inflammation of the appendix severe enough to produce abscess, is undoubtedly a very rare affection. Hopfenhausen has collected statistics of 743 cases of appendicitis, of which 5 per cent. were due to typhoid fever. This must be a very much larger percentage than usually exists.

It is a noteworthy fact that appendicular symptoms are not infrequent in early typhoid fever, and often disappear under rest in bed, and with the full development of the infection. Rarely the inflammation goes on to the formation of an appendicular abscess or perforation. The swelling of the lymph node in the mesoappendix and the presence of ulcers in the cæcum explain why it is that pain in the appendicular area is by no means rare. (See Plate I.) Sudden pain in the lower zone of the abdomen may be indicative, not of appendicitis, but of the presence of an iliac thrombosis.

Tympanites in typhoid fever is always present to some degree at some stage of the disease. When very marked, it is an evil symptom because it indicates active fermentation in the bowel, and the presence of intestinal atony, and because the gas presses on the abdominal thoracic viscera and disturbs their functions. By distending the intestine it may also predispose the patient to a hemorrhage or perforation by the strain on a severely ulcerated Peyer's patch.

HEPATIC COMPLICATIONS.—The *liver* and *gall-bladder* rarely show signs of active infection during the early part of an attack of typhoid fever. Jaundice is one of the rarest complications of this disease. Aside from some swelling and tenderness in the hepatic region, no symptoms in the hypochondrium are usually observable. It is, however, important to note that secondary involvement of the gall-bladder as a sequel of this malady is by no means rare, a true *cholecystitis* developing in a goodly proportion of cases as a result of infection of this viscus by the bacillus of Eberth. This cholecystitis may be severe enough to result in empyema of the gall-bladder and perforation of its walls with symptoms resembling intestinal perforation. A still more interesting fact is that such a cholecystitis due to this organism may develop many years after the attack of typhoid fever, and again that the clumping of these organisms in the gall-bladder may give rise to the formation of gallstones.

Louis, in his work on typhoid fever, published in 1836, states that changes in the bile and gall-bladder occur more frequently in typhoid fever than in other acute diseases, and cites 3 fatal cases in which cholecystitis, unrecognized during life, was found at autopsy. Grisolle and Andral mention similar cases. In 3 instances French found the gall-bladder of persons who had died of typhoid fever filled with turbid albuminous fluid, and Rokitansky speaks of having found "fibrinous exudations" in the gall-bladder of several patients who died from the disease. Murchison refers to the cholangitis and cholecystitis which may accompany typhoid fever, and reports a case of rupture of the gall-bladder, followed by general peritonitis. In 1876 Hagenmüller reported 18 cases of cholecystitis complicating typhoid fever. He concluded that it was a more frequent complication than had generally been supposed. Hölscher, in the 2000 Munich autopsies, found empyema of the gall-bladder 5 times.

In 1889 Bernheim suggested that typhoid bacilli might give rise to gallstones by producing alteration or stagnation of the bile. In 1893 Defourt reported 19 cases of cholelithiasis, in which the first attack of biliary colic occurred at varying periods after typhoid fever. Osler has reported a case of hepatic colic occurring for the first time in the fifth week of typhoid fever. At operation nothing could be found to account for perforation of the gall-bladder, but nine months later a gallstone was discharged.

Fournier found bacteria in 38 out of 100 gallstones which he removed at autopsies. The colon bacilli predominated, while the typhoid bacilli were found to be second in frequency. Milian, Chantemesse, and Horton Smith report similar experiences.

Chiari found typhoid bacilli present in the gall-bladder in 19 out of 22 cases, and obtained pure cultures from 15. In 9 out of 10 cases at St. Bartholomew's Hospital, London, *Bacilli typhosi* were found. Cushing mentions 5 cases of cholecystitis complicating typhoid fever, in which pure cultures of colon bacilli were obtained from the pus. Marsden reports a case in which cultures resembling *Bacillus typhosus* were obtained. Van Dungern obtained pure cultures of typhoid bacilli from pus surrounding the gall-bladder fourteen years and a half after an attack of typhoid fever. Pure cultures have often been obtained from six to eight months after the attack (Chantemesse, Dupré).

Mason thinks that the bacilli gain entrance through the biliary ducts. Councilman believes that they are carried through the blood, and that areas of necrosis in the liver afford them portals of entrance. Hagenmüller, Mayo Robson, and Mark Richardson believe that biliary complications, especially cholecystitis, are due to ascending infection of the ducts. Marsden is of the opinion that the most important passage of bacilli into the gall-bladder is through the blood, the liver, and the biliary ducts. He is undoubtedly correct.

Typhoid cholecystitis during the course of the fever is frequently latent. In more than one-half the recorded cases, either on account of latency of symptoms or typhoidal stupor, nothing unusual was observed during life.

The two most constant symptoms are pain and swelling, the former being paroxysmal and most marked in the region of the gall-bladder and under the scapula. Maurice Richardson says that it may be in the epigastrium or over McBurney's point. According to Mayo Robson, if a line be drawn from the umbilicus to the ninth rib on the right side, there is almost always tenderness at the beginning of the second third of this line. Jaundice is rarely met with, but there may be repeated chills and sweats.

GENITO-URINARY COMPLICATIONS.—*Albuminuria* in typhoid fever is quite a constant condition, occurring as frequently as in 70 per cent. of all cases, and being most marked in the second week. Usually its presence is not associated with that of tube casts unless the patient is already a sufferer from nephritis prior to the attack. When casts are present, the albumin is usually present in large amount. Albuminuria without casts is not a serious complication. Probably *true nephritis* is present in almost 20 per cent. of the cases, but this is usually not productive of renal symptoms. An antecedent nephritis may take on renewed activity and a true hemorrhagic nephritis may occur, usually in severe cases only. The urine is apt to be scanty and of high specific gravity unless the physician insists upon the patient drinking freely of water.

Pyuria in slight degree is common. Blumer says it occurs in 17 per cent., but it is a noteworthy fact that pyelitis due to typhoid fever is almost unknown. While this is true, it is also of interest to note that enormous numbers of the bacillus of Eberth are to be found in the urine after the second week of the disease, and often far into convalescence. Petruschky has estimated that 1 c.c. may contain 170,000,000 bacilli. A profuse *polyuria* is often present when the stage of convalescence is entered upon.

Orchitis and *epididymitis* rarely occur as a result of a direct infection with the specific bacillus. They differ from the changes due to gonorrhœa in that they are less painful and more rapid in their course to suppuration or recovery. They are usually unilateral and the testicle is first affected. Typhoidal *cystitis* due to the presence of the bacillus of Eberth rarely occurs.

RESPIRATORY COMPLICATIONS.—The respiratory disorders met with in connection with the course of typhoid fever, aside from the *bronchitis* already mentioned, are quite numerous. In the later stages of the disease we may meet with severe *laryngeal ulceration*, which in turn may be complicated by perichondritis or œdema of the glottis. Hoffman found 28 cases of ulcer of the larynx in 250 autopsies in this disease, and Griesinger in 26 per cent. of those dying of the malady, so that it is by no means rare. Keen collected 146 cases of severe laryngeal disease due to this cause, and found that necrosis of the laryngeal cartilages when it occurred was a very fatal complication, death occurring in 95 per cent. of the cases.

Intense hypostatic congestion is one of the most constant pulmonary changes seen at autopsy; in some cases the blood may inundate the air vesicles, causing solidification. How often this change is agonal cannot be determined with any degree of certainty, but as it depends on more or less prolonged maintenance of one position aided by an enfeebled circulation the danger can be greatly lessened, if not avoided, by frequent changes in posture.

Pneumonia develops in typhoid fever in three forms and in different stages of the disease: (1) As an acute lobar pneumonia ushering in the attack of enteric fever, and due to the *Micrococcus lanceolatus*, or, it is thought by some, to the infection of the lung by the bacillus of Eberth, the so-called "pneumotyphoid." True croupous pneumonia in the later stages is very rare. (2) Bronchopneumonia, probably arising from terminal infection or by hypostatic congestion due to the profound toxæmia and cardiac degeneration and feebleness, is more common. (3) Acute tuberculous pneumonia sometimes seizes the typhoid fever patient when he seems about to begin his convalescence.

It is not to be forgotten that infarction of the lung may occur as the result of cardiac or venous emboli. Such an infarction may mislead the physician into a diagnosis of lobar or lobular pneumonia by reason of the dulness on percussion, the rise of temperature, and blood-tinged sputum. An infarction may, if the patient survives, result in pulmonary abscess or gangrene.

Pleurisy arises very rarely as a primary lesion. It is usually secondary to infarction, pneumonia, or gangrene. Cases of *empyema* due to the specific bacillus have, however, been recorded.

NERVOUS COMPLICATIONS.—The nervous disturbances vary very greatly. In the average case there is in the early part of the onset no mental change save that of unfitness for mental occupation, with dreamful sleep which is apt to be restless. Later the patient continually dozes off, yet awakens easily, and for a moment may be a little confused between the mental impressions left on his brain by the dream and the conditions he finds about him on returning to consciousness. Still later, if the infection is severe, he becomes more *apathetic* when awake, less easily aroused when asleep, and often delirious in his sleep, his dreams being evidently vivid, so that he keeps muttering the conversation he thinks he is actually having, or calls out loudly, as his dream seems to lead him to a point where an imperative call or sudden action is needed. Sometimes the *delusions* in the delirium amount to imperative conceptions, and the patient believes that he is away from home and must return there at once, or that he is being restrained by force, or, again, that some member of his family is in distress and needs his aid or is calling for him. Often this form of mental disturbance is painful to witness, difficult to overcome, and harassing to the patient. In these cases the hands may be moved continually in active motions, as if to illustrate the ideas of the patient. Such cases are apt to be grave if for no other reason than that they exhaust themselves if relief is not given. The more encouraging type of delirium is of the quiet, muttering form, as if the patient was gently "speaking in his sleep" as in health, and this may be taken as the natural form of delirium in the disease. Later the stupid condition becomes more and more marked in some cases, and absolute mental stillness is reached, in which only rough shaking or loud calling will arouse the patient. In severe cases with marked toxæmia we find at times a state of mental confusion, staring eyes, and semi-stupor, with persistent muttering, the so-called *coma vigil*.

During convalescence *mental aberration*, depending usually upon exhaustion, may develop. The prognosis in such cases is usually good.

Rarely in the course of typhoid fever symptoms of *irritation* or *inflammation of the meninges of the brain* develop, and it is important to remember that these symptoms may arise from several causes. The most common of these is congestion and engorgement of the meningeal vessels without any true inflammatory process; the next most common form is that due to the extension of an infection from abscess in the middle ear; the third form is that in which there is infection with the streptococcus or pneumococcus, and very rarely the meningitis is due to the bacillus of Eberth. Cole has recorded three instances in which the typhoid bacillus was obtained from the cerebrospinal fluid by lumbar puncture in typhoid fever. In one the meningitis was serous, in another purulent; the character of the other is not stated.

The frequency of this complication in the different periods of the disease when due to true typhoid infection of the meninges is in direct ratio to the length of the malady, namely, in the third or fourth week. In the great majority of instances in which the complication has appeared the patient was under thirty years, and usually between twenty and thirty years. That is the period in which typhoid fever is most commonly seen.

In every case of true *typhoid meningitis*, so far recorded, death has occurred, but this is a statement which does not possess as great prognostic value as would appear at first glance, since an absolute diagnosis of true typhoid meningitis can not be made during life, for the positive test is the bacteriological examination of the skull contents. Nevertheless, the presence of marked meningeal symptoms is of the gravest import in all cases.

Sometimes, because of degenerative changes in the vessels, a hemorrhagic effusion into the meninges of the brain takes place, but this does not commonly produce marked symptoms unless it is profuse.

Convulsions, generalized or localized, with coma and delirium may arise from thrombosis of the cerebral sinuses or of the cerebral arteries, but they are very rare from any cause. Murchison only met with them in 6 cases out of 2960. If due to the lesions named, they indicate a fatal termination in the near future. In Osler's case death followed convulsions, produced by thrombosis of the branches of the left middle cerebral artery, in twelve hours. If they occur in neurotic children or females, the outlook is not so gloomy, as they probably do not depend upon an actual lesion in the brain.

Sometimes *acute otitis media* produces violent headache and finally symptoms of meningitis, but its presence is often unrecognized as a cause until a discharge takes place from the ear.

Neuritis, generalized or localized, is met with occasionally in the later stages, producing wrist-drop or toe-drop, and sometimes causing severe pain. When there is a multiple neuritis the symptoms may closely resemble locomotor ataxia or anterior poliomyelitis. Sometimes the skin of the toes or of the whole foot becomes exquisitely sensitive.

When *hemiplegia* occurs, which is quite rare, it results from cerebral embolism or thrombosis or very rarely from actual hemorrhage.

COMPLICATIONS IN THE BONES, JOINTS, AND MUSCLES.—Secondary disease of the bones, consisting of *post-typhoidal osteomyelitis* due to the specific bacillus or to infection by associated micro-organisms, may occur. The tibia and the ribs are the bones most commonly involved, and the changes are subacute or chronic rather than acute. So, too, *arthritis* may be due to pyogenic micro-organisms or to the Eberth bacillus, and is usually of a subacute or chronic type. *Spontaneous dislocation of the hip* may occur in very rare instances.

Many years ago V. P. Gibney, of New York, described, under the name of *typhoid spine*, a condition in which there develops, often some days after the patient is up and about, and often only after some very slight jar or trauma, great tenderness of the spine, with pain in the back, and in the legs when they are moved. Usually it is held that this condition is not dependent upon a spondylitis, neuritis, or Pott's disease, and is probably a neurosis closely allied to the neuroses seen in cases of severe trauma.

Fraenkel has recently shown that in fatal cases of typhoid fever the bacillus may be obtained from the cancellous tissue of the bodies of the vertebræ and some of these cases of so-called typhoid spine may be instances of osteomyelitis involving these structures.

Sometimes in the stage of convalescence a curious state is developed in which the muscles of the lower extremities become painful, somewhat brawny, and even slight redness may appear in the skin covering them. Usually this is unilateral, but it may be bilateral. Most commonly it affects the calf of the leg, and pain is developed on pressure or on movement, active or passive. This is due to a *myositis*. It should not be confused with phlegmasia dolens due to thrombosis.

TYPHOID FEVER COMPLICATING PREGNANCY.—In a very large number of cases of typhoid fever complicating pregnancy, abortion or premature labor comes on. Corbin collected 364 cases of typhoid fever occurring in pregnant women, and Fellner, of Vienna, has added 7 others to this number, making a total of 371 cases. Of these 371 cases 228, or 61 per cent., ended in premature births, and in 202 cases pregnancy terminated before the sixth month. Most of the full-term children were born dead, and those who were born alive were weak and did not long survive. The mortality in the mother under these circumstances is about 16 per cent.

Diagnosis.—The diagnosis of typhoid fever is to be based on the characteristic ascent of the temperature, the general malaise of the patient, the peculiarly coated tongue with red edges, the tumid belly, and the development of the rash about the seventh to the ninth day. If to these symptoms are added an enlargement of the spleen and liver, the diagnosis becomes still more certain, and is confirmed if the laboratory tests mentioned on the following pages are positive. The laboratory aids to diagnosis are the Widal or agglutination test; the isolation of the bacillus from the blood, from the stools, from the urine, and from the rose spots, and the diazo reaction. The objection to these tests is the difficulty as to technique for the general practitioner, and, more important still, the fact that some of them are obtainable in many instances so late in the course of the disease as only to confirm the clinical diagnosis already made. (See page 48.)

Typhoid fever must be separated from a number of maladies which closely resemble it. Pure typhoid infection may result in the production of a fever which closely follows the remittent or intermittent malarial types, and which is often associated with so much gastric disturbance and vomiting and so lacking in the more prominent typhoid symptoms usually seen that the picture of remittent malarial fever is clear, while the true picture of typhoid fever is clouded. Again, there can be no doubt that cases of true malarial infection occur in which the symptoms so closely resemble those of typhoid fever that a purely clinical diagnosis is almost impossible if an epidemic of typhoid fever is in full swing at the time. Finally, there can also be no doubt that it is possible for the patient to have a double infection with the bacillus of Eberth and the plasmodium of Laveran, in which case, however, the malarial manifestations are usually dwarfed by the typhoid poison, and only are marked at the onset of the enteric fever and at its termination. To this mixed infection the term typhomalarial fever may be correctly applied to indicate not a separate disease, but a double infection. Etymologically, this term might also be used to define a condition of malarial fever in which, because of profound debility, the patient was in a typhoid state—that is, in a condition of which typhoid fever is a type. Practically, however, it should be discarded or limited in its use to the double infection just described.

How far constant fever occurring day after day and associated with manifestations of general loss of strength and debility can be relied upon in the diagnosis of typhoid fever is hard to determine. Certain it is that if a physician makes a diagnosis of enteric fever upon these symptoms alone, without bearing in mind the fact that similar conditions are equally well developed under other forms of infection, he will find himself in error in not a few instances. Chief among these conditions may be mentioned tuberculosis of the lungs or peritoneum, that form of influenza in which the chief symptoms are abdominal, cases of ulcerative endocarditis, septicæmia, and pyæmia, and those of cholecystitis with ulceration, as from impacted gallstones. It must not be forgotten, too, that syphilitic fever may in very susceptible persons resemble typhoid infection. The febrile movement, rose rash (if it be scanty), malaise, and signs of general infection in this disease may readily mislead the physician. Again, in the more advanced, or tertiary, stages of syphilis a prolonged low, septic fever may be present. Any case of so-called typhoid fever which lasts more than four weeks without the attack being prolonged by a relapse is almost certainly suffering from another disease, often tuberculosis.

It is not to be forgotten that trichiniasis may resemble typhoid fever, for in it we have fever, pains in the limbs and back, headache, stupor, and nausea, with pain in the belly and diarrhœa.

The differentiation of typhoid from other fevers is aided by a study of the following table:

Typhoid Fever.	*Typhus Fever.*
Onset gradual.	Onset abrupt.
Face dull and apathetic.	Face livid, anxious, swollen, conjunctiva reddened. Pupils contracted.
Delirium a late symptom.	Delirium an early symptom.
Coma a late symptom.	Coma an early symptom.
Eruption very late.	Eruption early.
Eruption chiefly on trunk, well defined, and appears in several crops of small rose-red spots.	Eruption over trunk and limbs and ill-defined. Does not appear in crops, and is dusky red or petechial in character.
Leukocytes decreased.	Leukocytes increased.
Widal test positive.	Widal test negative.
Bacilli of Eberth in blood.	Bacilli absent.

Typhoid Fever.	*Acute Miliary Tuberculosis.*
Rash appears in crops.	Rash, if present, not in crops.
Profuse sweats rare.	Profuse sweats constant.
Temperature curves regular.	Temperature curves irregular.
Pulse rarely over 100.	Pulse usually rapid.
Bacillus of Eberth in blood.	Absent from blood.
Widal test positive.	Negative.
No eye changes.	Choroidal tubercles.
Respirations slightly increased.	Greatly increased.
Cyanosis rare.	Cyanosis common.

Typhoid Fever of the Cerebral Type.	*Cerebral Meningitis.*
Regular temperature.	Irregular temperature.
No marked blood change.	Increase in polynuclear white cells.
Herpes very rare.	Very common.
Rose rash on trunk chiefly.	Petechiæ over whole surface.
Cerebrospinal fluid negative.	Positive for the specific bacillus.

Typhoid Fever.	*Æstivo-autumnal Fever.*
Onset gradual.	Onset acute.
Fever gradually rises.	Fever rises irregularly.
Chills rare in onset.	Severe chills common.
Unaffected by quinine.	Improved by quinine.
Heavy facial expression.	Anxious facies with slightly icteroid conjunctiva.
Herpes rare.	Herpes common.
Early delirium rare.	Early delirium common.
Anæmia moderate.	Anæmia marked.
Moderate reduction in leukocytes.	Great reduction in leukocytes.
Rose rash.	No rash.
Bacilli in blood.	Plasmodium in blood.

Typhoid Fever.	*Influenza.*
Onset gradual.	Onset sudden.
Enlarged spleen.	No enlargement of spleen.
Rose rash.	No rash.
Prostration gradual.	Prostration rapid.
Lasts several weeks.	Lasts a few days.

Typhoid Fever.	*Typhoid Pneumonia.*
Onset gradual.	Onset more rapid.
Nervous symptoms moderate.	Nervous symptoms severe.
No leukocytosis.	Marked leukocytosis.
Widal test positive.	Widal test negative.
Bacilli in blood.	None in blood.
Lasts weeks.	Lasts a shorter time.
Disease of youth.	Disease of old age.

Typhoid Fever.	*Ulcerative Endocarditis.*
No cardiac murmurs.	Cardiac murmurs.
Regular temperature.	Irregular septic temperature.
Sweats rare.	Sweats common.
No leukocytosis.	Marked leukocytosis.
No cardiac dyspnœa.	Cardiac dyspnœa.
No petechiæ.	Petechiæ.
No infarctions.	Infarctions.
No leukocytosis.	Leukocytosis.
Widal test positive.	Negative.
No retinal emboli.	Retinal emboli.
No chills.	Repeated chills.
Bacilli in blood.	No bacilli in blood.

Typhoid Fever.	*Trichiniasis.*
Rose rash.	No rash.
Face not swollen.	Face swollen.
Muscles normal.	Myositis.
Eosinophiles decreased.	Eosinophiles numerous.
A common disease.	A rare disease.

For the diagnosis of paratyphoid fever from typhoid fever, see the article on that disease.

TESTS.—The so-called Widal test depends upon the fact that if a small amount of blood, or blood serum, or even the breast milk or tears from a patient having or recently having had typhoid fever, are brought in proper dilutions in contact with living typhoid bacilli these organisms soon cease to move, that is, lose their motility, and gradually come together in clumps, or, in other words, agglutinate.

Nahrt and Bolton have found that agglutinating properties are transmitted from mother to child by means of the milk, and that the typhoid bacillus, when present in the fœtus, produces agglutinins in its blood. They were unable to determine whether agglutinins were transmitted from mother to fœtus through the placenta.

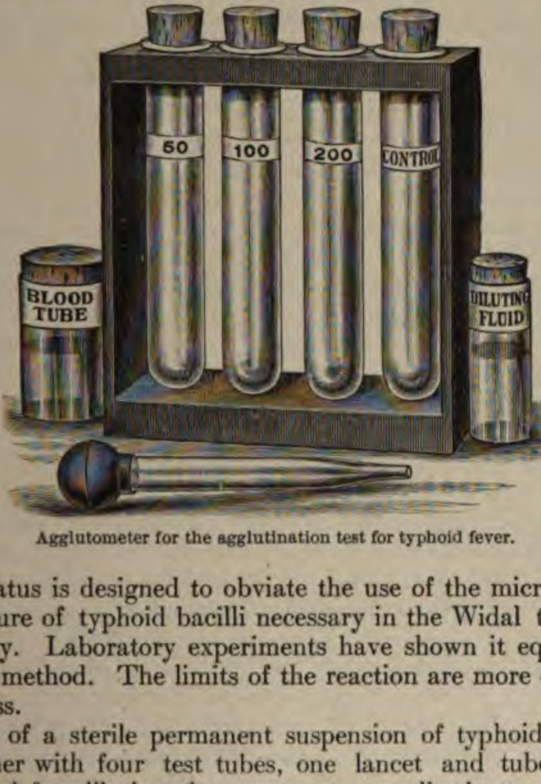
The typhoid bacilli to be employed in the test are not such as have been recently isolated from a case of typhoid fever, but those which have been modified by repeated transplantation on artificial media. These bacilli are kept in sealed tubes of nutrient agar-agar in an ice-chest; from such a stock culture inoculations are made, and when the test is to be used are placed in broth-bouillon, incubated for twenty-four hours at a temperature of 37° C., and then employed for the test. It is essential that it be proved beforehand that this culture is composed of organisms reacting to known typhoid serum and not to healthy serum. From this test culture a proper dilution is made by adding the bacilli to blood diluted with normal salt solution. A hanging drop is now placed under the microscope and examined with a magnifying power of about 800 diameters. The bacilli should appear as actively motile organisms which do not clump.

The finger-tip or lobe of the ear is pricked, and by means of the "white pipette" of a blood-cell counting apparatus the blood is drawn up to the mark 0.5. Then the pipette is dipped in distilled water and the water is drawn up till the figure 11 is reached. This gives us a dilution of 1:20. One drop of the mixture of bacilli in salt solution and one drop of the diluted blood are then placed on a cover-glass, which is inverted over a hollow slide and the drop examined. A positive reaction consists in an absolute immobilization of all the bacilli and of a clumping of a majority of them. This reaction should occur in five minutes if the dilution of blood has been 1:20, and in thirty minutes if it has been 1:40, and in two hours if the dilution has been 1:60. A rapid clumping with a weak dilution is to be regarded as a very positive test. On the other hand, it is to be remembered that a dilution of blood in the proportion of 1:10 may give a reaction even if normal blood is used. An exact estimate of the strength of the solution and of the time of reaction is therefore of importance.

This test is an exceedingly accurate one, if properly employed. The chief difficulty about it is that the reaction is often absent until the seventh or even the twelfth day of the disease. Out of over 8000 cases reported by a number of observers, the test was positive in 94 per cent. A negative result is unimportant if it is obtained prior to the third week. But cases have been recorded in which bacilli have been isolated from the blood during life and at autopsy the lesions were those of typhoid fever, but at no time during the course of the disease did the blood yield the agglutinative reaction. Repeated tests should also be made before it is decided that the blood does not give the reaction. When dried blood is used its volume as near as may be should be restored by the addition of distilled water, and from this the proper dilution is to be prepared and the resulting dilution used as already indicated. The fallacies of this test lie in the possibility that the patient may have had typhoid fever at some previous time and so give the reaction, and in mistaking irregular and delayed clumping as true agglutination or as a partial reaction.

This test has now been brought within the reach of everyone by the use of an agglutometer which has been placed on the market by a well-known house. (See Fig. 12.)

FIG. 12



Agglutometer for the agglutination test for typhoid fever.

This apparatus is designed to obviate the use of the microscope and the fresh live culture of typhoid bacilli necessary in the Widal test when made in the old way. Laboratory experiments have shown it equal in delicacy to the former method. The limits of the reaction are more distinct than in the old process.

One bottle of a sterile permanent suspension of typhoid bacilli is furnished, together with four test tubes, one lancet and tube for collecting blood, one vial for diluting the serum, one small pipette for distributing the diluted serum, and one large pipette with two graduations (each corresponding to ten drops of the size delivered by the small pipette) for filling the tubes with suspension. The three tubes labelled 50, 100, and 200, are to be used for the test; the fourth is a control tube to which no serum should be added.

Let blood flow into the blood tube until the bottom is covered with a layer one-eighth to one-fourth inch thick. The blood will flow much more

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rapidly if the lobe of the ear is squeezed intermittently between the thumb and index finger.

Cork the tube and replace in an upright position.

In a short time (an hour) the serum will have separated, or may be readily made to do so by carefully loosening the edges of the clot with the lancet.

After the serum has separated, insert the pipette into the blood tube, the point resting in the lateral depression, and incline both slightly, when the serum will readily enter the pipette.

Add one drop of serum to ten drops of clear water in the diluting tube, and shake well. If the diluted serum is cloudy, let it clear by standing a few minutes before distributing to the tubes of suspension.

By means of the large pipette put 20 drops (two graduations) of the suspension of typhoid bacilli in each of the four test-tubes.

Add the serum dilution to the typhoid suspension in the following amounts: four drops added to the tube marked 50 gives a dilution of 1:50; two drops added to the tube marked 100 gives a dilution of 1:100; one drop added to the tube marked 200 gives a dilution of 1: 200.

No serum should be added to the control tube.

After adding the serum dilution, cork the tubes and shake well. Put away in a warm place.

Examine the tubes at the end of one and four hours, and again on the following day. The rapidity of the reaction depends both upon the agglutinating power of the blood serum and the temperature at which the tubes are kept. The reaction may be seen with the greatest distinctness when one stands near the middle of the room facing a window. The tubes should be held on a level with the eye and inclined slightly away from the observer.

When the reaction is positive, floccules appear in one or more of the tubes, depending upon the agglutinating power of the serum tested. These flakes are small at first and disseminated through the fluid. They gradually increase in size and settle to the bottom of the tube.

In a *complete* reaction the supernatant fluid is perfectly clear.

In a *positive* but incomplete reaction, floccules are seen in the still cloudy fluid.

In a *negative* reaction the fluid in the tubes remains uniformly clouded, as in the control.

All apparatus and corks should be thoroughly washed before using a second time.

The diazo reaction, sometimes called Ehrlich's reaction, depends upon the fact that in typhoid fever the urine of the patient contains a chromogen which, when treated with diazo-benzene-sulphonic acid and ammonia, produces a distinct red hue in the urine, which may be as deep as garnet red. Other diseases give this reaction, such as tuberculosis and some cases of pneumonia, but it is of considerable value in determining the presence of typhoid fever if taken in conjunction with other signs. It is usually present as early as the sixth day, and lasts until about the eighteenth day. The test itself consists in using two solutions. One of these consists of a 5 per cent. solution of hydrochloric acid to which has been added sulphanilic acid in the proportion of 1 gram for each 100 c.c. The other is a 0.5 per cent.

solution of sodium nitrite. When the test is to be made the two solutions are mixed in the proportion of 40:1. Equal parts of urine and this mixture are then shaken together and rendered alkaline by the addition of ammonium hydrate, which is allowed to flow down the side of the tube, forming the layer above the mixture just named. At the dividing line between these two fluids the reaction appears. If typhoid fever is present a garnet-red hue develops. If it is not present, only an orange tint is seen unless one of the other maladies which give this test is present. After the test tube containing these liquids has stood for some time a green sediment forms, which Ehrlich considers very characteristic of a true reaction.

Another method of reaching a positive diagnosis is the examination of the blood itself for the specific bacillus, which, as already stated, is present in this fluid in nearly all, if not all, cases of typhoid fever. While it is true that this examination is not possible for one who is not trained in its technique from the bacteriological standpoint, it is also a fact that this test is not open to the fallacies of the Widal test, and that the bacilli are often found as early as the fifth day, whereas the Widal test is frequently not positive till the ninth day, or even later. The urine and stools may be examined for the specific infecting micro-organism, but they are rarely discoverable in these discharges early enough to aid the diagnosis.

The Widal test and the discovery of the bacillus of Eberth in the blood enable us to differentiate true typhoid fever from paratyphoid fever.

Finally, it is to be remembered as a valuable diagnostic fact that the fever of the first stages of typhoid fever is more resistant to the cold bath than in any other malady, although it yields readily enough later on in the course of the malady to this therapeutic measure.

Prognosis.—The prognosis in typhoid fever depends upon several important factors. One of these is the time at which the patient comes under medical care, not because active medication is of great advantage, but rather because patients that go to bed late in the onset of the disease usually become more seriously ill than those who conserve their vital forces by rest from the very beginning of the malady. Patients who travel long distances in the early stages of typhoid are wont to have severe attacks, and if, after the disease is well developed, travelling is resorted to the illness nearly always increases in violence. Another factor is the state of the patient at the beginning of the malady, as to his vital resistance and general health. Fat persons usually do not withstand it well. Children nearly always recover from typhoid fever in its uncomplicated forms, and aged persons, while rarely affected, succumb when attacked in direct proportion to their years. (See Fig. 6.)

A third factor is the degree of toxæmia which develops in severe cases, particularly if they are not treated skilfully at first.

Aside from these general considerations it is impossible to make an accurate prognosis as to the severity of the attack or probable recovery of the patient in the first week of the disease, because the malady develops slowly and because a fatal termination is nearly always due to some intercurrent complication which cannot be foreseen. Even when the disease is ushered in with violence of all the symptoms, particularly an exceedingly high temperature, it often happens that it follows a very short and fairly mild course, so that a severe onset justifies the physician in expecting a speedy recovery in many instances. When, however, complicating conditions such as pulmonary, cerebral, or meningeal manifestations develop, the prognosis is of course correspondingly grave.

Recovery in typhoid fever, under the modern and favorable methods of treatment, takes place in about 93 per cent. of cases in the best types of private practice and in hospitals in which the patients are received early and in fairly good condition. In private practice among the poor it is much lower. In army practice the mortality may vary from 2 or 3 per cent. in time of peace to 50 per cent. in time of war, illustrating very well the fact, already stated, that early rest in bed, perfect quiet of mind and body, and proper nursing are most favorable in their influence, whereas an absence of these aids to recovery is most harmful. Under the cold-bath treatment of typhoid fever, when it is instituted early, the mortality of about 7 per cent. is largely due to those unavoidable accidents, hemorrhage and perforation of the bowel.

Much depends in all cases upon the severity of the infection. In some widespread epidemics the mortality is singularly low even when the care of the patients is not in many cases very skilful; in others it is correspondingly high. In the United States army in the Spanish war it was only 7 per cent., a remarkably low rate for war time; whereas in the Boer war the English troops suffered from a death rate of nearly 21 per cent.

Sudden death sometimes occurs in typhoid fever without the autopsy revealing any adequate cause, the real cause being in all probability an acute cardiac dilatation.

Treatment.—The following is the plan pursued by the author in the treatment of this disease. As soon as the patient comes under observation, unless his bowels have already been moved by the aid of calomel, he is given 1 to 2 grains of this drug in quarter-grain doses every hour. If his bowels are not moved in twelve hours, a movement is produced by the aid of a large rectal injection of soap and water, and if need be by the ingestion of a Seidlitz powder. Twelve hours later he receives 5 to 10 minims of dilute hydrochloric acid with a teaspoonful of essence of pepsin ; this is repeated regularly every six hours throughout the disease after food.

HYDROTHERAPY.—An order is given that if the temperature rises as high as 102.5° the patient is to be rubbed with tepid, cool, cold, or ice-water, or even with a piece of ice, according to the degree with which his temperature resists the bath and according to the degree of toxæmia present. If toxæmia is very great, it is often necessary to give a thorough, brief and brisk, rub-off with a small piece of ice, not so much to reduce the fever as to cause reaction and arouse the patient's vitality. With this application of cold, in different degrees according to the needs of the case, there must be employed by another nurse, or by the free hand of the nurse who uses the cold, active friction to the skin as the cold comes in contact with the integument, because friction increases the heat loss 50 per cent., aids in producing those most essential conditions, reaction and equalization of the capillary circulation, and prevents the patient from being chilled. It is a cardinal rule that if the patient has been ill so long that reaction does not occur under the bath, it is contraindicated and we must endeavor by gentle measures and the use of tepid or even of hot water to redevelop the power of the body to react. In other words, that temperature of water should be used which is necessary when combined with active friction to reduce the temperature at least 2° in fifteen to twenty minutes, provided reaction can be produced. Without reaction we simply increase internal congestions by the use of cold water. It is interesting to note that Hirschfeld has treated over 1000 cases with tepid immersion baths of 80° to 90° with a mortality of only 3.4 per cent.

Whenever cold is used, an ice-bag or cold cloth should be applied to the head to prevent cerebral congestion.

While the method of bathing just described is that nearly always pursued by the writer, it is proper to give definite information concerning the so-called Brand method of cold bathing, a plan which was introduced by Brand, of Stettin, many years ago, but which has only received its full share of credit during the past fifteen years. This plan consists in immersing the patient, when his temperature reaches 102° or 102.5°, in a tub of water the temperature of which is 70°, and keeping him there with active friction for fifteen or twenty minutes, until the temperature is reduced to 100°. In order to combat chilling and aid the circulation it is customary to give the patient one-half to one ounce of whiskey before, during, or after the bath. The bath is repeated whenever the temperature rises to 102°. Usually it is needed every two or three hours. In order that the patient's strength may be conserved he should be lifted into and out of the tub. When cold is properly used it should, after the first week of the disease, produce changes in the temperature, as shown in the following chart (Fig. 13).

This so-called plunge bath, or Brand bath, is a remedy of the greatest possible value, but is not needed in every case as a matter of routine. When used it is essential to produce reaction and to use friction, and to apply ice to the head. The indications for its use are identical with those just named. It is actually contraindicated in the very young and very old, in whom it is often difficult to produce reaction, and if the case comes under treatment so late as the beginning of the third week, since reaction to cold is usually then lost. The presence of a complicating pneumonia also contraindicates it. Other disadvantages are that the back cannot be rubbed, although the muscles in that part of the body contain much heat and the skin is most prone to suffer from bed-sores, and that the patient must be lifted or raise himself out of the tub. The temperature of the plunge bath when its use is deemed wise should not be placed at a tepid level and then reduced while the patient is in the water, as this does not administer a stimulating and awakening shock to the system, but simply chills the patient, thereby doing no good, for the object in using water in typhoid fever is to produce reaction, eliminate poisons, and reduce temperature, and the means by which this is best accomplished can be determined in each case by the physician.

Personally the writer has never failed to successfully accomplish all these results by cold rubbing, with friction, if it is properly given, but many physicians prefer to follow the method of Brand as a routine practice. An enormous array of statistics prove its value as a life-saving agent.[1]

Some form of bath at least once a day is absolutely necessary even if the temperature never exceeds normal, to establish cleanliness and equalize the circulation everywhere, and he who treats typhoid fever without resort to efficient hydrotherapy, if it can be used, is not doing all for his patient that can be done.

The use of hydrotherapy greatly lowers the mortality, saving about 10 in every 100 cases, but it does not diminish the frequency of perforation or hemorrhage, and it apparently increases the frequency of relapse. This may be due to the fact that more are saved to run the chance of relapse, but also may depend upon the fact that mild cases are more prone to relapse than severe ones. Hydrotherapy does not shorten the duration of the fever, but it often shortens the length of the illness by preventing complications.

FIG. 13

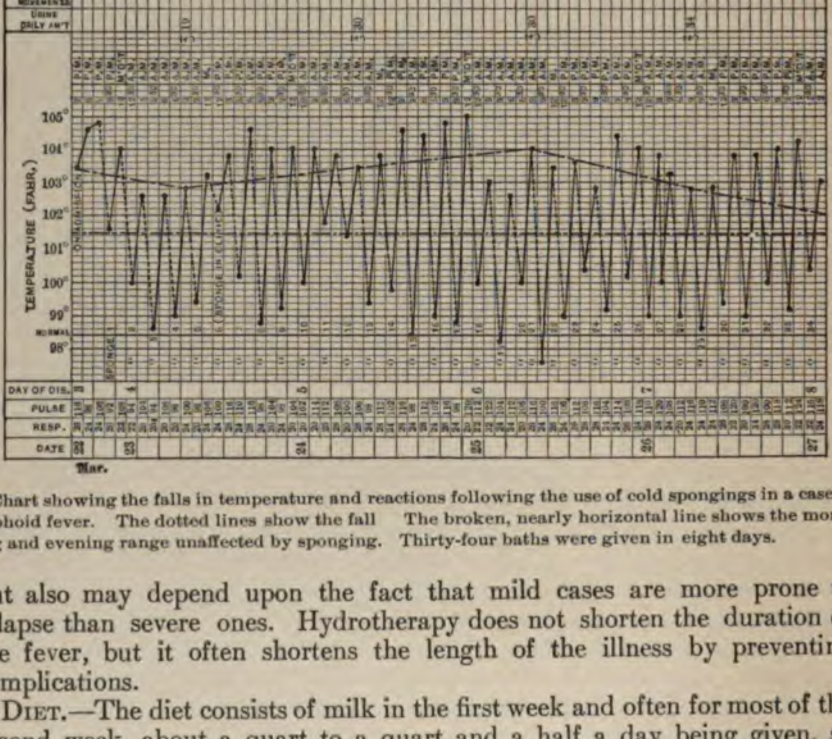

Chart showing the falls in temperature and reactions following the use of cold spongings in a case of typhoid fever. The dotted lines show the fall The broken, nearly horizontal line shows the morning and evening range unaffected by sponging. Thirty-four baths were given in eight days.

DIET.—The diet consists of milk in the first week and often for most of the second week, about a quart to a quart and a half a day being given, so divided that the patient gets it every three or four hours. It is followed by the acid and pepsin already named, unless the stomach is irritable, when a little lime-water may be given as a substitute, or a little Celestins Vichy water may be used. When the digestion of milk is difficult it is well to add to it hot water or to dilute it with an alkaline or carbonated water. If the taste of the milk is unpleasant to the patient, it may be flavored by the addition of vanilla, nutmeg, coffee, tea, or cocoa in small amounts. After the first week or ten days the patient is allowed from one to two soft-boiled eggs each day, so soft that they can better be taken as a drink than eaten with a spoon, and flavored with a little salt. Well-boiled rice strained through a fine sieve, and even thin cornstarch or barley-gruel, if well cooked, may be given at this time with advantage, particularly if at the same time a little taka-diastase is used to aid their digestion. The author is firmly convinced that by this means terminal infections and general feebleness can be largely avoided and the patient brought to the stage of convalescence ready for speedy return to health and with greater vital force. Broths and other liquid animal soups are inadvisable, for they are good culture-media, and often tend to increase tympanites and diarrhœa. They are largely used by many physicians, but never by the writer. When curds appear in the stools, the quantity of milk should be diminished or it should be peptonized, or its digestion aided by the use of pancreatin given after it is taken. The use of 5 to 10 grains of citrate of soda in the milk will also prevent the formation of curds.

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[1] See article by the author in Therapeutic Gazette for March, 1898.

The use of cows' milk in large amounts for a considerable time has been shown to increase the coagulability of the blood, as cows' milk contains an excess of calcium salts. Wright has suggested that this is one of the reasons why thrombosis is so common in typhoid fever. To prevent the increase in coagulability a mixed diet should be given and a few grains of citric acid be administered daily for a few days at intervals of a week.

MEDICINES.—Drugs are not to be given if they can be avoided—that is, they are not to be used unless they are certainly needed to combat some definite condition which should be alleviated. In the great majority of cases, if not in all, the so-called antipyretic drugs are not only useless but harmful, and particularly harmful if their use is resorted to simultaneously with bathing. Their only justifiable use in a case which can be properly nursed and bathed is for the purpose of relieving headache and backache, when they may be given in small doses, such as 2 grains of acetanilid three or four times a day. Quinine is of little, if any, value except as a tonic in small doses.

Stimulants are to be used when the pulse is actually weak and the cardiac first sound distant or feeble. The best of them is whiskey or brandy, diluted with milk or water, and given in doses of half an ounce every three to six hours as needed. Many cases do better without any stimulation, whereas others need much larger doses of alcohol than those just named. Digitalis is rarely of any service because it does not act well in the presence of fever, rarely supports the degenerated muscle fibres of the heart, and is apt to disorder the stomach. When the cardiac condition is desperate, Hoffmann's anodyne in drachm doses every two hours in cool water is very valuable. When profound adynamia develops and the patient is critically ill, much good may result from the injection hypodermically of ½ grain of camphor in 30 drops of sterilized olive oil every eight hours for five or six doses. Another method of value when the vascular system is relaxed and the patient adynamic is the use of hypodermoclysis, adding to the pint of a 0.9 per cent. saline solution 1 drachm of adrenalin chloride solution of the strength of 1:1000. Strychnine may also be used, but it is a mistake to employ it for more than a few doses in the active stage of this disease. It is better to keep it in reserve for attacks of sudden circulatory failure.

ANTISEPSIS.—Absolute intestinal antisepsis can not be produced by any known means, although it is possible to modify very materially the growth of micro-organisms in the bowel by the use of proper remedies. If the physician takes the ground that by the use of these substances he destroys the *Bacillus typhosus* and so benefits the patient, he is largely in error, and his use of them is not rational because the bacillus in the early stages at best is not free in the bowel, and in the later stages is widely distributed in every part of the body. If, on the other hand, these remedies are given to combat intestinal fermentation, as shown by foul-smelling stools and tympanites and other evidences of an excessive growth of the non-specific bacteria which throng the bowel during the progress of this disease, his use of them is rational in that by this means other toxic materials are prevented from being generated in excess. So far as we know, the most active and harmless intestinal antiseptic is acetozone given in the daily dose of 30 grains dissolved in ½ gallon of pure water flavored with some one of the volatile oils, but not with sugar. This quantity may be given the patient as a drink in the course of one or two days. Another is the sulphocarbolate of zinc in the dose of 2 to 3 grains in pill form three or four times a day. Still another drug of far older use is turpentine in emulsion in the dose of 10 to 20 drops three or four times a day. The latter I prefer. In many of these cases also the use of a few small doses of calomel or salol is advantageous.

TREATMENT OF SPECIAL SYMPTOMS.—*Constipation* is to be relieved preferably by the use of enemata of soap and water, to which may be added in obstinate cases a teaspoonful or two of glycerin. Many of these patients have no constipation in the sense that the ileum or colon is sluggish; but, on the other hand, the sigmoid flexure becomes packed with hardened feces, and mechanical obstruction occurs. The use of purgatives by the mouth is therefore useless unless very strong drugs are used, which are dangerous. If it is thought that the bowels are really sluggish a little cascara sagrada (20 to 30 minims of the non-bitter extract) may be given each evening.

Diarrhœa, if excessive—that is, more than three or four stools a day—may be controlled by 5 to 10 drop doses of aromatic sulphuric acid in simple elixir or by adding to these two ingredients a half drachm of fluid extract of hæmatoxylon. If much fermentation is present, an intestinal antiseptic should be used, such as zinc sulphocarbolate or acetozone.

Vomiting is to be primarily prevented by regulating the diet as already referred to. If it persists, as little food and drink should be given as possible for a few hours to let the stomach rest; and if there be much nervous irritability, 60 grains of sodium bromide in a little starch-water should be given by the rectum to quiet the vomiting centre. Counterirritation should be applied over the epigastrium in the form of a mustard plaster or turpentine stupe. If alcohol is being used as a stimulant its use must be stopped, or, if this is impossible, then a very old brandy or wine should be substituted for the whiskey and given in very small quantities.

For *tympanites* a turpentine stupe is to be placed over the belly, if possible, before the gas accumulates in any amount, and if it persists a rectal injection of the emulsion of asafœtida, with or without a drachm or two of turpentine, should be given. The efficiency of this injection may be much increased in the way of expelling gas, and if marked adynamia is present, by adding half an ounce of Hoffmann's anodyne to the injection. Turpentine in the dose of 10 drops, in emulsion or capsule, may also be given by the mouth for this condition. When the gas fails to come away, its passage may be aided by the introduction of a long rectal rubber tube.

Hemorrhage from the bowel does not offer very much opportunity for direct rational treatment. In the majority of instances the best we can do in the way of real benefit to the patient is the maintenance of body heat by the application of hot bottles; and if the circulation becomes markedly feeble, the employment of normal salt solution by hypodermoclysis, a pint of it being given once, twice, or thrice in the succeeding twenty-four hours, according to the needs of the patient. Bandages may be applied to the limbs to limit the circulation to the vital parts, and the foot of the bed be raised for a similar purpose. The large number of remedies which have been suggested for the direct control of the hemorrhage indicate how feeble they all are. There is no more reason for supposing that astringents given by the mouth can check hemorrhage from an ulcerated vessel in the bowel than that they can check a hemorrhage from a branch of the anterior tibial artery; and when they are given and hemorrhage ceases, the arrest is due more to coincidence than to the effect of any drug. If any remedy of this type is of value, it is probably Monsel's salt (ferri subsulphas), which should be given in a hard pill or compressed tablet enclosed in a capsule, with the hope that it will escape from the stomach into the intestine without being dissolved, and thereby exert its styptic influence. Of course, if it is dissolved in the stomach, its chemical characteristics are altered. Many physicians apply a small ice-bag over the centre of the belly to influence the circulation in the small intestine, with the hope that in that way hemorrhage will be controlled. There is no objection to this plan of treatment, and the author often resorts to it; but it should be used with caution, if the hemorrhage is severe, lest it aid in devitalizing the patient by abstracting heat. Simultaneously with the application of the ice-bag to the belly, hot bottles should be applied to the other parts of the body, for it is to be remembered that the loss of bodily heat is an important factor, not only because the vital processes can not be well performed at a low temperature, but also because the sudden reduction of temperature caused by the hemorrhage deprives the heart and other organs of the stimulating effect of the fever which has been present for days. Another popular method of treatment is the administration of a pill containing a grain of opium and a grain of acetate of lead; the opium being expected to diminish peristalsis and so aid clotting, and the lead to act as a styptic. The opium is probably of value, but it is doubtful if the lead ever reaches the bleeding spot without becoming altered by the gastric and intestinal juices. When there seems to be continued oozing of blood from a large intestinal ulcer without free hemorrhage, the administration of turpentine and the use of adrenalin chloride is to be seriously considered, as there is reason to believe that they both control capillary hemorrhage. Wright and his co-laborers have shown that the hypodermic use of calcium lactate and the internal use of calcium chloride increase the coagulability of the blood and these salts may be used. (See Purpura.) After the hemorrhage has ceased, particularly as convalescence is begun, small doses of iron should be administered to combat the anæmia.

The *treatment of perforation* of the bowel from a medical point of view consists in giving opium to relieve pain, and employing heat and stimulants to combat shock. If a skilled abdominal surgeon can be obtained, all such cases should be operated upon at once, since the mortality under operation is less than with no operation. Statistics seem to show a mortality of about 80 per cent. with operation and 95 per cent. without it. My colleague, Dr. Keen, has published 158 cases, which were all he and Dr. Tinker could find in literature up to January 1, 1900. They gave a recovery percentage of 23.41. I have collected 54 cases which have been reported since January 1, 1900, and find that 35 of the number were followed by recovery. This gives a recovery percentage of 61.54, which is far too high for the general run of cases. Harte and Ashhurst place the recovery after operation at 26 per cent. Among children the results have been excellent. Out of 25 cases collected by Elsberg 16 recovered, a postoperative mortality of only 36 per cent. These figures are of value as showing that recovery may take place, but they do not give the real percentage of deaths, for most of the cases that are operated upon and die are not reported. I have seen 3 of these within a year. To be successful the operation ought to be performed at the earliest possible moment after perforation, although if the patient when seen is profoundly shocked it may be necessary to rally him by stimulation before the operation is commenced or even postpone operation until sufficient time has elapsed to allow him to rally.

Statistics clearly prove that the prospect of recovery from perforation treated by operation steadily diminishes with each hour that passes after the accident occurs. Cases are on record, however, and I have seen more than one, in which perforation took place and recovery occurred without operation.

Persistent insomnia is rarely a troublesome symptom in typhoid fever. Although patients complain of wakefulness at night, careful observation will usually show that they get sufficient sleep in twenty-four hours. In some cases, however, when they are wakeful, largely because of active delirium, and are rapidly exhausting their vital forces by continued nervous activity, life can be saved by the hypodermic injection of $\frac{1}{4}$ grain of morphine, to which may be added $\frac{1}{100}$ grain of nitroglycerin to prevent secondary nausea and depression.

The application of an ice-bag to the head throughout the attack will usually prevent ordinary delirium from becoming excessive.

Bed-sores are usually prevented by the friction applied to all portions of the skin in the baths which are given every few hours. If they appear over the sacrum, the patient should lie as much as possible on his sides, all bony prominences on which the patient rests being protected from contact with the bed by circular air-cushions. When the skin first reddens the irritation may be allayed by painting it with a solution of nitrate of silver, 20 grains to the ounce. If the bed-sore has begun to form, a useful dressing consists in equal parts of powdered chloretone and boric acid. If the slough becomes large, all that portion which is actually dead should be cut away, the part thoroughly sprayed with peroxide of hydrogen, dried by the gentle application of soft lint, and then dressed with the dusting-powder just named.

The treatment of the other complications of typhoid fever will be found under the headings of the respective diseases, such as Pneumonia, etc.

During *convalescence* the patient should be fed with increasing quantities of nutritious, easily digested food, but stimulants if possible should be avoided. If the patient is out of bed care should be taken that food is not ingested until after he has lain down to rest, in order that he may not be tired and so lack nervous energy during the progress of the digestive processes.

As already pointed out when discussing the prevention of typhoid fever, urotropin or uritone should be given in doses of 5 grains three times a day in water, to destroy the bacilli which are usually present in the urine.

The ordinary diet should not be restored until from two to three weeks after all fever has ceased. The author has often been impressed with the fact that the use of green vegetables, such as lettuce, spinach, asparagus, and similar substances, seem to exercise a most valuable influence in convalescence in typhoid fever, perhaps because they antagonize scorbutic tendencies.

PARATYPHOID FEVER.

Paratyphoid fever is a disease caused by infection with paratyphoid bacilli, and presents a symptom-complex closely resembling or indistinguishable from that of typhoid fever.

Bacteriology.—The paratyphoid bacilli belong to a group of organisms intermediate between the *Bacillus typhosus* and *Bacillus coli communis*. Buxton has shown that by appropriate methods organisms occupying this position may be divided into several groups; one resembling the colon bacillus, for which he proposes to use the name paracolon; another group closely allied to the typhoid bacillus, called the paratyphoid, and producing the condition termed paratyphoid fever. The last-named group may by appropriate cultural methods be further divided into a species culturally unlike the paracolon type, and a second distinct species resembling the paracolon group.

Pathology.—Differing anatomically from typhoid fever in essential details, this disease possesses no characteristic morbid anatomy, resembling in this respect the other forms of septicæmia. The most constant change is splenic enlargement, which was present in all of the cases coming to autopsy. In 3 cases ulcers were present in the intestine, but they resembled the ulcers of dysentery more than those of typhoid; in 2 cases the ulcers were numerous; in 1 case with abundant hemorrhage the most extensive ulceration was observed. The patches of Peyer and the solitary follicles are but slightly if at all changed. The mesenteric glands are but slightly altered; focal necroses, not containing endothelial cells, occur in the liver. The proliferative and phagocytic endothelial changes of typhoid are practically absent. There are no constant changes found in other organs, although toxæmia in severe cases may induce its usual anatomical changes. Gütig examined the blood of six paratyphoid patients and found that the neutrophiles are diminished during the early stages of the disease. The lymphocytes also are diminished at first, but become increased later in the disease, and in convalescence they constitute more than half the entire number of white cells. The eosinophiles disappear from the blood during the fever, but reappear just before or shortly after the temperature becomes normal.

The bacilli have been found in the heart's blood, in a cardiac thrombus, in the liver, lungs, spleen, adrenal bodies, cerebral cortex, and in the fluid of pericardial and pleuritic effusions.

Cases have been reported from France, Germany, Holland, Roumania, England, the United States, and the Philippine Islands. The disease affects chiefly young adults, and, like typhoid fever, is more prevalent in autumn than at other seasons of the year. V. Sion and V. Negal and De Feyfer and Keyser found that the epidemics under their observation were produced by drinking infected water. Hunermann traced several cases occurring among children to one of their number who had suffered from a mild infection. Of the other recorded cases nothing definite concerning the mode of infection is known.

Symptoms.—The disease may be mild or severe. The onset is gradual, and the symptoms of invasion are the same as those met with in typhoid fever, namely, *headache*, *lassitude*, and *slight bronchitis*. During the course of the disease the spleen becomes enlarged, rose spots may appear, and the patient develops the typhoid state. In Cushing's case a relapse occurred. Johnston states that *diarrhœa* and termination of the fever by crisis are of more frequent occurrence than in typhoid fever. Brion found diarrhœa in 18 per cent. of the recorded cases. In uncomplicated cases no leukocytosis has been observed. *Hemorrhage* from the bowel occurred in 5 per cent.

Complications.—Pratt has published a list of complications which includes all that have occurred in the 84 recorded cases. He divides them into three categories, viz.: 1. Those occurring in cases caused by *Bacillus paratyphosus α*. 2. Those occurring in cases caused by *Bacillus paratyphosus β*. 3. Those occurring in cases in which the species of the causative organism was not determined. He has shown that the complications of paratyphoid fever are of about the same character and frequency as those of true typhoid fever.

Diagnosis.—The diagnosis depends upon the isolation and cultivation of a paratyphoid bacillus from the patient's blood, urine, or feces, or from a localized lesion. Bacilli thus obtained should be agglutinated by the patient's blood serum, and the latter should agglutinate known paratyphoid bacilli. In case an organism cannot be recovered Pratt thinks a diagnosis of paratyphoid is justifiable if the patient's blood serum agglutinates known strains of paratyphoid bacilli. While the Widal reaction is usually negative, or positive only in very low dilution, Johnston believes that all cases in which it is negative must not be regarded as cases of paratyphoid rather than true typhoid fever.

Prognosis.—The prognosis would seem to be favorable, as only 3 deaths have occurred in 83 undoubted cases of the disease, and 1 of these was a mixed infection with the *Bacillus typhosus.*

Treatment.—This is identical with that of true typhoid fever.

TYPHUS FEVER.

Definition.—Typhus fever is an acute, infectious, self-limited disease of sudden onset which is characterized by fever, mental apathy, and the development of a rash which does not recur in crops as does the rose rash of typhoid fever. It is particularly prone to attack large numbers of persons in unhealthy surroundings, and is distinctly a disease of starvation or overcrowding. The period of incubation of typhus fever as a rule varies from eight to fourteen days, but there can be no doubt that many cases have been attacked within seven days after exposure. A few cases are said to have developed as early as the fourth day. Typhus fever is one of the most contagious of the acute infectious maladies.

Some confusion has arisen in the past between typhus and typhoid fever, but at present, they are clearly differentiated, although it is worthy of note that the symptoms of enteric fever are so much like typhus fever in their degree of adynamia that it is called "typhoid" or like typhus, while German writers of the present day still call typhoid fever "*ileotyphus.*"

Typhus fever is sometimes called Spotted Fever, Ship Fever, Putrid Fever, or Hunger Typhus.

The infection probably reproduces itself solely in the body of the infected patient, and is spread by direct contact with the patient or by his garments or discharges. It is not transmitted any great distance through pure air.

History.—Typhus fever was first described as occurring in 1083 by Corradi, but it was not fully recognized as a distinct malady till 1546, when Fracastorius wrote of the affection as he had seen it in Verona in 1505 and 1508. Several epidemics are reported as having occurred during the last half of the sixteenth century, in the eighteenth century, and in the early part of the nineteenth century in various parts of Europe; a most virulent epidemic ravaging Ireland and England in 1846. In America it first appeared in the New England States in 1807, and in Philadelphia in 1812, where it is said to have existed in isolated cases until 1836.

Distribution.—Typhus fever seems to occur in all parts of the world if the conditions favorable to its development are present in the sense of unhealthy surroundings and provided the necessary germ is introduced. Because of its intimate association with unsanitary conditions it has been epidemic in great armies, during famine, and on ships in which the crew or passengers often were huddled together for long periods of time. On the continent of Europe it spreads usually from east to west and is disseminated chiefly by the poorest classes when they travel from place to place. Persia, China, Hungary, and Turkey are never free from typhus fever, and small epidemics occasionally arise from these sources. Sometimes small epidemics or sporadic cases arise without it being possible to find any source of infection. The disease is exceedingly rare in the United States, but the possibility of its occurrence must always be borne in mind by quarantine officers and physicians in charge of large hospitals in crowded cities.

Etiology.—As already stated, typhus fever is a malady which depends upon a specific cause and the presence of unsanitary conditions for its development. The specific contagion, whatever it may be, retains its virulence for long periods of time in garments and in furniture. Much difference in its infectiousness also exists, for in some epidemics nearly everyone exposed is taken ill, whereas in others but few are affected. Kelsch has advanced the theory that the micro-organism of the disease is one which is usually benign, but may become malignant under favorable states. The specific micro-organism has never been isolated, although several investigators have claimed its discovery.

Exposure for a considerable period of time to the atmosphere of a room which is poorly ventilated and which contains typhus patients is the most effective way of contracting the disease, whereas if ventilation is good and the exposed person in perfect health there is much less danger of infection. When a large number of cases of typhus fever are grouped together in a ward, the infection becomes very virulent and both the attendants and the physicians are extremely prone to contract the disease. Now that vaccination, antidiphtheritic serum, and antiplague inoculations protect the physician and nurse from these infectious diseases, there is no malady so apt to attack them and against which there is so little prophylactic aid as typhus.

The most infectious period of the disease is in the early stages and at the height of the fever, although Moore asserts that it is most contagious during convalescence. It is also infectious even during the stage of incubation. Mild cases are probably as capable of spreading the infection as severe ones. After the febrile condition has passed away there is reason to believe that the patient ceases to be a direct source of infection, and if an attendant who comes in contact with him for the first time now contracts the disease, it must be from the poison which has found lodgement in the clothing of the patient during his illness. So far as we know, the most common means by which infection gains access to the body is by the organs of respiration and perchance by the skin. Indeed, some clinicians of experience assert that actual contact between the body of the patient and that of the attendant is necessary for infection to take place, but this is not generally conceded. Certain it is that a very brief period of exposure is sufficient for the transmission of the disease. The infectious agent or agents of typhus fever is rarely if ever carried by water or other liquid media. Lowered vitality of the individual naturally increases his susceptibility. The influence of age and sex is very slight, for all ages, after early infancy, seem equally susceptible, the greater predominance of the malady between twenty and forty probably being due to increased opportunities for exposure.

The influence of climate and season upon the spread of typhus fever is only an indirect one, in that the poor ventilation of the houses of the lower classes during the winter months aids in the dissemination of the disease among the occupants, whereas in summer the better supply of fresh air and the greater amount of out-door life tends to diminish the danger of infection.

Prevention.—There is no disease in the prevention of which fresh air plays so large a part as it does in typhus fever. Indeed, it may be stated that if a healthy man be supplied with plenty of fresh air while in the presence of the sick he will have a fair chance of escape, whereas if the air of the room be impure infection is almost certain, for, as already stated, typhus fever is a malady of darkness and poor ventilation. Practical experience seems to indicate that the various disinfectants usually employed have little value in preventing its spread unless they are used in concentrated form upon the garments which have been infected. By far the best means of preventing the spread of typhus fever are the admission of a plentiful supply of fresh air and sunshine, the application of steam or scalding water to all woodwork and clothing, or the use of dry heat if steam heat cannot be employed. The bed-clothing and mattresses should be destroyed by fire. The value of formaldehyde gas as a disinfectant is still undecided for preventing the spread of this disease.

Pathology and Morbid Anatomy.—Typhus fever, unlike typhoid fever, has no peculiar morbid anatomy, and it is therefore impossible from autopsy findings alone to determine that the cause of death has been typhus. The skin, it is true, may show very soon after death numerous petechiæ, and early decomposition constantly occurs after death from this disease. The body is usually not greatly emaciated because the disease lasts so short a time. The muscles, which are somewhat dry, may also show, when examined under the microscope, signs of granular or fatty degeneration just as they do in typhoid fever.

The respiratory passages may be inflamed or congested. Thus there may be laryngeal ulceration as in typhoid fever and a considerable degree of bronchitis. Often hypostatic congestion of the lungs is present. In other instances a true lobar pneumonia occurs. Indeed, Curschmann says it occurred in 15 per cent. of his cases. The heart muscle is friable and suffers from the form of myocarditis seen in all infectious diseases, and the blood is found to be darker and more liquid than normal. It is a noteworthy fact that the intestines show no lesions whatever either in Peyer's patches or in the solitary glands. If such lesions are present the disease is typhoid, not typhus, fever. The liver is usually swollen and is found to be the seat of cloudy swelling, while the spleen is also enlarged to some degree but very soft. It may contain infarcts. The kidneys are also the seat of cloudy swelling.

Symptoms.—The symptoms of typhus fever are quite characteristic. Unlike those of typhoid fever, the *invasion is usually abrupt*, the patient suddenly feeling ill about twelve days after exposure, and being seized by a *chill* or series of *chills*, with *headache, backache*, and *general prostration*. The *fever* rises rapidly, reaching its acme it may be as early as the second day, but as a rule it rises steadily during the first four or five days, and during this period of rise the characteristic morning remissions of typhoid fever do not occur to so marked a degree. By the fifth day the temperature often reaches 105° or even 107°, but when the infection is not severe it may not go above 103°. After having reached its highest point it remains fairly constant with a slight decrease each morning, the evening temperature, however, remaining high until the period of crisis, at about the fourteenth day. In cases characterized by severe infection the temperature may continue to rise until it reaches as high as 109°, when death usually occurs. To those cases in which the temperature reaches this very high point within the first few days of the illness the term *Typhus Siderans* is usually applied.

The *pulse* is full and rapid, but usually not so feeble as in the early stages of typhoid fever. The *face is deeply flushed* the conjunctiva congested, and the expression one of apathy, although in some cases delirium varying from mild wandering to actual maniacal violence may be met with. Sometimes the delirium is wild (delirium ferox), sometimes it is like that of delirium tremens, and sometimes it is low and muttering—the typhomania of Galen. If the toxæmia is severe extreme prostration and feebleness may ensue by the tenth day.

In from three to five days the *eruption* appears upon the abdomen and chest, and soon spreads to the legs, arms, and face. It possesses two peculiar characteristics. In the first place it is papular, as in typhoid fever, but the spots are rarely as rosy as they are in that disease, and may finally become as dark as actual petechiæ, which indeed they really are. The second peculiarity is that there seems to be a subcuticular eruption or area of congestion, or mottling, so that the skin is marbled or mottled. Unlike the rash of typhoid fever, that of typhus does not disappear on pressure, and is distinctly manifest after death. Even if the case is mild the petechial character of the rash is present. In some instances the skin is said to give off a peculiar, musty or mouse-like odor. In children the rash may be so profuse as to resemble an attack of malignant measles.

As the disease advances to the second week the evidences of toxæmia become more marked. The *active delirium* which perchance was present at first is replaced by *stupor* and *coma vigil* with subsultus tendinum, the tongue is dry and heavily coated, the teeth covered with sordes, and the heart's action rapid and feeble. The respirations are quickened but shallow, and diarrhœa may be marked. This stage is called the "putrid," "malignant," or "typhoid" stage of the malady. It is essentially one of profound toxæmia, and the patient lies in a state of deep prostration, indifferent to all sounds and objects in the immediate vicinity, mumbling a few disjointed sentences, his tongue being so coated and dry that it is almost impossible for him to move it. The *pupils* are often strongly contracted and the tendons twitch, while there may also be carphologia, or picking at the bedclothes. Finally, if the illness becomes more severe, the patient lies with open eyes, gazing into space, with dilated pupils, a thready, imperceptible pulse, and a cold, clammy skin, which heralds the approach of death, which is due to the toxæmia, asthenia, and hypostatic pulmonary congestion.

If the patient survives the early stages of attack, the fever usually ends by the twelfth or fourteenth day and the temperature undergoes much more rapid defervescence than it does in typhoid fever. Indeed, it is generally thought that the fever ends by crisis; so that the patient passes, during a prolonged sleep, from a state of severe illness with a clouded mind to early convalescence with a clear mind, a *critical fall of temperature* taking place. This remarkable change in the aspect of the case has been alluded to by some authors as quite pathognomonic of typhus fever, but the statement that the fall of the temperature is always by crisis is not universally conceded to be correct. Thus, Moore, of Dublin, states that the end is by crisis; while Curschmann asserts that in the great majority of cases it ends by lysis, although he admits that a critical fall occasionally takes place, covering a period of from two to three days. This is hardly a crisis in the usual acceptation of the term.

Whatever may be the true method of the fall of temperature, it is certainly a fact that convalescence is rapidly established; so that the patient proceeds to complete recovery more rapidly than after typhoid fever, health being completely restored, it may be, by the end of a month.

Relapse in typhus fever very rarely takes place, and in the vast majority of cases one attack produces immunity against further infection.

The *complications* of typhus fever are those which we would expect to meet with in the presence of any severe infection. Bronchopneumonia or lobar pneumonia may occur. In very poorly nourished individuals noma may develop, and symptoms of generalized paralysis develop as the result of neuritis. So, too, septic arthritis and infection of the parotid glands may occur.

Diagnosis.—Several characteristics of typhus fever have already been emphasized. The most noteworthy of these are: the sudden accession of the disease, the rapid rise of temperature without morning remission, the development of a peculiar rash between the third and fifth day, the dusky appearance of the face, the musty odor of the skin, and the early appearance of active delirium or profound apathy. All these symptoms are quite different from those met with ordinarily in typhoid fever, but it is not to be forgotten that sometimes typhoid fever begins suddenly and presents manifestations closely resembling those of typhus; so that during the presence of an epidemic of typhus or typhoid fever cases of either one of these diseases may readily be overlooked. In the differentiation the early development of the rash (third to fifth day) in typhus fever is of great practical value, and the distribution of the rash is still more helpful in aiding a decision; for the rash of typhus fever, if profuse, involves the extremities as well as the trunk, whereas that of typhoid fever is chiefly limited to the body; a profuse and dusky rose rash on the hands and legs is therefore distinctly in favor of typhus fever. Again, the rash of typhus fever is constant, whereas that of typhoid fever fades and recurs in crops. Sometimes however, the rash of typhus fever, like that of typhoid fever, is very scant, only a few rose spots being present. Indeed, the disease may occur without any exanthema being manifest.

As the illness progresses much additional differential information can be gained if the case be one of typhoid fever by the discovery of the Widal reaction, the recovery of the *Bacillus typhosus* in the blood and in the rose spots. Then, too, typhoid fever does not end so abruptly nor so early as does typhus fever. Malignant measles and variola may in their earliest stages resemble typhus, but their later course clearly separates them.

Relapsing fever is separated from typhus fever by the clear mental condition of the patient notwithstanding his high temperature, by the lack of petechiæ, and the absence, as a rule, of severe initial symptoms.

Prognosis.—The prognosis in typhus fever varies greatly with the previous condition of the patient, and also to some degree with the severity of the epidemic. Usually the mortality rate varies from 10 to 20 per cent. in young adults, but in children it is often much less than this. In advanced years the mortality is very high.

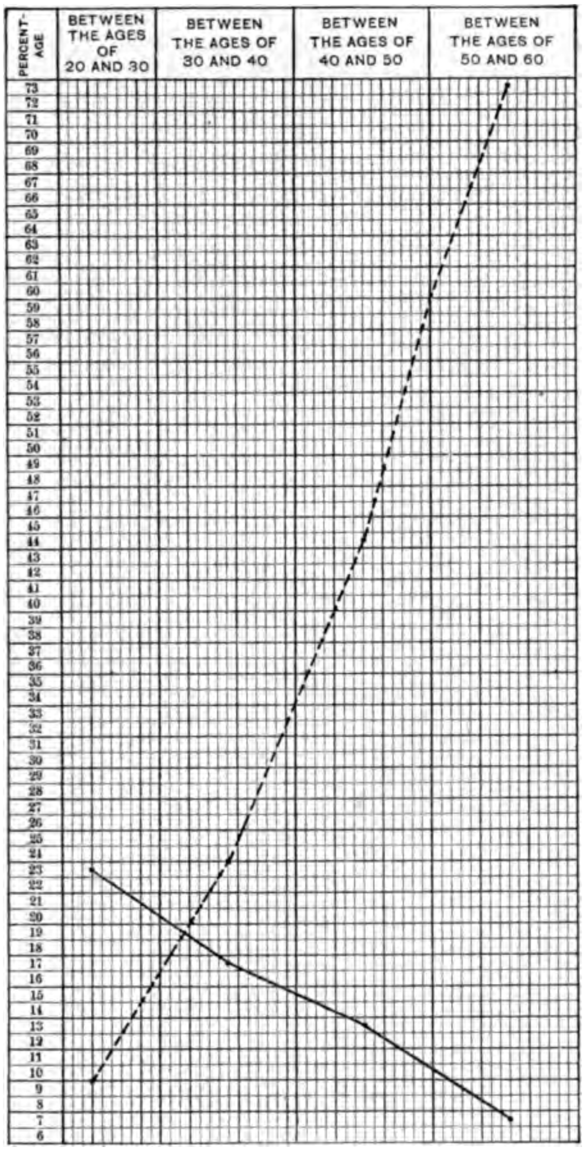
Curschmann has stated that "old age makes itself felt as early as the fortieth year and that after fifty almost 50 per cent. die." The accompanying chart, Fig. 14, made from the statistics of Murchison, Guttstadt, and Curschmann indicates further the influence of age on the prognosis.

Death in typhus fever rarely occurs before the second week. After the end of the second week it seldom takes place except as the result of some untoward complication.

Treatment.—The treatment of typhus fever is in many respects identical with that now recognized as useful in typhoid fever. The patient should be isolated, of course, and provided with an abundance of light and air. As already stated, in no disease are these aids to health more essential for recovery. As the course of the malady is one toward profound asthenia, easily assimilated or predigested foods should be given as freely as the patient can utilize them. Milk to which is added a little pancreatin and sodium bicarbonate, barley- and rice-gruel in which is placed some takadiastase, and copious draughts of water to flush the kidneys and aid in the elimination of poisons are to be administered. The fever is to be treated by cool or cold bathing as the patient lies in bed, according to the directions given under typhoid fever, and cold is to be kept applied to the head continuously. The coal-tar antipyretics are not to be used if they can be avoided. When signs of cerebral and pulmonary hypostatic congestion manifest themselves the patient may be immersed in a bath of about 90°, and cold water at 60° poured over his head and shoulders as a douche, active friction of the body and limbs being performed by the nurse for several minutes before the sick man is returned to his bed. Should the circulation fail, alcohol in the form of whiskey or brandy, well diluted with water, is to be employed for the purpose of supporting the heart and nervous system. Camphor in $\frac{1}{2}$ grain doses is useful for this purpose. If the nervous restlessness of the patient is sufficient to endanger life by the resulting exhaustion, a hypodermic injection of morphine may be given to produce sleep or nervous quiet.

The bowels should be kept open by the use of gentle laxatives, or be evacuated by a saline purge if obstinately confined. The activity of the kidneys must also be maintained by the use of alkaline diuretics and sweet spirit of nitre and by the free administration of a pure drinking water. As retention of urine often occurs, the state of the bladder must be carefully watched.

FIG. 14



Showing the decreasing morbidity and increasing mortality percentage of typhus fever with advancing years. Solid line represents morbidity from Murchison's statistics. Broken line, mortality percentage from the statistics of Murchison, Guttstadt, and Curschmann.

RELAPSING FEVER.

Definition.—Relapsing fever, as its name indicates, is characterized by an attack of fever which lasts about six days, this in turn is followed by a period in which fever is absent, and this again by a recurrence of a period of fever. These alternating periods may be repeated three or four times. It is due to a specific micro-organism, the so-called spirillum or spirochæte of Obermeier. Sometimes it is called *Febris Recurrens*, "Seven Days' Fever," and "Famine Fever."

History.—The history of relapsing fever is, when compared to some other infectious diseases, fairly modern, for the first descriptions of it occurred in medical literature about 1729, although it was not until 1739 that Rutty gave a clear description of its course. After this for nearly a century no reports of its existence are to be found, but between 1842 and 1852 it appeared over a wide area, occurring in England, Ireland, Scotland, Germany, and finally in America, to which country it was brought by a shipload of immigrants who came from Liverpool and landed in Philadelphia in 1844. It became epidemic in the United States in the decade from 1861 to 1870, and it is interesting to note that as the American, Gerhard, first aided in the differentiation of typhoid fever from typhus in Philadelphia, so Pepper, Rhoads, and Parry, of the same city, have contributed to medical literature the best account of the disease as it has appeared in this country, having observed a larger number of cases than any other clinicians.

Distribution.—Relapsing fever has occurred in almost all parts of the civilized world.

Etiology.—It has been claimed that filthy surroundings and bad food are active in the development of relapsing fever, but they probably exercise a general influence by lowering vitality rather than by directly aiding infection. Sex, age, and nationality exercise no influence, and it is doubtful if any one season of the year increases the prevalence of the disease. The actual cause of relapsing fever, as already stated, is a spirillum known as the spirillum of Obermeier, which is constantly found in the blood of patients suffering from the disease during the stage of fever. It is absent from the blood in the intermissions, although small, glistening bodies, said to be spores, can be seen. The disease is contagious, that is it requires contact with the patient or with his garments for the infection to be spread. Patients may be infected by insects, for example by bed-bugs which have previously bitten patients suffering from relapsing fever. That the disease is ever conveyed by the air is doubtful.

Pathology and Morbid Anatomy.—The changes produced in the body by relapsing fever are not only not marked, but not at all characteristic. The spleen and liver are swollen and engorged, as in nearly all febrile infectious diseases, and the voluntary muscles may undergo granular degeneration. Similar changes may be found also in the heart muscle. Sometimes multiple infarctions and hyperplasia of the bone-marrow are present and ecchymotic spots, which are found ante-mortem, are seen in the skin and subcutaneous tissues.

Symptoms.—As a rule about six or seven days after exposure to the disease the infected person is abruptly seized by a *severe chill*, or more rarely by *headache* and *vomiting*. The face becomes flushed, but the expression is not dull and apathetic as it is in typhus or typhoid fever, unless the infection is very severe, when typhoid symptoms may soon develop. A moderate degree of *jaundice* also is present in many cases. No characteristic eruption appears on the skin, although small petechiæ or ecchymotic spots may

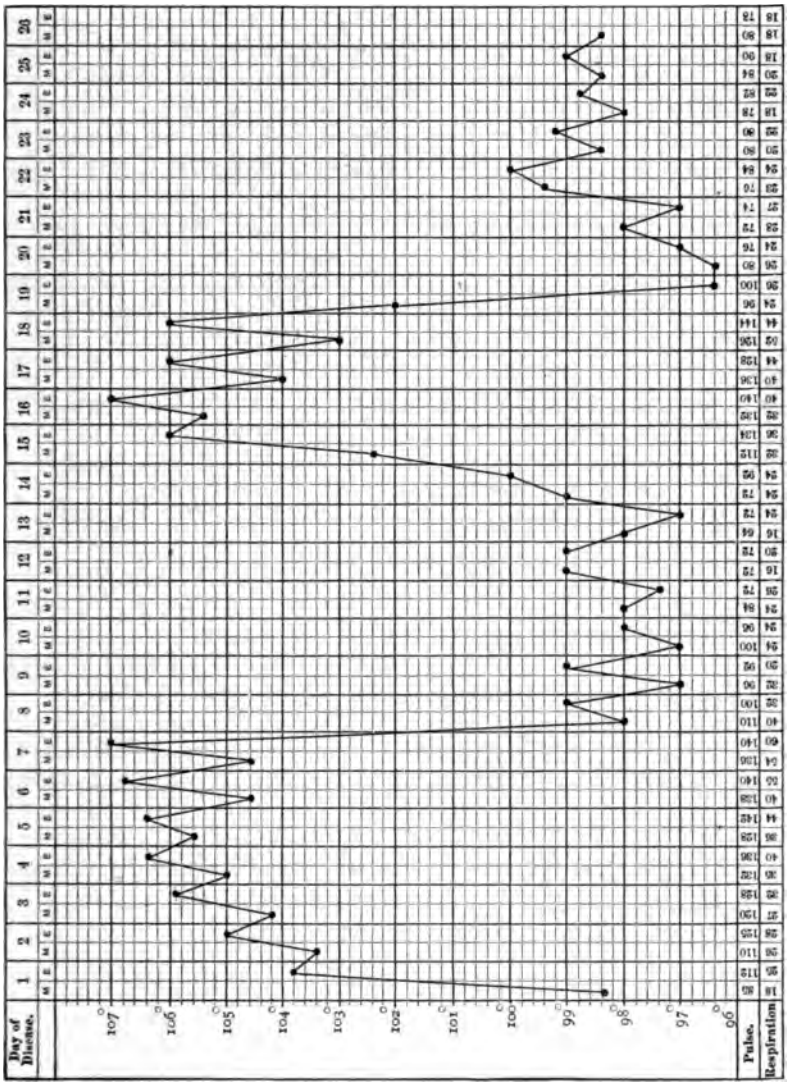


FIG. 15

Typical case of relapsing fever terminating in recovery. One relapse with slight post-critical rise in temperature.

occur in severe cases. Many observers have recorded the presence of a disagreeable, *musty odor* about the patient.

The *febrile movement* is the most notable manifestation of the disease. The fever begins to rise while the stage of chill is still young, and reaches 102° or 103° in the first twenty-four hours and 104° or 106° in the next twenty-four hours. During the febrile stage of about six days this level of temperature is fairly constantly maintained, although irregular remissions of 1° or 2° may occur. Sometimes the fever reaches 108°. In some cases the primary febrile period lasts only two days. While the febrile movement just described is characteristic, its ending is more so, for a critical fall of temperature takes place with a suddenness and completeness which is rarely met with in any other disease unless it be croupous pneumonia (Fig. 15). Not rarely it falls 6° in three hours, although a fall of 1° an hour is more common. The rapid fall may carry the temperature a little below normal. After a few days of no fever the *relapse* takes place with the same sudden onset as occurred with the primary attack. It runs a course in all respects like the original seizure, but it more commonly ends by lysis than does the first paroxysm. The third and fourth attacks, if they occur, are usually milder than the first two. The duration of the period of intermission varies from one to ten days, although it is usually six days or a week, and the duration of the entire illness may vary from eighteen to ninety days, according to the number of relapses.

The pulse during the early attacks is rapid, and it may be bounding, but if the patient be enfeebled by prolongation of the illness it may be small and compressible. Severe frontal and occipital headache is often experienced by the patient in the first attack, but delirium is rare except it be due to serious complications or to very high fever.

Prognosis.—The prognosis as to ultimate recovery is quite good, the mortality of the disease usually being about 4 per cent.

Treatment.—There is no specific treatment for relapsing fever. Good nursing, careful feeding, and the use of stimulants, if the patient is feeble, are of course needful. The action of the bowels and kidneys, as in all infectious diseases, should be carefully attended to. No results from the use of hydrotherapy in relapsing fever have been published so far as the writer is aware, but the course of the febrile movement scarcely indicates this plan of treatment.

VARIOLA.

Definition.—Variola, or smallpox, is an acute infectious disease affecting the entire body, but manifesting itself chiefly by the development upon the skin, more particularly that of the face and forearms, of an exanthem which is at first macular, then papular, then vesicular, pustular, and finally umbilicated.

History.—Smallpox is one of the ancient diseases, for records exist which show it to have occurred many centuries before the time of Christ. The first authentic medical record of the malady did not appear, however, before the tenth century, when Rhazes, of Bagdad, wrote his *Treatise on Smallpox and Measles.* It is generally considered that smallpox did not gain entrance to Europe till about A.D. 710, when the Arabs conquered the Spaniards. It reached Germany about the tenth century, at which time it also appeared in England. At times since the tenth century it has swept away thousands of persons in a single epidemic, and very few escaped its ravages. Indeed, a large part of the population of London were at one time pock-marked. It was first introduced into Mexico in 1520, and into Massachusetts in 1633. Until the introduction of vaccination it was one of the most death-dealing maladies known to man. (For the influence of vaccination in diminishing smallpox see article on Vaccinia.)

Distribution.—Smallpox has occurred in all parts of the civilized world, from the Arctic to the Tropics, and is of equal virulence in very cold and in very warm climates.

Etiology.—Variola is believed by some to be due to a parasite named by Guarnieri, in 1892, the *Cytoryctes variolæ,* and carefully studied by Wasielewski in 1901. Its evolution has been more fully known by the labors of Councilman, Magrath, and Brinckerhoff in 1903 and Brinckerhoff and Tyzzer in 1905, the latter being an extensive investigation of experimental variola and vaccine in Philippine monkeys. These in every respect confirm the previous findings in human beings. Basing his views upon previously accomplished work, but especially upon the study of Councilman and his students, Calkins has attempted to formulate the different stages in the life history of the parasite. A full review of these and previous inquiries into the nature of the specific organism of variola and vaccinia will be found in the *Journal of Medical Research,* February, 1904, vol. xi., No. 1, pp. 8-360 and January, 1906, vol. xiv., No. 2, pp. 209-359. (For the process of the development of this organism see Pathology and Morbid Anatomy.)

The disease affects persons who may be exposed to it at all ages, and remarkably few people who are unvaccinated are able to resist the infection, not more than from 1 to 5 per cent. The negro race is peculiarly susceptible, and in this race the rate of mortality from the disease is usually very high. Smallpox affects males more frequently than females. It is more common in the winter and spring than in the summer, perhaps because of the crowding in the homes of the poor during the cold months.

The contagion of smallpox is spread in several ways—viz., directly, that is, by contact with the patient's body and his clothing; and indirectly, by the air. Stokes has recently published a paper indicating that the infection usually enters the body through the lungs. A nurse may convey the disease from a patient to a healthy individual, and rats, mice, and flies may do likewise. The patient ill of smallpox is capable of infecting a healthy person from the initial stage of the disease to the moment when, recovery having occurred, every particle of pustule or desquamating skin has been cast off. The most contagious periods are, however, those of vesication, pustulation, and exfoliation.

The fact that the disease is spread by aerial convection is never to be forgotten, and it may be carried in this way from a few feet to several yards (Fig. 16). Much difference of opinion, however, exists among those who have studied the question of aerial convection. Power, of Fulham, and Barry, of Sheffield, England, found a noticeable influence exercised by the propinquity of a smallpox hospital, but Savill, from investigations carried on at Warrington, came to the conclusion that aerial currents influenced the spread of the disease but little. It must be remembered, moreover, that before we accept these figures as to aerial convection we must be sure that the contagion was actually carried by the air and not by insects or animals. I know of one smallpox hospital from which flies, mice, rats, and cats passed freely, and surrounding which smallpox was almost constantly present.

FIG. 16

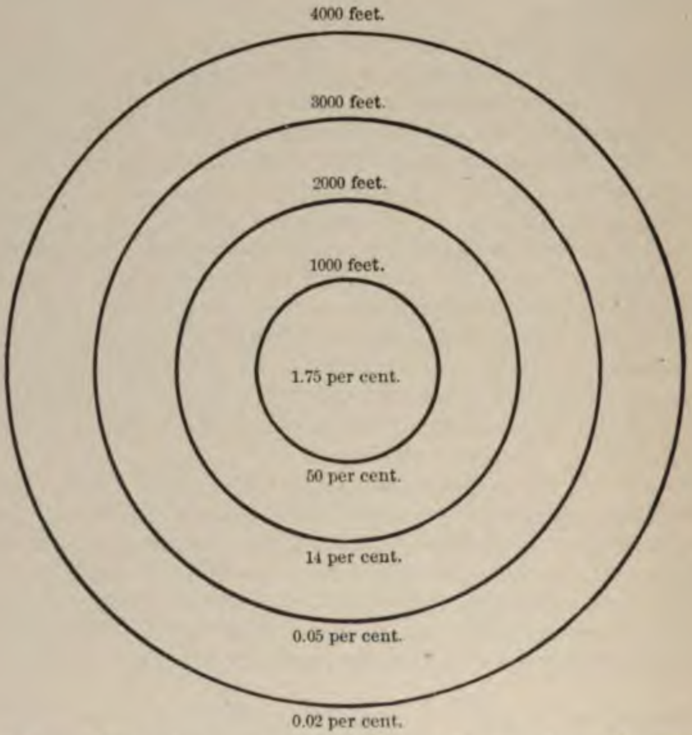


Diagram showing the percentage of aerial convection of smallpox. (Moore.)

Bodies dead of smallpox can also spread the disease among those who handle them.

The severity of the infection depends not so much upon the violence of the disease in the giver as in the susceptibility of the receiver of the malady. A mild case may therefore be provocative of most virulent epidemic.

Incubation.—The period of incubation of smallpox varies from five to twenty days, but as a rule it is about twelve days. Cases occurring in less than five days after exposure are very rare.

Prevention.—There is one measure above all others to be used in the prevention of smallpox, and that is vaccination, which by its beneficent influence has changed smallpox from a common and fearful scourge of mankind to a disease so rare that many physicians practice a lifetime without seeing a case. (See Vaccine and Vaccination).

It is very important to bear in mind the clinical fact that vaccination not only protects the patient who may be subsequently exposed to smallpox, but also that it protects the patient who, having been so exposed, is subsequently vaccinated. Even if the vaccination be performed so long after the exposure that smallpox nevertheless develops, the severity of the disease will be modified, the degree of modification being in direct ratio to the length of time between vaccination and the appearance of the variola.

A most interesting illustration of this has been sent me most kindly by Dr. Allan Warner, of the Borough Isolation Hospital, Leicester, England. The history of the cases is as follows:

A boy, aged fourteen years, unvaccinated, sickened with smallpox on April 14. He was removed to the hospital on April 18, where he had a severe confluent attack. The father consented to his wife and three children being vaccinated, stating that personally he would not be vaccinated, but would be a "test," to see if there was anything in it. Ten days later his daughter, aged three years, developed a smallpox eruption; she had less than one hundred spots and never appeared ill. No other person in the house suffered from smallpox except the father, vaccinated in infancy, his eruption appearing fourteen days after the son had been removed to the hospital. A photograph of the father and daughter, taken on the twelfth day of the father's eruption, may be seen in Fig. 17, and requires no comment.

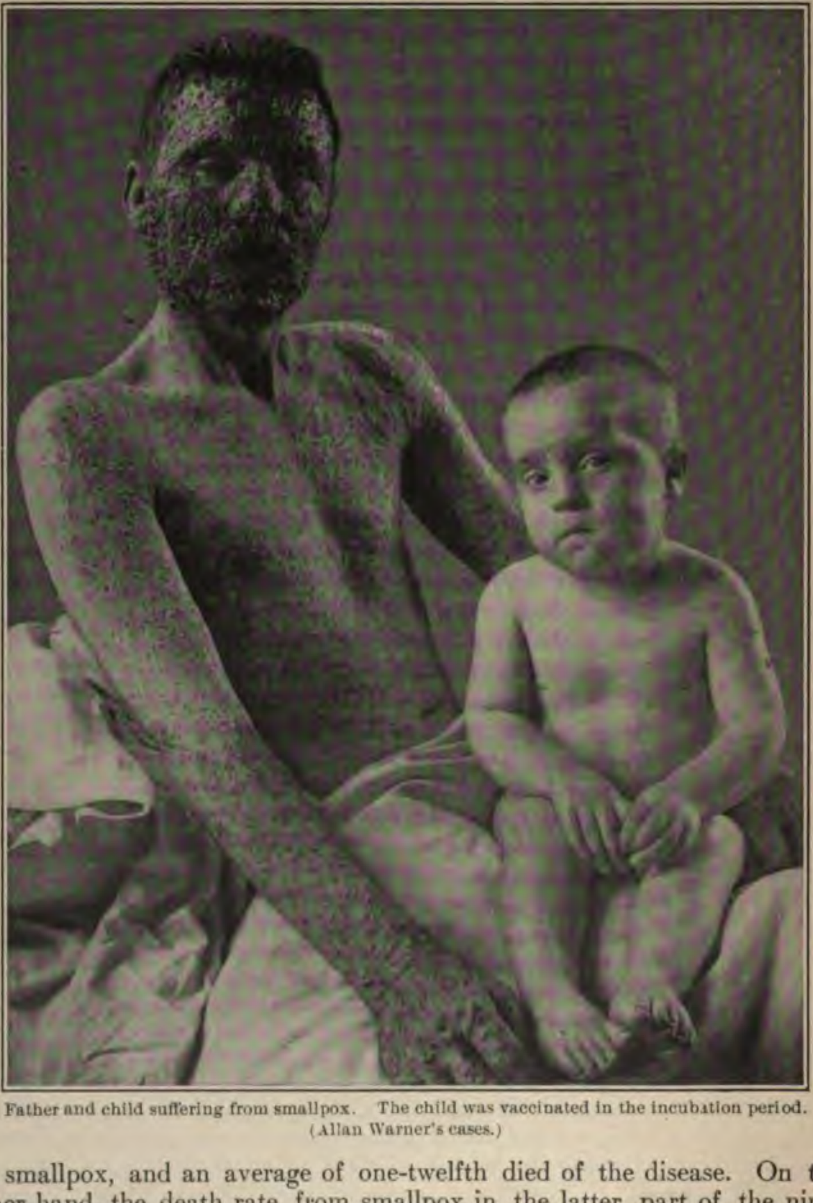
In cases of urgency it is generally held that humanized virus is more valuable than calf virus, but as humanized virus is often difficult to obtain it is better to vaccinate the patient in different places with glycerinated vaccine made by different manufacturers, since in this way there is little doubt but that one will surely take.

The second preventive measure of importance is the absolute isolation of the patient, and the third the complete disinfection or destruction of all garments and bed-clothing which have been about the sick person, including those worn by his attendants. Finally, all individuals exposed to the contagion should be quarantined for a period of twenty-one days, in order that the physician may be sure that they are not going to be attacked and so spread the infection.

Frequency.—Smallpox is so constantly present in the poorer part of large cities that it may be said to be almost endemic in all of them, but to a very moderate degree. Occasionally when a considerable number of unvaccinated persons have accumulated in a city or country district, the disease bursts out in a small epidemic, and sometimes, without any such apparent cause, certain districts seem to be affected, many unvaccinated persons being attacked. During the winter of 1901 and 1902 smallpox appeared almost all over the United States in scattered localities. It can, however, always be stamped out by house-to-house vaccination, and its spread depends upon imperfect quarantine and inefficient vaccination.

As an illustration of the extraordinary effect of vaccination and sanitation upon this malady it is interesting to note that during the eighteenth century fully two-thirds of all children born in Europe were sooner or later attacked

FIG. 17



Father and child suffering from smallpox. The child was vaccinated in the incubation period. (Allan Warner's cases.)

by smallpox, and an average of one-twelfth died of the disease. On the other hand, the death rate from smallpox in the latter part of the nineteenth century in London was 98.5 per cent. less than one hundred years before. To put it differently, the death rate from smallpox in 1838 was

1064 per million, while in 1889 it was 1 per million, and in 1890 nil per million.

During 1904 the disease was totally eradicated from New York and Philadelphia by vaccination and quarantine.

Pathology and Morbid Anatomy.—The most noteworthy lesion produced by smallpox takes place in the skin. The dermal papillæ become hyperæmic, the cells of the rete Malpighii swell and so raise the epiderm, and under this epiderm serum exudes and pushes the stratum still farther upward. The cells of the rete are more or less elongated, pigmented, and form fibrils extending from the epiderm to the base of the inflamed zone in the derma, constituting the vacuolar focal degeneration described by Councilman, Magrath, and Brinckerhoff. In this reticulum still further serous exudation occurs, and so forms a vesicle which increases at its margin, where the exudation takes place very rapidly, while degenerative and necrotic changes progress in the epithelium of the area involved. As a result the area under and around the vesicle becomes indurated and we have the characteristic hard pock of variola. The persistence of this free exudation at the margin of the pock and the greater density of the centre lead to depression of the latter, giving rise to umbilication. Wright has shown that the central depression in the pock may be due to diphtheroid degeneration. It may also be due to retraction by a hair or small gland. Councilman, Magrath, and Brinckerhoff do not believe that the pock is always produced by the same cause, but that a number of factors enter into its formation.

Following this stage, the serum in the pock is infiltrated with leukocytes, and these becoming great in number, the contents of the pock become opaque or turbid, and finally resemble pus. Sometimes if the inflammation in the adjoining pocks is very severe the deeper layers of the skin become involved, undergo necrosis, and so great local destruction of tissue takes place. After this stage epithelial regeneration progresses beneath the scab, which dries up and ultimately falls off, leaving a red or pink depression in the skin, which depends for its depth upon the degree of pustulation or necrosis present during the acute stage. Not only do vesicles form on the skin, but upon the mucous membrane of the mouth, pharynx, tongue, and even the rectum, anus, vagina, penis, and conjunctiva in some cases.

The changes just noted probably arise from the minute organism recently studied by Councilman and his assistants. He has described its development in the following words: "In the lower layers of skin epithelia, before there is present any anatomical evidence of vesicle formation, there are found small, structureless bodies from one to four microns in diameter. The cells present, at this time, little or no evidence of degeneration. The bodies, one or more in number, lie in vacuoles in the cells. The vacuoles are, at first, but little larger than the enclosed bodies. The bodies increase in size, and evidence of structure, consisting of granules more distinctly stained, and lying in definite spaces, begin to appear. With the increase in size of the body, the vacuole of the cell enlarges until a large, central space around the nucleus is formed. At this time there is but little evidence of degeneration of the nucleus. The body continues to increase and becomes granular, the granules lying in a reticular structure. The form of the larger bodies is irregular and suggests an amœboid character. They may become as large as, or larger than, the nucleus of an epithelial cell. A definite nucleus has not been made out in them, unless the reticular structure which stains more intensely than the rest of the body be so considered. At this time segmentation takes place, leading to the formation of small, round bodies about one micron in diameter. All this is best seen in the acute hemorrhagic cases. It may also be seen in the advancing edge of a young vesicle, the bodies becoming larger and more definite in structure as the formation of the vesicle advances. The bodies in the cells we regard as living organisms, and the gradual growth and final segmentation as a cycle in its life history. Up to this time the nuclei, although showing such evidences of degeneration as the massing of chromatin in the periphery, are not greatly altered.

"At the period of segmentation, and when most of the intracellular bodies have disappeared, small, round or oval, ring-like bodies appear in the nucleus. These increase in size and acquire a definite structure, consisting of a series of vacuoles around a large, central vacuole. The rim of the central vacuole stains more distinctly than do other parts of the body. One or more of these bodies may appear in a single nucleus. When several are present they are smaller, but have the same structure. The bodies become larger; the nuclear rim grows less distinct, and finally disappears, and the body lies in a completely degenerated cell, or this breaks down, setting free the body. With the growth and development of the intranuclear body the vacuolar structure becomes less evident, and finally a structure is formed which contains numerous fine vacuoles. At this time small, circular bodies begin to appear in it, and groups of these are surrounded by a faint ring, which probably represents the remains of the body in which they were formed."

Myocardial degeneration is present in most cases, and a variolous myocarditis has been described.

In general the cardiovascular changes of smallpox resemble similar alterations occurring in other infectious diseases.

Proliferative changes occur in hæmatopoietic organs (spleen, lymph nodes, and marrow), associated with the production of basophilic mononuclear cells which enter the circulation and also phagocytic endothelial elements. The basophilic mononuclear cells infiltrate the testicle and usually the kidney, liver, and adrenals.

Cloudy swelling occurs in the glandular viscera and a diffuse toxic degeneration takes place in the liver, kidneys, adrenals, and testicles.

Pharyngitis and fatal bronchopneumonia occur; less commonly croupous pneumonia and pulmonary abscess or gangrene develop.

Endocarditis, pleurisy, and empyema are infrequent.

The kidneys are more or less altered in all cases; in milder degrees this may amount to little more than intense cloudy changes, but in other cases acute diffuse, glomerular, or, less commonly, suppurative nephritis occurs.

Many of the lesions produced in the internal organs in smallpox are the result of the secondary infection from the skin and respiratory tract, and this usually depends upon the presence of the *Streptococcus pyogenes.*

As in most of the acute infectious diseases, bronchitis is present in many cases of variola, and the secretion of the bronchial mucous membrane may be profuse and mucopurulent.

Bronchopneumonia may also develop.

When hemorrhagic smallpox takes place we have transudations of blood into the pocks and into the conjunctiva, the retina, the muscles, the subpleural tissues, into all the abdominal organs, and into the kidneys and the perirenal fat.

Submucous extravasations also take place in all the organs of the body lined with mucous membrane.

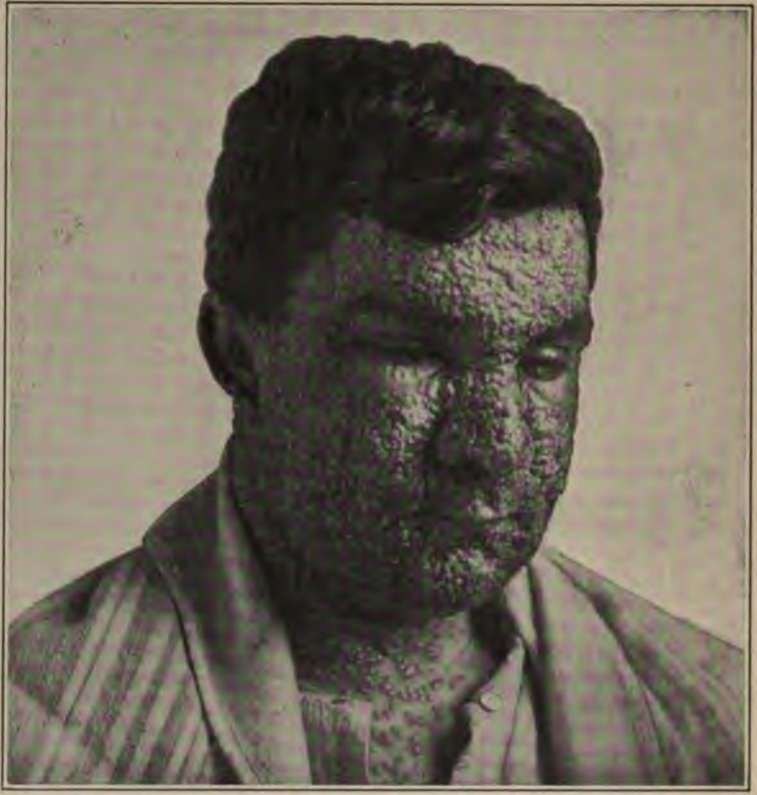
Symptoms.—After an incubation period of about twelve days the symptoms develop. As in many acute infections, *headache* and *backache* are the predominant initial symptoms of smallpox, but they are peculiar in their severity in this disease, so that their very intensity possesses diagnostic significance. Sometimes the pain in the back extends down the posterior portions of the legs. *Rigors* also occur and *pain in the epigastrium* and *vomiting* may come on. Sometimes drowsiness and sleep with muscular twitching develops as a prominent initial sign in children. The *urine* is *often scanty,* loaded with urates, and usually contains some albumin. The *temperature* in smallpox is usually high from the onset, so that it may reach 104° as early as the latter part of the first day, and 105° or 106° by the end of the first forty-eight hours. It maintains this high degree with very slight remission until the eruption is developed. The *pulse is rapid* often as high as 120 per minute, in adults, and unless profound depression is very early manifested it is fairly strong. The abdominal organs present no signs of any importance, but constipation is more frequently present than is diarrhœa.

The true *variolous eruption* makes its appearance, in the majority of cases, on the third day, although many writers state that it appears most commonly on the fourth day, while others insist that it appears on the second. The facts are that the time of the appearance of the rash varies materially in different cases, for it is delayed in mild attacks and develops early in severe ones. Sydenham said of the confluent form of this disease: "This kind usually comes out on the third day, sometimes earlier, but scarcely ever later; whereas the distinct (discrete) form appears on the fourth day or later, but rarely before." Boerhaave said: "The slower the small pocks come out, the milder they prove and the better they ripen. Those appearing on the first day of the illness are esteemed the worst kind; those on the second, milder; those on the third, still more gentle, and on the fourth the most favorable." Very rarely indeed the rash may be delayed till the fifth day, but this is an unfavorable sign.

It must be borne in mind that the first signs of the eruption may be very scanty. But one or two papules may be present on the face or hand or forearm. In other instances the papules are very numerous on the face, the extensor surfaces of the forearms, and then on the trunk, these being the parts which are particularly prone to present the first sign of the eruption. In still other cases the entire surface of the body is speedily covered and the mucous membrane of the mouth, pharynx, and vulva also are involved. The portion of the skin least affected in most cases is that of the anterior part of the thorax, the abdomen, and the flexor surfaces of the extremities.

The eruption of smallpox proceeds through the following five stages of development with considerable rapidity: For the first few hours minute bright-red macules are present, which disappear on pressure. They soon become hard and elevated—that is, the macules become papules. By the end of the first twenty-four hours of the eruption the papule begins to show at its apex a tiny vesicle, which rapidly develops so that by the fourth or fifth day of the rash the vesicular stage has reached its full development. This vesicle is, as a rule, less than a sixth of an inch in diameter, contains fairly pearly looking fluid (lactescent), and is surrounded by a narrow areola of red. A peculiarity of the vesicle of smallpox is that though some serum may escape when it is pinched, it never empties itself or collapses because of the fibrilla which are present in the cavity of the vesicle, as already

FIG. 18



Well-developed variola.

described. With the advent of the fifth or sixth day the centre of the vesicle is seen to be slightly depressed, showing the beginning of the *stage of umbilication.*

The fluid in the vesicle now rapidly becomes cloudy and purulent, the surface of the pock gradually loses its umbilication, and by the seventh or eighth day of the eruption the eruption exists as a pustule, which by the tenth day is dome-like and surrounded by an areola. This pustule, when it is punctured and pressed upon, discharges pus and cloudy serum. If the pustule is not meddled with it ruptures in about twenty-four to forty-eight hours and the pus escapes, dries, and forms a dirty-looking scab, so that by the eleventh or twelfth day of the eruption the primary macule has advanced through its stages of maturation to the ruptured pustule. These scabs produce a disgusting odor. Sometimes the pustule does not rupture, but simply dries up; when the scab falls off it leaves under its former site a red or pink depression in the skin, the future pockmark. This stage of desiccation or drying, followed by exfoliation, may last in severe cases for several weeks, and it is followed by a period of desquamation of fine scales of epidermis, during which time the reddened pockmark gradually heals and cicatrizes. This desquamation rarely takes place earlier than the sixteenth and often about the eighteenth day.

The eruption on the mucous membranes runs a much more rapid course than that on the skin, so that as early as the fifth day the pustule ruptures, leaving an ulcerated surface, which, if the eruption on the mucous membrane of the mouth has been confluent, may resemble the ragged, dirty-looking exudate of diphtheria.

There are two additional facts of importance in connection with the eruption not yet named—viz., a peculiarity of the papule of smallpox is that when the finger is drawn over it it feels indurated as if a shot were under or in the skin. The second point is, that the rash does not all appear at once, but different parts of the body are affected, one after the other, so that one part may present vesicles while another is beginning to show pustules.

Another point of interest from a diagnostic standpoint is the characteristic course of the fever. Primarily high till the eruption begins, it speedily falls to 99° in moderate cases, or to 100° in confluent ones, and remains low until pustulation begins, when the so-called *secondary fever* develops, which rises to 102° or even 104°. This fever, unlike the primary fever, has morning remissions of 1° to 2°, and gradually ends by lysis, so that at about the twelfth day, which is the period at which the pustules rupture or become dry, the temperature reaches normal.

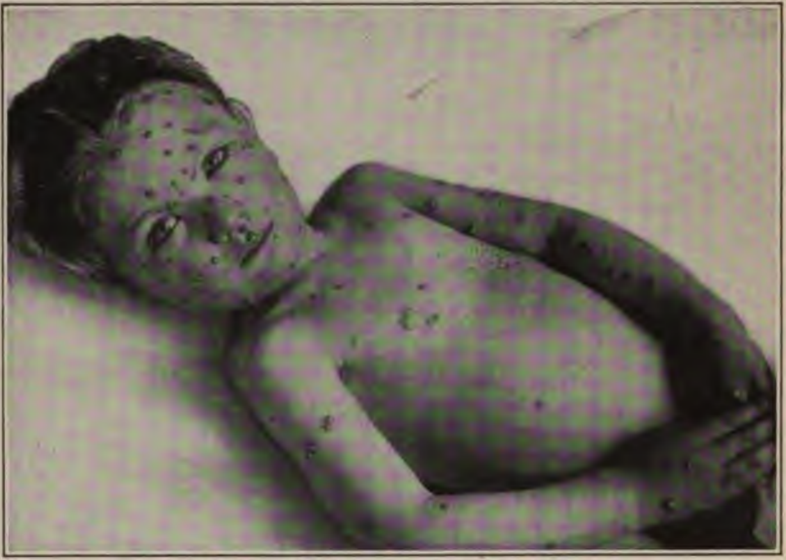
As would be expected from the severity of the eruption the skin during the active stage of the disease is deeply inflamed and so greatly swollen that the features of the patient may be unrecognizable. (See Fig. 17.)

In many cases the mind is clear throughout the illness, but in others it is clouded, and *active delirium,* which may be violent, is met with in severe cases.

In the earliest stages of variola *initial rashes* may precede the true eruption and mislead the physician if he be not on his guard. In some instances an erythema, like that of early scarlet fever, is present, and in still others a rash appears which strongly resembles the early stages of the eruption of measles. These rashes may last from a few hours to a few days, and usually appear on the trunk and limbs and but slightly on the face. The scarlatiniform rash is to be separated from that of scarlet fever by the fact that it is not so punctate, nor so bright in hue, and is not associated with the presence of the sore throat of that disease. The rash which resembles measles is scarcely raised at all, as is the real rash of that disease; it develops much more rapidly, covering the entire body in a few hours, and disappears with a speed equal to that of its onset, rarely lasting over thirty-six hours.

In some cases both the scarlatiniform and morbilliform rashes appear in very small patches on the wrists or about other joints. These initial rashes possess a considerable degree of prognostic importance, since they usually appear in mild cases.

FIG. 19



Variola in a child with scant eruption. (Schamberg.)

Still another initial skin lesion, of some importance because of its prognostic features, is an intensely red rash, which appears on the second day of the illness and spreads over the entire body so that the surface may after a few hours look as if it were affected by a generalized erysipelas in its early stages. Such a rash is said to indicate the future development of the hemorrhagic or malignant type of the disease.

Petechial rashes also occur as initial or preliminary lesions. They usually involve the suprapubic or inguinal regions, but sometimes they appear in the infraclavicular areas. The individual petechiæ may be bright red, or dull red, or purple in appearance. In still other cases an eruption which closely resembles that of true purpura develops. In very malignant cases death may occur before any typical eruption of smallpox appears.

Something more must be said in regard to the variations which occur in the eruption of smallpox. In the first place, it is possible for smallpox to occur without eruption, although, of course, such instances are exceedingly rare. In all probability, careful examination of such patients will reveal one or two papules which otherwise might be overlooked. Indeed, this type of smallpox may be considered as belonging to so-called varioloid, and to occur in those patients who have been imperfectly protected by early vaccination.

Councilman, Magrath, and Brinckerhoff describe secondary vesicles usually formed on the surface of the primary vesicle, but occasionally seen in the base.

Confluent smallpox, as its name implies, may be localized or general; that is to say, the confluence of the various pocks may occur only in certain portions of the body, while in other instances all portions of the body may be covered by a coalescence of the eruption. In these cases there is always an extensive dermatitis. There is usually great restlessness, delirium, marked circulatory disturbance, and death very frequently occurs from the ninth to the eleventh day. It is in this type of case, too, that the greatest degree of the œdema of the subcutaneous tissues appear, and the temperature usually maintains a high degree. Sometimes, however, in confluent smallpox, the vesicles do not seem to reach as great a degree of fulness as in ordinary cases, and there is not the same degree of swelling of the subcutaneous tissues, although the skin is apt to be harsh and thickened. Curiously enough, this form of confluent smallpox is considered by experts to be more frequently followed by death than that form in which the eruption seems to be more completely matured.

Very rarely in the pustular stage, the epiderm at the base of a pustule may be displaced by the formation of a bulla, or bleb, which contains a clear, straw-colored serum, and which holds in its centre the pustule.

Under the name of *hemorrhagic* or *black smallpox,* which is by no means rare, and which takes place both in sporadic and epidemic cases, a form of the disease occurs in which the initial symptoms are always very severe, and in which hemorrhages into the skin occur early. Not only do the spots become purpuric by extravasations of blood into the skin, particularly about the joints, but the hemorrhages also occur on the eyelids under the conjunctiva, and even on the tongue, the palate, the fauces, and the vagina. Bleeding also frequently takes place from the gums, and nose-bleed, bloody vomit, and bloody stools may occur. Sometimes hæmaturia also develops. In these instances the temperature usually does not rise above 100°, and the mind remains clear and unclouded, but they are distinctly typhoid in type, and death often occurs, sometimes as early as the third day, but more commonly between the third and sixth day, as the result of the profound toxæmia and associated cardiac failure.

Under the name *variola pustulosa hæmorrhagica,* a form of the disease is described in which the eruption does not become hemorrhagic until the stage of pustulation is reached. This type is not so severe as that just described.

Under the name of *variola fulminans,* an exceedingly fatal form, with a high temperature of 105°, delirium, coma, and collapse occur. In these cases death comes on within a few hours after the onset of the disease, and, while no hemorrhages are manifest in the skin, since the eruption is as yet scarcely developed, internal hemorrhages are, nevertheless, found at autopsy. It is much more apt to occur in unvaccinated than in vaccinated persons.

While the symptoms detailed up to this point may be considered as those of ordinary smallpox which runs a natural course, it is not to be forgotten that a modified form of the disease quite frequently occurs, in which by reason of vaccination many years before, or natural immunity, the manifestations of the affection are quite markedly modified. To this type of the disease the term *varioloid* is applied.

The whole of the eruption may appear within half a day after the first papule is developed. The vesicles which in an ordinary case reach their maturity by the fourth or fifth day in these cases become fully developed in seventy-two hours, and they are often very small. Instead of the fluid in the pock becoming cloudy on the fifth day this change develops as early as the third or fourth day, and many of the vesicles never become pustules but dry up. Those that do develop into pustules reach this condition by the fifth or sixth day instead of as late as the seventh or eighth in the unmodified form of the disease. It is evident, therefore, that, as most persons have been vaccinated in all civilized countries, physicians will often meet with a modified type of smallpox rather than the severe form.

The temperature in these cases runs a very mild course, often remaining at the normal point as soon as the rash develops, and never partaking of a secondary rise. Indeed, the entire symptom-complex of the illness may be of the mildest possible type as to objective symptoms, suffering, or discomfort. The appetite is good, the patient sleeps well, no complications develop, and convalescence is rapid.

The important fact to be remembered concerning these mild or modified cases is that they are quite as competent to spread the disease as are the more severe types of variola, and they require as strict quarantine as any severe cases of the disease that may occur. There is therefore every reason why a case of varioloid should be quarantined most strictly.

Even in some cases of modified smallpox, coalescence or confluence takes place with associated œdema. In these instances the confluence is not to be regarded as a very grave omen, since the pocks mature early, frequently do not rupture, and convalescence may begin as early as the eighth or ninth day of the illness.

Smallpox almost never occurs a second time in the same individual. In nearly every instance where a second attack is stated to occur, there has been an error in diagnosis, either at the time of the first or second illness.

Complications and Sequelæ.—When the severity of variola as an infectious disease is considered, it is remarkable that it has so few severe complications, and, aside from the state of the skin, so few serious sequelæ. In some instances where the infection of the skin seems to be very severe, *multiple abscesses* may develop, varying in size from a small bean to a large slough.

They usually do not appear until after the eruption has passed on to the stage of desiccation, but they may persist for a long period of time and so prolong the illness. Moore speaks of a case in which a patient who suffered from this condition could not be discharged from the hospital until after a period of nine months and nine days, because he had forty-two large abscesses following confluent smallpox. The most common seat for these abscesses is upon the extremities and about the buttocks and shoulders, and occasionally on the scalp. Much more rarely abscesses which are more deeply situated form, as, for example, ischiorectal abscess. Such abscesses may produce marked systemic symptoms, but ordinarily evidences of septicæmia are not severe.

Occasionally *erysipelas* occurs as a late complication of the disease, either upon the face and scalp or on the scrotum. Under these circumstances it is a most serious malady, and frequently destroys the patient, since he has not the vital resistance to withstand the new infection.

Bed-sores are rare if proper nursing has been carried out, but boils may occasionally occur, and are caused most frequently by the *Staphylococcus pyogenes aureus.*

Gangrene of the skin complicating smallpox is almost unknown. But when it occurs it usually affects the scrotum. The eyelids sometimes become the seat of abscesses, or more rarely slough, as the result of the swelling and œdema, but actual disease of the eyeball complicating smallpox is not common. The ears, on the other hand, are not rarely affected, and deafness occurs in a certain proportion of cases. When earache is complained of, the possibility of an extension of the suppurative process to the mastoid should be borne in mind, as this sometimes occurs with serious results.

So far as the respiratory organs are concerned, it is important to note that smallpox sometimes produces *laryngitis,* varying in severity from a catarrhal to an ulcerative type. As in typhoid fever, the development of aphonia, due to ulcerative laryngitis, is an exceedingly serious complication, since the cartilages of the larynx may become eroded. *Bronchitis* and *bronchopneumonia* may develop, and occasionally *pleurisy* results from an extension of the infection from the lung or by direct involvement of the pleura by pyogenic organisms.

The circulatory system does not suffer with anything like the degree of severity which we would expect.

Pericarditis and *endocarditis* are exceedingly rare complications.

Myocarditis, on the other hand, is more frequently met with as a result of the infection, as it is, indeed, in all of the acute infectious diseases.

The kidneys, aside from the ordinary albuminuria of all acute infectious maladies, usually escape, as does also the nervous system. That there is irritation of the kidneys is evident from the fact that Arnaud, in 1898, found albuminuria in 95 per cent. of his cases.

Septic arthritis occasionally occurs.

The occurrence of smallpox in a pregnant woman very frequently results in abortion, but if the mother goes to term, the child is to some extent protected from smallpox, although cases are on record in which children have apparently had smallpox *in utero*, and, extraordinary to relate, there are instances reported in which the child bore the eruption at birth, although the mother seemingly did not have smallpox. MacCombie even states that one case is recorded in which the mother contracted smallpox from her newborn infant.

Diagnosis.—In the later stages of well-developed smallpox there is little difficulty in making a positive diagnosis; but in the early stages, when the initial skin lesions which have been named are present, the diagnosis may be for a time impossible. Indeed, great difficulty may be experienced in expressing a positive opinion as to the presence of smallpox, even when the papular stage is in its early development. The unusually severe headache and backache, with chills, and pain in the epigastrium, are strongly in favor of smallpox, particularly if there is a history of exposure to this disease within the incubation period already named. The absence of throat symptoms, of enlargement of the cervical and submaxillary glands, and of the peculiar coating of the tongue of *scarlet fever* may enable us to determine that the initial scarlatiniform rash sometimes seen is probably to be followed by smallpox, and, furthermore, as has already been pointed out, this scarlatiniform rash lacks the punctated appearance of true scarlet fever.

On the other hand, it is to be borne in mind that in persons in whom the protective effect of an early vaccination is waning, it not rarely happens that true smallpox, or varioloid, develops in so mild a manner as to present but a few pocks and very mild systemic symptoms. A similar state may also be present in those who possess a natural immunity even if they have never been vaccinated. (See Symptoms.)

When the measles-like rash is present, the absence of the characteristic catarrhal symptoms of that disease, with its cough, running at the nose, and puffiness of the face, should cause the physician to hesitate in making a diagnosis until a sufficient time has elapsed for the eruption to be well developed. The papules which form in *measles*, while they are often confluent, do not possess the shot-like feeling so typical of the early papular stage of smallpox. Finally the measles-like rash preceding smallpox disappears in twelve to twenty-four hours, leaving no stain on the skin, while that of true measles pursues a course lasting several days. (See Measles.)

Chickenpox is one of the diseases which is most frequently confused with smallpox. In this disease, however, the initial symptoms are always mild, and the temperature does not rise as rapidly as it does in variola. Then, too, in variola, the eruption occurs on the arms and face; whereas, in chickenpox it is most abundant on the trunk, and sometimes on the scalp. It is always discrete, and it appears in successive groups. The vesicles of varicella, when punctured, collapse, since they are unilocular; while, as has already been pointed out, those of smallpox are multilocular, and so do not completely discharge their contents when punctured. The vesicles in chickenpox also reach their full development in twenty-four hours, after the appearance of the papule; whereas, in smallpox they are not completely developed for five days.

Next to varicella, *syphilis* may be considered as the disease which most frequently produces confusion in diagnosis, for variola must be separated from that form of pustular syphiloderm which is sometimes called variolaform syphilide. In most instances pustular syphiloderm is preceded by macular or papular syphilitic eruptions, but in certain instances a history of these previous eruptions may not be present. Pustular syphiloderm is more frequently met with in negroes than in the white race, and occurs, as a rule, somewhere between the sixth month and the second year of the syphilitic infection. Important points in the differentiation are that in pustular syphiloderm, the patient does not present the well-marked prodromal symptoms of smallpox, such as intense backache, although there may be a moderate fever and some pain and aching. Again, in syphiloderm there is no marked remission of the temperature such as occurs when the eruption appears in smallpox, and syphilitic patients presenting such an eruption do not, as a rule, appear very ill or have to take to their beds. Further than this, the syphilitic eruption comes out in successive crops, is often profuse upon the trunk, and the individual pustules never become so large and deep seated as do those of variola. Again, they are practically always non-confluent. Many cases of syphilitic eruption have associated with the vesicles copper-colored papules, which should render the diagnosis easy.

Drug eruptions, which are sometimes papular and pustular, are differentiated by the absence of fever and of constitutional symptoms.

Prognosis.—The prognosis of smallpox differs greatly in different epidemics and in different individuals. The greatest difference, of course, exists between those who are vaccinated and those who are not vaccinated. The mortality present in the unvaccinated may be said to amount to nearly 45 per cent., and in the vaccinated to about 8 per cent. If a patient has been vaccinated more than once, the mortality of the disease is wonderfully decreased. Thus, while among those who have been vaccinated once the mortality may be 8 per cent., those who have been vaccinated twice have a mortality of less than 4 per cent. If the mark of both vaccinations is a satisfactory one, the prognosis is exceedingly favorable, for death very rarely occurs unless the patient is already suffering from some serious disease which has undermined his constitution and therefore aids materially in causing death. In most of the instances in which smallpox has occurred after even a single vaccination, the vaccination mark has been so unsatisfactory that there has been grave doubt as to whether the patient has been protected at all.

The age of the patient influences the prognosis materially. It is much more grave in early infancy and after thirty years of age, and best at about the end of the second decade of life.

Chronic alcoholism and the presence of any antecedent disease in the heart, lungs, or kidneys makes the prognosis more grave.

Marked severity of onset is an evil prognostic sign, but a mild onset does not necessarily promise recovery, for in many instances cases which seem mild afterward become severe and fatal. Petechial rashes are always of evil import, whereas early maturation of the eruption or an aborted maturation, so that it does not go on to pustulation, is a favorable omen. Confluent smallpox, if it has not been modified by previous vaccination, is more dangerous than the discrete form, and varies in its mortality with the age of the patient. Young children almost invariably die from it. Older children and adults often recover, and it may be said that prognosis is favorable in confluent cases in direct proportion to the age of the patient until after the third decade.

Great swelling of the hands and feet, associated with salivation and swelling of the face, in confluent smallpox has long been regarded by physicians, who have had a large experience, as possessing considerable prognostic value, since if the eruption fails to appear the patient very frequently dies. The swelling is, of course, due to non-maturation of the pustules.

Hemorrhagic smallpox, if at all well developed, always ends in death.

When death takes place from smallpox, it most commonly occurs about the twelfth or sixteenth day, as the result of pneumonia, hypostatic congestion of the lungs, or from the profound exhaustion and septicæmia.

Treatment.—As in most infectious diseases, the treatment of smallpox consists chiefly in good nursing and the maintenance of vitality by the use of proper nourishment and care. The air of the room should be fresh and cool, and frequently changed. Draughts should be avoided, and food should be given frequently in small quantities. Water should be given freely for the purpose of allaying thirst and flushing the kidneys, and there is no objection to the patient receiving a small quantity of ice to relieve the dry condition of the mouth. If the urine is scanty 5 grain doses of citrate of potassium or citrate of lithium should be given every six hours. Stimulants are not needed, unless there are evidences of circulatory feebleness, when alcohol is considered by most practitioners of experience to be valuable. Good brandy and whiskey are the best forms of alcohol to employ. For the relief of intense nervous irritation, opium or morphine may be administered in small doses, particularly if the condition of the skin seems to be the chief cause of the patient's suffering. These drugs are also, perhaps, the best for the purpose of allaying excessive delirium, since they do not irritate the kidneys as do some of the newer hypnotics. Where the delirium is active and threatens to exhaust the patient, a hypodermic injection of ¼ to ½ grain of morphine will often produce several hours of restful sleep, with benefit.

For the relief of the intense irritation of the skin all over the body, a very useful dressing is ordinary carron oil—that is, lime-water and olive oil mixed in equal parts. To this may be added 1 per cent. of carbolic acid for its local antiseptic and anæsthetic properties, and where great pain is experienced, because of the occurrence of the eruption in the thick skin of the hands and feet, prolonged hand-baths and foot-baths of lukewarm water may be employed, or hot poultices used. An ointment of aristol of the strength of one drachm to the ounce may also be used.

It seems to be generally considered that local applications to the eruption are of little value in the sense of modifying its severity, although certain parts of the skin which seem to suffer from an excessive degree of irritation may be relieved by cool compresses or by the application of antiseptic poultices. MacCombie states that the best dressing for the face is a mask with holes cut for the eyes, nose, and mouth. Upon this mask is smeared on its inner surface a small linseed poultice, over which is placed some vaselin which contains iodoform. This poultice should be changed every two hours. It aids materially in separating the crusts, and so leaves the skin free for the application of the dressings, which tend to prevent ulceration and the formation of scars. The local use of antiseptic drugs to the surface of the entire body has not met with favor.

The mucous membrane of the mouth should be kept cleansed by mouthwashes of boric acid or chlorate of potassium and myrrh. When the mouth is exceedingly dry, flaxseed-tea, sweetened with a little white sugar and acidulated with lemon-juice, may be used.

The primary fever of smallpox does not last long enough to require treatment, but the secondary fever may be sufficiently high to demand relief. Cold compresses may be applied to the head, and sponging the body with cool or tepid water may be employed, but the cold-bath treatment, so successfully employed in typhoid fever, has not apparently given good results in smallpox, and it is practically never employed.

Should any irritation or inflammation of the eyes appear, they should be carefully washed every few hours with boric acid solution, and, if necessary, cold wet compresses should be applied, great care being taken that the warmth of the body does not speedily change the cool compress into a hot poultice.

During the suppurative stage, it is exceedingly important that the nutrition and vitality of the patient be preserved by the frequent administration of easily digested and predigested food.

In considering the general condition of a patient who is suffering from smallpox, it must be borne in mind that the disease is essentially one which is prone to produce profound toxæmia, since it is incredible that such widespread infection can take place all over the body without simultaneously resulting in septic absorption on the one hand, or profound exhaustion on the other. For this reason the degree of suppuration should be controlled as far as possible, measures should be introduced to aid in the escape of pus, and the treatment should be stimulating and supporting.

Finally, mention should be made of the so-called red-light treatment of smallpox, in which patients are kept in rooms to which no light is allowed to enter save through red glass, it being claimed by advocates of this method that the severity of the eruption, and so indirectly the severity of the disease, is greatly modified, and, further, that scarring of the skin is diminished. Suffice it to state, that while certain European clinicians have claimed to have obtained excellent results from this method, Welch and Schamberg in Philadelphia, and others, have found it entirely useless.

There are several points in the treatment of variola which should be carefully avoided. For the relief of the severe backache and headache, counter-irritation is sometimes employed in the early stages of the disease. Such treatment frequently results in severe ulceration or sloughing of the part to which the irritation is applied. Again, the application of powders, antiseptic or otherwise, is, as a rule, disadvantageous. The opening of individual pocks by means of a needle or the fine blade of a knife is not advisable.

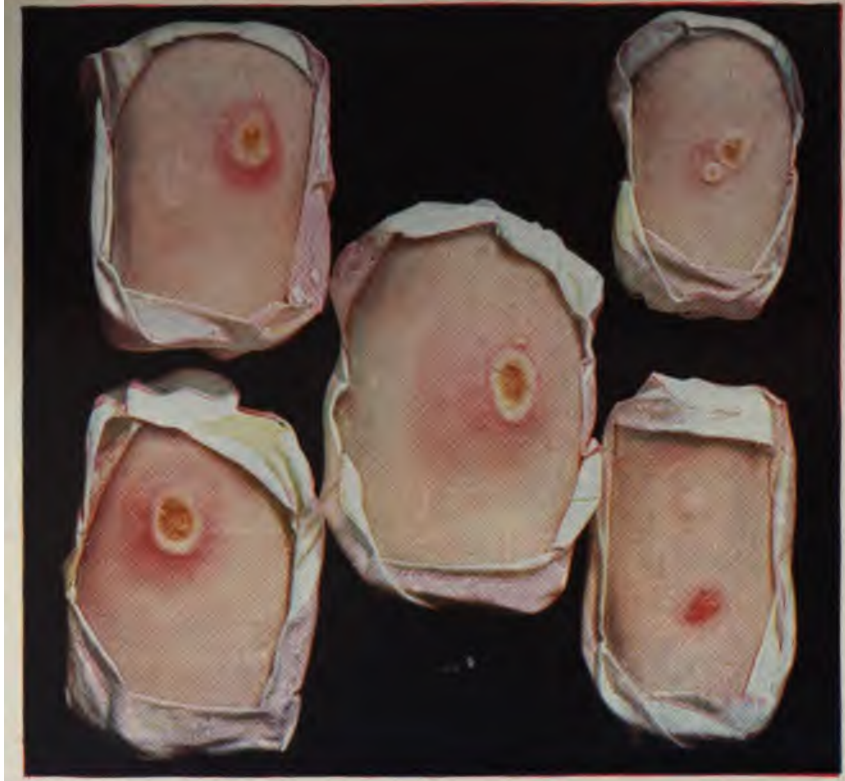
VACCINIA AND VACCINATION.

History.—Little is known of the history of vaccinia, save that it has been recognized for many years as a disease which affects heifers and cows, and that it causes an eruption to appear on the teats and udder or neighboring parts. Although it was known among those persons who milked these animals, or otherwise handled them, that the disease could be transmitted from the cow to the human being, and although many of these persons also knew that this transmission protected the human being from smallpox, it was not until Jenner, on May 14, 1796, first inoculated a patient with the contents of a cow-pock that the preventive influence of vaccination was first tried in a scientific manner. Two years before this an English farmer, by the name of Benjamin Jesty, inoculated his wife and two children in a similar manner, but at the time no report of the procedure was made. From this small beginning so-called vaccination, or the inoculation of human beings with vaccine virus, has spread all over the world, and is a well-recognized procedure, by which millions of lives have been saved. There are a few persons, not medical men as a rule, who still express doubt as to its efficacy, but they are not worthy of credence, and the statistics of every civilized land prove that vaccination is one of the greatest blessings yet discovered for mankind. It is only necessary here to state that vaccination is now obligatory in most civilized lands, and that the frequency of smallpox is in direct ratio to the laxity with which vaccination laws are enforced. Immense statistics as to its protective value are to be found in all works on public health.

Vaccination, when properly performed, and when an active vaccine is used, may be said to be a sure preventive of smallpox for a very considerable space of time, if not for the lifetime of the individual; but it is safer to be vaccinated every few years, and every year if exposed during an epidemic. Not only does vaccination protect the individual for a long period of time, but it also modifies the severity of smallpox if the patient contracts this disease before the vaccinia can completely protect him. This has been proved by practical experience so often that it is a fact beyond all doubt, and it bears this important truth with it, namely, that when a person who has not been recently vaccinated is exposed to smallpox he should be revaccinated at once, since if the vaccine fails to confer complete immunity it will modify the disease if it develops. The degree of immunity, or the degree of modification, if smallpox develops, depends upon the space of time elapsing between exposure to the smallpox and the vaccination. Further than this, if the patient contracts smallpox many years after a vaccination, the severity of the disease is usually modified. Thus in 58,278 cases of variola collected from various sources, occurring in individuals who had been vaccinated, but in whom the "takes" were not known to be good, there were 4872 deaths, a percentage of 8.35; whereas in 23,360 cases of variola, occurring in individuals who had not been vaccinated, there were 8682 deaths, a percentage of 32.88.

Method of Vaccination.—The skin on the arm or calf of the leg, having been cleansed by washing it with soap and water, is scarified or scratched by a

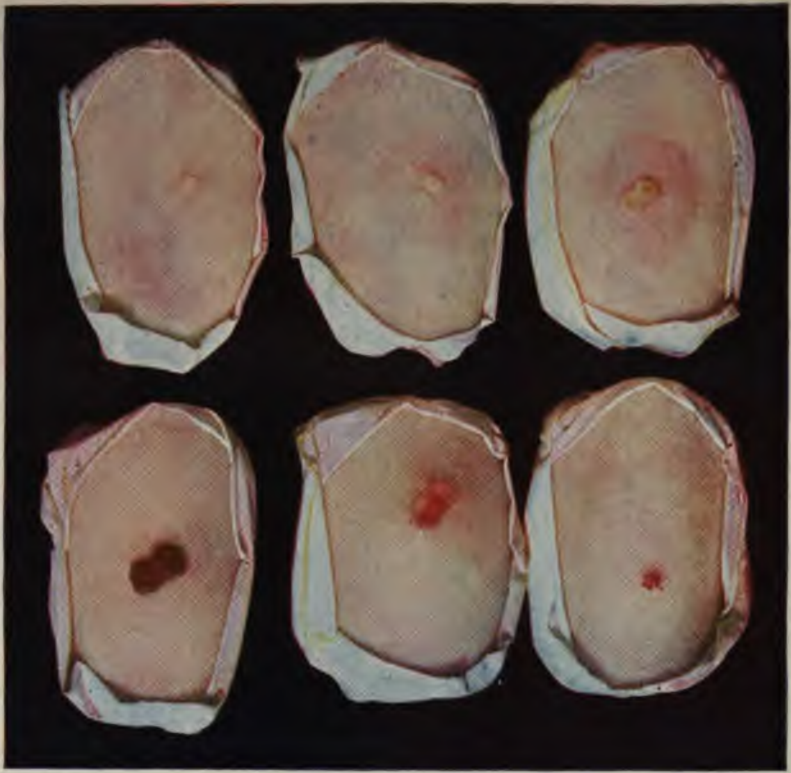
PLATE II.



Revaccination.
7th day.
Primary Vaccination.
8th day.

Revaccination.
7th day.

Revaccination.
7th day.
Revaccination.



Evolution of Primary Vaccination.

4th day.
6th day.
8th day.

Crust 20th day.
Scar 28th day.
Spurious result.

From Polyclinic Collection of Drs. Schamberg and Wallis. Wax Models made from Life by Dr. J. F. Wallis.

needle or knife-blade in such a manner as to remove the epiderm and expose the true skin over an area of about an eighth of an inch in all directions. Care should be taken that the spot is not so deeply scratched as to cause free bleeding. Upon this area is now deposited the vaccine, which is then gently rubbed into the part and allowed to dry before any clothing comes in contact with it. Several forms of vaccine are used, but that most commonly employed at present is known as "glycerinated vaccine lymph," prepared from the contents of the vaccine vesicles as they have developed on the belly of a heifer. This glycerinated lymph is put up in small glass tubes, which are hermetically sealed at the ends, so that it may not be contaminated before it is used.

Primary Vaccinia in Man.—Three or four days after vaccination has been performed the infected area begins to be slightly reddened, and this reddening increases while at the same time a reddish papule develops which by the fifth day begins to look like a vesicle, particularly if the margin of the area inoculated be examined. This vesicle increases in size, becomes filled with thin, clear lymph, and by the eighth day reaches its greatest development. At this time the contained fluid begins to be more opaque and yellow and the top of the vesicle is seen to be slightly sunken—that is, the early stage of its umbilication has been reached. The skin surrounding the vesicle is now surrounded by a zone or areola of red which by the ninth or tenth day becomes very well developed, so that it extends for a considerable distance in all directions; the spot inoculated is painful and the neighboring lymphatic glands may be swollen and tender. At this time, too—that is, about the tenth day—constitutional symptoms may come on and the patient suffer from moderate chills, a slight rise of temperature, and malaise. Sometimes roseola (*roseola vaccinosa*) may develop over the body. By the eleventh or twelfth day these symptoms are modified, the vesicle begins to desiccate, and by the end of the fifteenth day it is completely dried up, although the scab may not fall off till the twenty-first or twenty-fifth day. The crust or scab is dark red in color and thin at its centre and at its edges, but there is a thickened area, or ridge, between the centre and the periphery. After the crust falls off it leaves a pink spot which gradually fades and leaves, after some months, a foveated or pitted mark from which small scars may radiate. It is to be borne in mind that in some cases the constitutional symptoms are so mild as not to be worthy of note, while in others they may be quite severe. To be a true "take," the full development of the pock by the stages named is essential, but it is possible for the "take" not to ensue for a month after inoculation. (See Plate II.)

Secondary Vaccinia in Man.—Very few persons who have once been successfully vaccinated present the conditions just described when inoculated a second time. It is this variation from the appearance of true primary vaccination that has led to much misunderstanding in secondary cases. In other words, the secondary vaccination of a person who has lost the protective effect of the primary attempt may in its effects be very like primary vaccination, but usually it is so greatly modified as to be very different in its appearance. The difference, however, is one of degree, not one of kind, and vesiculation and umbilication should appear in all cases.

It is a point worthy of note that the so-called "raspberry excrescence" which sometimes follows vaccination on the fourth or fifth day, looking like a small nævus, is not a vaccine pock and confers no immunity to smallpox upon the patient—that is, it is not to be considered as a "take." Care must be taken, too, that the sore or mark produced by the injury of the operation be not taken for the specific lesion of vaccinia.

Children should always be vaccinated during the first year of life, or immediately after birth, if exposed to smallpox. Vaccination should be repeated through life every five years and oftener if smallpox is prevalent. If one inoculation fails it should be repeated at least three times, since sometimes primary failure is due to poor vaccine or to an error in technique. If after three attempts no "take" is produced the patient may be considered as immune, at least for a time.

VARICELLA.

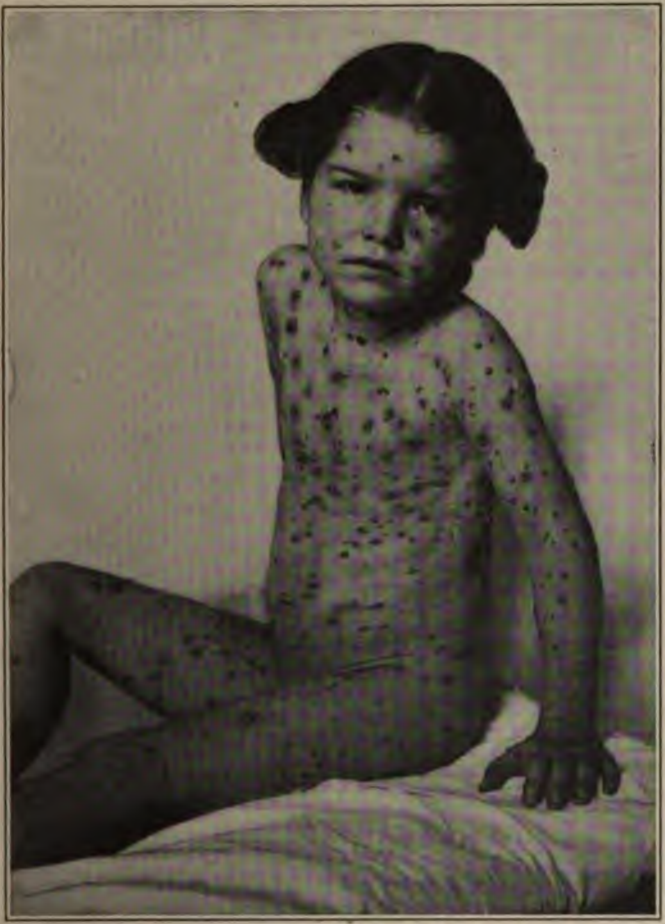
Definition.—Varicella is usually called chickenpox. It is an acute infectious disease which usually occurs in children under ten years of age, and rarely attacks individuals after puberty. In adults it is still more uncommon, although Tyzzer reports 38 cases occurring in adult male Filipinos and states that at the time of the last observation 300 cases had been recorded. The men were prisoners, and this, together with race and climate, are considered possible factors in increasing susceptibility. In all probability one of the reasons for its rarity in those of mature years is that it affects so large a proportion of all children that most adults are rendered immune by an attack in childhood. The most marked characteristic of the disease is the appearance within the first twenty-four or forty-eight hours of fever and malaise and of papules, followed by vesicles, upon the skin of the forehead and face or upon the chest and back. (See Fig. 20.)

Etiology.—Like all acute infectious diseases, chickenpox is produced by a micro-organism, but as yet it has not been isolated. Tyzzer found specific nuclear and cytoplasmic inclusions in all the lesions, but obtained no evidence favoring the hypothesis that they are parasites. It also resembles the other acute infectious eruptive diseases in that it occurs in epidemics, although at times isolated cases take place that cannot be traced to any source of contagion. While the eruption in its peculiarities resembles to some extent that caused by smallpox, chickenpox bears absolutely no relation to that malady and in no way protects a patient from developing a typical attack of variola. (See Variola.)

Symptoms.—At a time varying from ten to fifteen days after exposure to varicella the child usually manifests some evidence of a beginning illness. If very young it may be unusually *restless* and *fretful,* there may be some disorder of the digestive apparatus, and vomiting may occur. *Fever* is an early symptom and it may be moderately high—that is, about 103° or even 104°. Often, however, it fails to reach such a height. If the child is old enough to describe its sensations, some aching in the back or in the limbs may be complained of.

After about twenty-four hours the *eruption* appears in the form of red papules, which speedily become vesicles containing clear or turbid serum. The vesicle is superficial, it is not surrounded by a zone of induration, as it is in smallpox, and it does not become umbilicated, although the top of the vesicle, when it is ripe, may be flattened. By the end of thirty-six to forty-eight hours the vesicle becomes a true pock, the previously clear serum becoming opaque but not purulent unless it is denuded by scratching, and then infected. These pocks speedily shrivel and by the fourth day form crusts, which readily fall off and rarely leave a scar unless the skin be scratched by the child so that the deeper layers become infected. Many individuals bear scars of this sort upon the face, and they are particularly well marked in women with a fair skin.

FIG. 20



Chickenpox. (Schamberg.)

The eruption of chickenpox develops in a *series of crops*, or, to speak more accurately, it continues to develop in new areas as those which were affected first begin to pass into the stage of crusts. An examination of the patient on the third day may therefore reveal the eruption in all stages of development.

It is a noteworthy fact that the eruption of varicella is always discrete and never confluent. It is never profuse as in smallpox. Rarely the vesicles appear on the mucous membranes.

The severity of the fever and of the signs of general illness vary greatly in children affected by varicella. In some cases the disease runs so mild a course that the child is not kept in bed, in others it causes a considerable degree of illness; but in the majority of instances it is a very mild malady. In children who are weakened by previous disease it sometimes develops into a dangerous malady in that the associated digestive disturbance still further impairs vitality, or because the lesions of the skin become infected and sloughing or gangrene appears. Sometimes erysipelas is developed in this manner in poorly nourished children. Rarely, if the child is exposed to cold, nephritis develops. Allaire reports peripheral neuritis of the left arm following an attack of varicella in a child aged eight years, the pocks having suppurated.

Diagnosis.—The eruption of chickenpox must be separated from that of modified or mild smallpox. The most important factors in this separation are the superficial character of the pock, the lack of the sense of induration when it is taken between the thumb and finger, the early appearance of the rash on the chest rather than on the forearms, as in smallpox, and the mild character of the general symptoms, combined with the brief course of the disease and the speedy completion of the illness.

Additional diagnostic factors are the presence of a good vaccination mark which largely excludes variola. Again, the onset of varicella is usually devoid of prodromes, whereas smallpox presents for some days backache, vertigo, fever, nausea, and chills. The mere fact that the eruption is scanty does not, however, exclude smallpox. The vesicles of varicella do not become umbilicated as do those of variola, but they rapidly dry up and make a dark-colored scab. The eruption of smallpox comes out in one crop, that of varicella in several crops; that of smallpox lasts from ten to twelve days in typical cases, never less than six days, whereas chickenpox lasts but from two to four days.

Prognosis.—The prognosis is always favorable unless the unfavorable preliminary states just noted are present.

Treatment.—Medicinal treatment of varicella is usually unnecessary. Careful nursing that prevents exposure to cold and wet, regulation of the diet, and the use of a few drops of sweet spirit of nitre in a teaspoonful of *liquor potassii citratis* every four hours, to keep the kidneys active, are all that is needed in most cases. The fever runs so brief a course that antipyretic measures are not necessary.

SCARLET FEVER.

Definition.—Scarlet fever is an acute infectious disease which chiefly affects children under fifteen years of age. It is characterized by the development of an intensely scarlet, punctated rash on the *second day* of the illness, accompanied by a marked febrile movement. It is sometimes called "scarlatina," and it is to be clearly understood that this word is synonymous with scarlet fever and that it does *not* describe a modified or diminutive form of the malady, although the laity often employ the term in this manner.

History.—Hirsch states that the oldest reference to an epidemic of scarlet fever dates from Sicily in 1543, but Sydenham, of London, first differentiated it from measles. Prior to his time it had been considered a form of measles.

Distribution.—Like almost all of the acute infectious maladies, scarlet fever occurs in all parts of the world, although it seems to be much more prevalent in the temperate zone than elsewhere. In the United States it occurs less frequently in the Southern States than in the Northern States. It did not develop in the United States until 1735, nor in South America until 1830. In Australia and in Polynesia the disease first appeared in 1848, assuming a mild type but a severe epidemic occurred in Melbourne in 1876. It is said that only imported cases are met in India, and only one case has been reported in Greenland. It does not occur nearly so frequently as does measles, and very many persons reach adult life without having suffered from it. This is in part due to the fact that it is not so readily transmitted as some of the other acute infectious fevers, and also because a large number of persons seem to be resistant to the disease. Johannessen states that of 185 children exposed only 28 per cent. developed scarlet fever, and out of 314 adults exposed only 5 suffered from the malady. If the same number of cases had been exposed to the infection of measles, very few of the children would have escaped.

Scarlet fever is more apt to occur in the winter months than at any other time, but statistics differ as to the winter months' frequency. Thus, Whitelegge from his statistics based upon cases occurring in nine English towns, found in the first quarter 219 cases; second quarter, 194; third quarter, 327; fourth quarter, 460; and Reece has supported his conclusions by the accompanying chart. (See Fig. 21.)

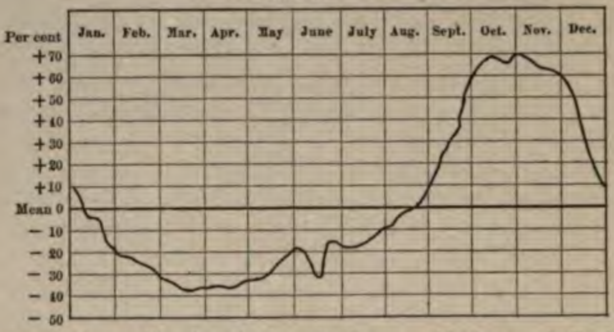
On the other hand, Seibert, of New York, gives a statistical table which shows that the last winter months are those of greatest frequency. (See Fig. 22.)

August Hirsch gives the following statistics based on an analysis of 435 epidemics occurring in all parts of Europe and North America: 178 epidemics occurred in winter; 157 in spring; 173 in summer; 213 in autumn.

The frequency and mortality of scarlet fever have greatly decreased in the last sixty years. (See Fig. 23.)

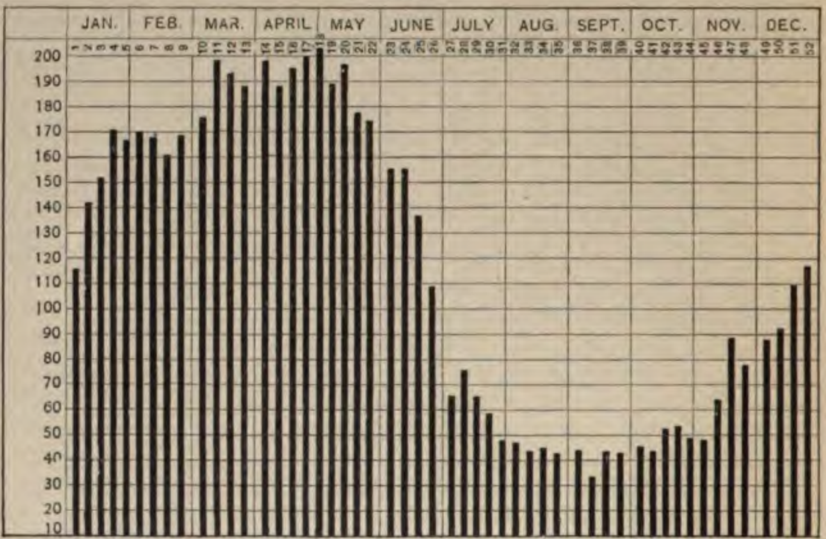
Etiology.—Scarlet fever does not disseminate itself through the air as does measles; direct contact or near association with the infected person, or with the desquamated scales from his skin, being needful for the transmission of the disease. The disease can be transmitted by the nasal mucus, clothing, and other articles which have been in contact with the patient. Thus books, cards, letters, and pets, such as dogs and cats, and other means of conveyance may assist in spreading the infection. The clothing of the nurse and physician may convey the disease, and cases are very numerous in which physicians have so communicated scarlet fever to their own children after visiting patients ill with this malady.

FIG. 21



Showing seasonal mortality of scarlet fever in all ages and both sexes in England and Wales. (Reece.)

FIG. 22



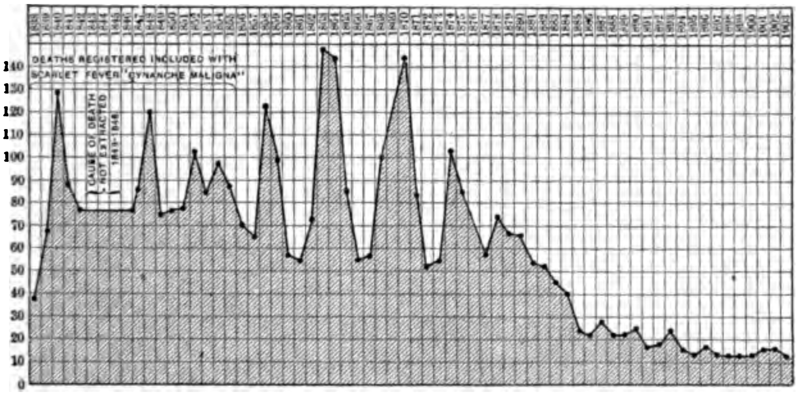
Frequency of scarlet fever, by weeks, throughout the year. (Seibert.)

The persistence of the infection in articles of clothing is very remarkable. No other acute disease renders the surroundings of the patient a source of danger for so long a period. Instances in which clothing or upholstered goods have transmitted the disease to healthy children two years after recovery of the first patient are recorded.

The breath of the patient and the air of the bed-room are probably incapable of transmitting the infection, unless the latter is laden with the dust containing the micro-organism. It is noteworthy that nurslings are not as susceptible as children of from two to five years, at which period of life the disease most often occurs. The age incidence is well shown in Fig. 24.

A patient who is a sufferer from the infection of scarlet fever is not capable of transmitting the disease until the rash develops. At the fourth or fifth day of the disease the infectiousness of the case is perhaps at its height, and the ability to transmit the malady exists as long as the skin of the patient continues to desquamate, which is often for as long a period as six weeks. It is important to remember that not only the desquamating skin, but the nasal mucus, the discharge from a purulent otitis media or from a chronic consecutive scarlatinal pharyngitis are also active sources of infection, and until all these parts are entirely healthy the danger of spreading the disease exists. Indeed, numerous instances are recorded in which children with such mild consecutive pharyngitis as to escape notice have conveyed the disease several weeks after apparent complete recovery from scarlet fever.

Fig. 23

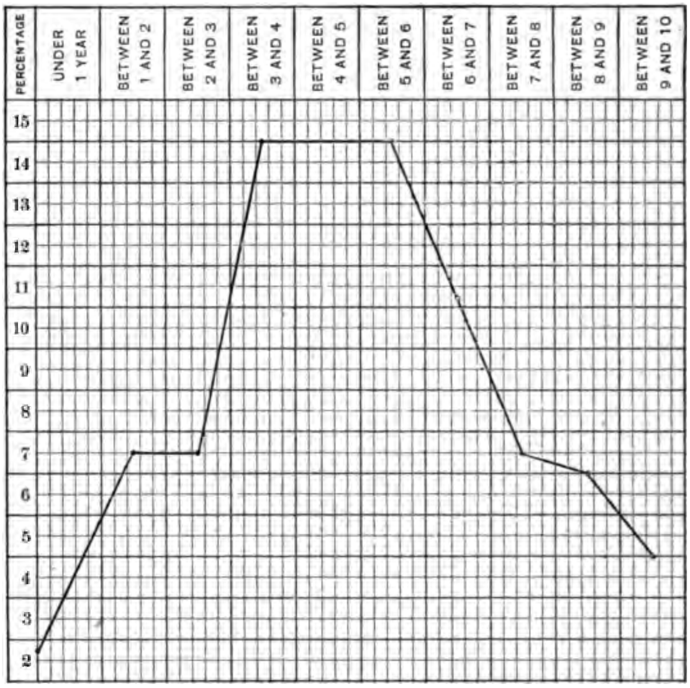


Showing the decreasing mortality of scarlet fever in England and Wales. Deaths per 100,000 population. (Modified from Wilson and Reece.)

Articles of food may also convey the infection. Thus Ekholm has recently reported an instance in which six families who partook of milk from a dairy in which there was a milkmaid who had a phlegmonous pharyngitis, suffered from scarlet fever.

Many investigators have endeavored to isolate the specific micro-organisms of scarlet fever, but without success. Loeffler, Fraenkel, and other German physicians first demonstrated the presence of streptococci in cultures prepared from secretions taken from the throats of scarlet fever patients, but their observations were limited to a small number of cases and are of interest from an historical rather than a practical standpoint. The same statement may be made concerning the researches of Klein in connection with an epidemic of scarlet fever (1885) caused by contaminated milk from a farm at Hendon, in England, for although Klein cultivated a micro-organism from lesions on the udders and teats of cows on this farm, which apparently was identical with one he found in the blood of scarlet fever patients, and although this latter organism when injected into calves produced a lesion resembling the one with which the Hendon cows were affected, the inquiry instituted by the Medical Society of Edinburgh and the investigation of Dr. Crookshank, of London, proved that the disease from which the cows suffered was a modified form of cowpox, and, moreover, that the persons who milked the cows did not contract scarlet fever. A similar history as to cows and patients has recently been recorded in Lincoln, England. In 1891 Kurth found in the throats of scarlet fever patients, in pus from the cervical abscesses and in the viscera of persons who had died from scarlet fever, a streptococcus which formed a twisted, gelatinous mass when grown in broth. This organism, called by Kurth *Streptococcus conglomeratus*, was subsequently studied by Mervyn Gordon, who found it present in the

Fig. 24



Showing age incidence of scarlet fever based on 7470 cases, and representing the combined statistics of Whitelegge, Ballard, and Keen.

throats of twenty out of twenty-seven scarlet fever patients, in the internal organs of most patients who died from the disease, and in the fluid of a scarlatinal pleural effusion. Baginsky and Sommerfield, who published the results of their investigations at about the same time as Gordon, found a streptococcus, having virulent properties and generating a toxin, in all cases of scarlatinal angina, and in cultures made from the viscera, bone-marrow, and blood of one hundred and forty-two children in whom the disease terminated fatally. This streptococcus they considered to be the specific organism of scarlet fever.

Of the work done by American bacteriologists that of Class, of Chicago, should be mentioned. In 1899 Class noticed the frequent presence of a diplococcus in cultures made from the throats of patients having different forms of angina, and upon further investigation he found that the organism invariably occurred in cases of scarlatinal angina. He then made cultures from the blood of scarlet fever patients and from desquamated epidermal scales, and found the same diplococcus. Gradwohl, of St. Louis, and Calvin Page, of Boston, have also found an organism identical with the one described by Class, but their observations were confined to a small number of cases.

From this brief *résumé* of the bacteriology of scarlet fever, it is apparent that streptococci are generally present in the throat of scarlet fever patients, and that they are often found in the blood and internal organs; but when we come to consider that streptococci have been found in healthy throats, that cases of streptococcic angina exist independent of scarlet fever, and that streptococci are found in the blood in other diseases, it is not justifiable to assume that any one of the forms thus far described is the specific organism of scarlet fever. Closely associated with the specific germ of scarlet fever, whatever it may be, we always find a variety of the streptococcus, and it has been claimed by some that this is the cause of the disease. There can be no doubt that it is responsible for a large number of the symptoms and complications of the disease.

Mallory has recently described a parasite of the animal group—a protozoon—which he suggests may be the cause of the disease. He suggests the name *Cyclasterion Scarlatinalis* for it.

Prevention or Prophylaxis.—Every case of scarlet fever should be promptly isolated and every attendant of the patient should also be prevented from mingling freely with the inmates of the house. The food should if possible be placed in an outer room and from there obtained by the nurse for the patient. If the nurse is to leave the room her clothes should be changed. Before she leaves the convalescent patient to take care of other cases she should take a hot bath and have her hair shampooed. The clothing she has worn in the sick-room should be sterilized by boiling. The physician should always change his clothes on entering and leaving the room, or at least wear over his street dress a long operating gown to protect him from the infection. If he is attending, or about to attend, a case of confinement he should refuse to take charge of a case of scarlet fever. The same rule holds true as to operative cases.

All clothing and bed-clothing should be immersed in boiling water, or in a disinfectant solution, before they are taken from the sick-room, and books and cards which have been in the patient's room should be burned. If possible it is better to burn the pillows and mattress than to attempt to disinfect them. If they are disinfected, steam should be used for this purpose. The hanging of sheets saturated with disinfectant fluids over doorways and the placing of pans of disinfectants about the house are utterly useless except that their presence constantly reminds the inmates or visitors that an infectious disease is present and so aids in the maintenance of caution. An amount of disinfectant in the air sufficient to destroy the contagium will destroy the patient and nurse. After the illness is over and the patient has left the room, it should be carefully disinfected by an adequate formaldehyde generator, the floors and walls being first moistened with water to aid in the efficiency of this gas. Afterward the floors and walls should be scrubbed with 1:2000 bichloride solution or one of chlorinated lime.

No case should be isolated less than four weeks, and no case should be allowed to mingle with other persons as long as desquamation or nasal, aural, or pharyngeal discharges exist. Before the patient is discharged he should receive at least three hot baths, after each of which he should be scrubbed with carbolic acid and water (1:100) or with bichloride solution (1:4000). Particular attention should be paid to the scalp and hair. Sleeping with other children is to be prohibited for several months.

After exposure a child should be placed in quarantine for at least a week to discover if the disease is to develop. When an epidemic is present all schools should be closed.

There can be no doubt that the use of inunctions of carbolized oil or vaselin is an active agent in diminishing the spread of the disease, particularly during desquamation, but great care must be taken that the acid is not too freely used, lest it be absorbed and increase or produce renal irritation. In many instances ordinary olive oil is equally useful.

Pathology and Morbid Anatomy.—A point of primary importance to be borne in mind in considering the pathology of scarlet fever is that the organs of the body suffer from a multiple not a single infection. Whether a special form of streptococcus is the cause of the disease, or whether an entirely distinct organism is the cause, the fact is that the disease is accompanied by streptococcus infection in all cases and not rarely by other forms of infection as well.

The organic changes produced in the body by an attack of scarlet fever are marked, but none of them can be said to be characteristic of the disease. Alterations in the skin and inflammation of the mucous membrane of the mouth and pharynx are the most constant changes, but even these may escape notice. The skin is the seat of a very acute inflammatory process involving to a varying degree all its layers and terminating, even in mild cases, in exfoliation of the superficial cells, often in large flakes. The pharyngeal mucosa is inflamed, the inflammation varying in degree from a mild acute attack to extensive necrosis involving the deeper strata of the uvula and tonsils. This inflammation in a modified form extends at times all the way down the œsophagus and by way of the Eustachian tube into the middle ear, where it not infrequently causes so destructive a change as to produce permanent deafness; or if the infection be severe and no vent for the pus is afforded the mastoid cells become involved and, finally, a secondary meningitis, or abscess of the brain, is produced. This is a rare sequel. In still other instances the inflammatory process extends into the nasal cavities and from them proceeds to an infection of the antrum of Highmore or even the frontal sinus. Extension of the pharyngeal lesions to the lymphatics of the submucosa may cause infection of the cervical and submaxillary lymph nodes, so that there is developed great swelling under the jaw, and in some instances suppuration, the so-called "collar of brawn."

Equal in frequency with these changes, and of more importance, are those which take place in the kidneys. These changes not only endanger the life of the patient during the illness, but occasionally leave him with kidneys structurally so impaired that complete restoration to health may never take place. The renal changes are primarily those of an acute diffuse nephritis involving the whole texture of the kidney, particularly the cortex, and accompanied by marked albuminuria, intertubular cellular infiltration and necrosis, and desquamation of the epithelium lining the tubes. Areas of necrosis and infarction and even acute suppurative nephritis occur, although infrequently.

When the infection with the streptococcus is particularly severe and the evidences of toxæmia are profound the autopsy reveals degenerative changes in the heart muscle, areas of necrosis in the liver, and bronchopneumonia with swelling and softening of the bronchial glands. Degenerative or necrotic changes in the myocardium and endocarditis, vegetative or ulcerative, may be present. Pericarditis may be marked. As in all septic infections arthritis may be found in numerous joints. Pleurisy, if present, often results in empyema.

With the onset of scarlet fever there develops a hyperleukocytosis amounting according to Tileston and Locke to from 18,000 to 40,000. After the eighth day, if there are no complications of an inflammatory nature, there is a gradual decline to the normal, somewhere about 6000 to 8000. The increase is chiefly in the polymorphonuclear cells.

Incubation.—The period of incubation of scarlet fever is about two to six days, but cases are recorded in which it has been as brief as twenty-four hours and as long as twenty-one days. Reimer gives the following figures: 1 day, 379 cases; 2 days, 928 cases; 3 days, 751 cases. The period of incubation is, therefore, the shortest of all the acute exanthematous fevers.

Symptoms.—The symptoms of an ordinary case of scarlet fever chiefly consist in *sore throat*, a *moderately high fever*, a *scarlet rash* first appearing on the chest, *albuminuria* of moderate degree, and a tendency to *middle-ear inflammation.*

The onset of the symptoms in scarlet fever is usually abrupt and the severity and abruptness of these symptoms is often indicative of the severity of the attack which is to follow. A child apparently in good health in the evening passes a restless night, and in the morning suddenly, without apparent cause and perhaps without preliminary nausea, *vomits* actively as soon as its breakfast is swallowed. If the *temperature* is taken, it will usually be found to be 101° or 103°, the skin feels hot and dry, the pulse is quick, the eyes bright, the expression listless, and the tongue and mucous membrane of the mouth distinctly reddened. Sometimes the first complaint on the part of the patient is one of *sore throat*, in other cases no such discomfort is mentioned; but if the mouth be opened the pharyngeal mucous membrane is seen to be angry and inflamed, and perhaps unduly dry. The child is manifestly ailing, is peevish, and is anxious to lie down. In from twelve to twenty-four hours from the manifestation of the preliminary symptoms just detailed, and in some cases in even less time than this, the *eruption*, or rash, develops, beginning on the neck and upper part of the chest, as a rule.

No one of the eruptive diseases is so characteristic in its appearance as is scarlet fever, the skin of the patient being, as the name of the disease indicates, actually scarlet or as bright a red as is the shell of a boiled lobster. Again, in no other one of the eruptive diseases does the rash appear over so wide a surface in the first hours of its appearance as in scarlet fever. Not rarely the entire body and extremities are involved in four or five hours.

There are four peculiarities about this rash which are worthy of note: first, it is punctate—that is, about each hair follicle in the skin the color is slightly deeper than elsewhere; second, the rash is often most marked in the folds of the joints, as about the groins; third, the skin of the face about the mouth or in the nasolabial line is pallid, forming a marked contrast to the scarlet hue elsewhere; and fourth, the rash on the upper part of the thorax is often very profuse.

When the rash is developed, the sense of heat conveyed to the hand and complained of by the child is notable. The eruption persists from three to seven days in the majority of cases, and ends in *desquamation* of the epiderm, which comes away in large flakes, rather than in fine bran-like scales. The skin may literally peel off the hands and feet. In rare instances it may be shed from the hand in the shape of an old glove. This desquamation lasts from a week to three weeks, beginning about the neck and continuing longest on the palmar and plantar surfaces, where the skin is thick. Indeed, I have seen it continue between the toes for six or eight weeks. The period of desquamation is, however, greatly shortened, as a rule, if during the illness the child has been anointed by some oily substance to allay dermal irritation, or if during convalescence it is frequently bathed. As long as desquamation lasts there is danger of the spread of the disease from the patient.

The stage of invasion, already described, varies in certain cases to a considerable degree. It may be so mild as to lead to a belief that the rash is due to indigestion, and it may be so severe that the patient is first convulsed, and then speedily overwhelmed by toxæmia. The eruption may not be widely diffused, but appear for a short time on the chest and abdomen, in the groin, or about the buttocks before it spreads elsewhere. It may not spread farther than these areas, and may last only one day. Such cases are often given the unfortunate name of "scarlet rash." They are just as capable of giving scarlet fever to another child as a more severe attack. In other cases, of a malignant type, the rash seems to be suppressed, the skin is mottled, but the true rash fails to appear, or it may appear in blotches, which may seem to be macular, as in measles. When doubt exists in such cases, the patient will be benefited and the diagnosis cleared by a hot bath or hot pack to stimulate the peripheral circulation and bring out the rash.

The *temperature* in scarlet fever runs its course side by side with the severity of the disease. It reaches its acme within a few hours from the onset, and is often as high as 105° within twelve hours. As a rule, this height is not maintained, but after twenty-four hours to three days it falls gradually to about 103°, and then gradually decreases daily by lysis, reaching normal, as desquamation begins, about the eighth or ninth day (Fig. 25). If it remains high or if a recrudescence occurs, some secondary trouble, such as middle-ear disease or bronchopneumonia, is to be sought for.

FIG. 25

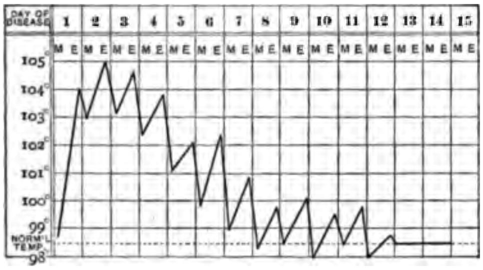


Chart of scarlet fever.

In the stage in which the disease is fully developed the clinical picture presents very great variations in different cases. In some children with a well-developed rash, the systemic symptoms are so mild that it is difficult to keep the patient in bed, and all the manifestations seem of little moment. In others the general symptoms are sufficient to show that the child is seriously ill, and in still others of a severe type the systemic state may be one of deep toxæmia, so that the child seems overwhelmed by the infection. The cases in which toxæmia is marked are not necessarily those in which great glandular involvement is present, although both sets of symptoms may occur simultaneously.

Sometimes the throat symptoms by their severity mask all others. Not only may the pharyngeal and tonsillar surfaces be ulcerated, but they may be covered by a false membrane, which, in some cases, is due to a concurrent diphtheria, but which may also be due to the streptococcus, and is always polymicrobic in nature. Such cases often present a horrid type of the disease, for the lips and teeth are covered with sordes, the tissues of the neck are infiltrated and swollen, and the head thrown far back to diminish pressure on the air-passages produced by the swelling. In such cases the general infection extends rapidly into the chest, and bronchial or pulmonary symptoms develop with great rapidity, thereby causing a fatal issue, although even with these grave complications recovery sometimes takes place.

If to these malignant manifestations are added a tendency to suppression of urine, because of the intense nephritis which has been produced, the signs of toxæmia deepen into stupor and death ensues. Cases of this type rarely die before the sixth or twelfth day, since this time is required to develop the condition described. There is, however, a fulminant form of the disease in which the malady, after being ushered in by severe convulsions, speedily develops into deep stupor, with hyperpyrexia and death. In some of these cases, however, the infection is so profound that a high temperature does not occur, the temperature never rising above 101°. These cases are very rare and are described more frequently by French clinicians than they are seen by Anglo-Saxon practitioners.

A few cases are on record in which no fever has developed, and others in which no rash has been seen.

Under the name "*surgical scarlet fever*" is described a febrile affection which attacks persons, usually children, after surgical operations or injuries. The term is an unfortunate one, for no such malady exists as a distinct disease. The condition is an erythema due to sepsis or else it is an attack of scarlet fever coming on during convalescence from the operation.

Complications and Sequelæ.—Scarlet fever depends very largely for its gravity upon its complications and sequelæ, which are not rarely met with. The most constant of these is a certain degree of *renal irritation or inflammation.* The condition of the kidneys from a time very early in the attack is such that slight albuminuria may be considered a fairly constant symptom. In some instances this febrile albuminuria is the only evidence that the kidneys are affected, but in others the character of the urine and the general systemic condition render it very plain that a true nephritis is present. Not only does the urine of such patients show considerable quantities of albumin and casts, but there is distinct puffiness of the eyelids and œdema of the ankles, or even a generalized anasarca. In such patients, if this state persists, transudation may take place into the serous cavities of the body,and the patient suffers from the pressure produced by the fluid upon the heart and lungs. He may develop uræmic symptoms, and these in turn may cause death. In many of these cases, however, the acute nephritis, responsible for these manifestations, speedily diminishes with the subsidence of the disease itself, and recovery follows with a rapidity which is extraordinary. I have seen recovery take place, even after the anasarca was so marked as to almost close the eyes and after repeated severe uræmic convulsions.

Suppression of urine may be the first symptom.

There is still another type of renal disorder met with in a few cases of scarlet fever in which the infection seems so intense that the kidneys are completely suppressed in their functional power very early in the attack, and in which we find great diminution of urinary flow, hæmaturia, and copious amounts of albumin and casts. In these cases the toxæmia of the disease and that resulting from the renal lesions produces death in a very short time.

The renal changes of scarlet fever are, therefore, to be carefully watched, and the greatest care must be taken that the kidneys are not permitted to be additionally congested by the patient being chilled. Exposure during and soon after scarlet fever may change a mild renal state into a most desperate condition.

As a sequel, rather than a complication of scarlet fever, inflammations of the joints sometimes occur. This is not acute rheumatism, but of the nature of a *septic arthritis*. Rarely the joint suppurates. The swelling does not persist, as a rule, if the effusion be simply serous. Another very rare sequel of scarlet fever is *dislocation of the hip-joint*. In 1804 J. Franck reported a case of dislocation of the hip occurring in an attack of scarlet fever. In 1894 Champenois published an account of three other cases, which were all he could collect from the literature. Since 1894 H. Stanfield Collier has reported two cases. Robert Jones, of Liverpool, states that one such case has come under his observation.

Much more common than arthritic changes during or after scarlet fever are those which are met with in the ears, due to an extension of the septic inflammation from the throat through the Eustachian tube to the *middle ear*. These have already been referred to when considering the pathology of the disease. The physician should always be on his guard for aural inflammation in the course of this malady and after it has run its course. Permanent *deafness* not rarely results from the otitis media due to this cause.

Parotitis sometimes occurs as a complication.

Next to acute articular rheumatism, scarlet fever stands as the most common of all the acute infections in producing *valvular disease of the heart*. These changes are in the endocardium and myocardium, and may be acute and transient or become permanent. Very rarely does the endocarditis become severe enough to be called ulcerative. Great responsibility rests upon the physician in regard to the cardiac changes in this disease, because, while it is true that he cannot prevent them, he can, by insisting on rest during the attack and during convalescence, to a large extent, limit their severity, both as to their temporary and permanent character. This is the more important, since, as in all acute infections, the heart is often the seat of a myocardial change.

Bronchopneumonia develops in a small proportion of cases. *Empyema* may be a sequel of scarlet fever, and is usually insidious in onset.

The *induration of the cervical glands*, which may suppurate, has already been referred to.

Nervous complications of scarlet fever, aside from delirium and convulsions due to the toxæmia, are rare. As a sequel, chorea may develop, or hemiplegia arise, caused by an embolus lodging in a cerebral vessel. Very rarely an acute ascending paralysis, which is the result of neuritis, may develop in the lower limbs.

An exceedingly rare complication of scarlet fever is *peritonitis*, due in all probability to a streptococcus infection of the peritoneum. McCollom and Blake, of Boston, have reported two such cases in the *Boston City Hospital Reports*.

Diagnosis.—While scarlet fever in its typical development is not difficult of diagnosis, it not infrequently happens that mild attacks render a decision as to the exact nature of the illness most difficult to determine. The chief reason for this is that children very commonly, and adults more rarely, develop a roseola or rose rash as a result of many different causes, and if the manifestation of scarlet fever be mild, or the rose rash be severe, the skin lesions may not only not aid in diagnosis, but greatly impede the physician in reaching a decision. The most common of these rose rashes is that produced by certain types of indigestion, and particularly that which follows eating fish, shell-fish seeming especially prone to cause it. As active vomiting and diarrhœa and even fever may be present in such cases, the patient at first sight quite markedly resembles one suffering from scarlet fever; but the absence of sore throat, of enlarged tonsils, of enlarged cervical glands, and of a history of no exposure to the specific fever, all aid in excluding scarlatina, particularly if it can be discovered that indigestible food has been ingested. Then, too, the rose rash of indigestion does not, as a rule, appear first on the chest. In some persons, with a very sensitive skin, contact with nettles or other irritants may cause a roseola. In all such cases the physician should not be hasty in making a diagnosis, but insist that enough time be given to permit him to make a careful study of the case for several days before expressing an opinion. In such instances the patient should be isolated until the diagnosis is decided.

The rose rash sometimes met with in German measles is never as scarlet as it is in true scarlet fever and is distinctly maculated. Further, it appears on the face before it is seen on the chest, the punctation of the rash of scarlet fever is absent, the fever is slight and lasts but two or three days, and flaky desquamation does not occur.

Roseola due to vaccination and that due to the use of diphtheria antitoxin are easily diagnosticated by the history of the patient.

Should a rose rash with fever develop in an adult there is much more likelihood of its being due to early secondary syphilis than to scarlet fever. The rose rash of syphilis is not, however, so bright a red as that of scarlatina. Such a rash, when due to syphilis, disappears and reappears, becomes dusky, and, finally, it is apt to be circinate.

Sometimes in acute and chronic nephritis not due to scarlet fever a rose rash develops. The absence of throat symptoms and the signs of nephritis revealed by the urine aid in the differentiation.

A condition called "erythema scarlatiniform" has a sudden onset with fever, and is characterized by a rash which develops rapidly over the whole body, lasts for several days, and ends in desquamation. The absence of throat symptoms in these cases is once more an important differential point. Further, the other symptoms are by no means so severe as the rash would lead one to expect. Such patients, too, usually have a history of repeated attacks.

A factor of very great value in diagnosis is the peculiar appearance of the tongue in many cases of scarlet fever. At the time of onset it may have a white coating, which soon diminishes in degree and becomes dotted with red and enlarged papillæ. This has been called the "strawberry tongue" of scarlet fever.

Another point of some importance is the time at which desquamation appears, for the mere occurrence of desquamation is by no means peculiar to scarlet fever. In this disease this symptom usually develops about the fourth to the sixth day on the face and about the sixth day on the chest and neck. The hands do not begin to desquamate until as late as the twelfth day, and the feet some days later than this. Other eruptions which resemble scarlet fever and desquamate usually begin to shed the skin in these areas earlier than the days just named.

In the cases of scarlet rash due to sepsis it is noteworthy that the progress of the malady is always aberrant or irregular, for the throat symptoms are often absent, the temperature is rather that of sepsis than scarlatina, and the septic symptoms may be severe. These cases are particularly interesting and worthy of the most careful study, because antiseptics, when absorbed, sometimes produce a scarlatiniform rash, and because if the case be one of true scarlet fever it is a menace to all other children, sick or well.

As the differential diagnosis of such cases cannot be made in some instances till the disease has lasted for some days or until desquamation has begun, all patients with such symptoms should be promptly isolated. A focus of septic infection is to be carefully sought for.

Prognosis.—This varies greatly in different epidemics and depends largely upon the severity of the symptoms in a given case. The malady is always to be considered a grave one. The actual mortality is shown in the following statistics. Of 26,921 cases of scarlet fever, 3216, or 11.9 per cent., were fatal. Holt states that the average mortality is from 10 to 14 per cent., but that for children under five years of age the mortality varies from 20 to 30 per cent. (See Fig. 26.) The diminution of mortality after the first decade of life is noteworthy.

Treatment.—In the treatment of scarlet fever the fact must never be lost sight of that the disease is self-limited, that it is bound to run its course, and the most the physician can do is to guide his patient through the illness with the hope that complications may be avoided and that severe symptoms may be modified.

First and foremost in the treatment of this malady, it is essential that the patient have hygienic surroundings, with plenty of fresh air and careful avoidance of draughts and exposure to sudden changes of temperature, since such exposures by chilling the surface of the body are almost certain to exaggerate the renal congestion or inflammation which is practically always present during the acute stages of this disease. Indeed, it may be said that the prime object of the physician and nurse, from the beginning to the end of the attack, is to use every effort to avoid sources of irritation to the kidneys, for it cannot be doubted that many cases of serious renal difficulty which arise in connection with scarlet fever depend upon carelessness in this respect. It is also important to remember that these precautions in regard to exposure are not only necessary during the acute attack, but until convalescence has been thoroughly completed and until the urine no longer shows any evidence whatever of renal irritation. As these lines are written I have seen in consultation a boy, aged fourteen years, who apparently had recovered entirely from an attack of scarlet fever, except that there was still some desquamation in the palms of his hands. He was allowed to play ball out-of-doors, became overheated and then chilled, and within forty-eight hours suffered from violent uræmic convulsions, which nearly cost him his life.

lesions may not only not aid in diagnosis, but greatly impede the physi
reaching a decision. The most common of these rose rashes is th
duced by certain types of indigestion, and particularly that which
eating fish, shell-fish seeming especially prone to cause it. As active
and diarrhœa and even fever may be present in such cases, the
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of a history of no exposure to the specific fever, all aid in excludi
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other irritants may cause a roseola. In all such cases the
not be hasty in making a diagnosis, but insist that enough
permit him to make a careful study of the case for several d
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the diagnosis is decided.

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are present and the patient seems anæmic,
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promptly, and particularly in those cases in which the skin is
marbled, indicating poor capillary circulation, it is exceedingly
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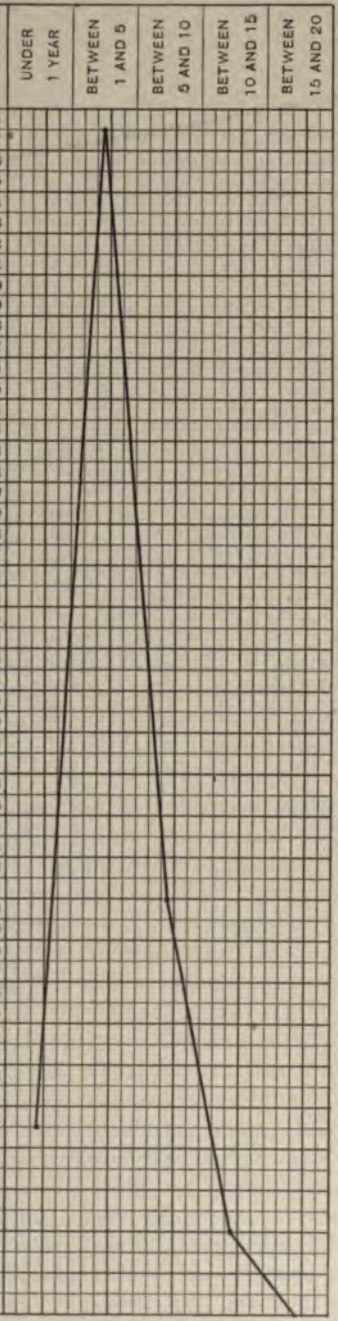
FIG. 26



Showing the mortality of scarlet fever according to age, based on Johannessen's 9855 cases.

Medicinally, it is usually well in cases of scarlet fever to prescribe from the first a mild alkaline diuretic, of which, perhaps, the best is 5 grains of citrate of potassium with 20 drops of sweet spirit of nitre in water three or four times a day to a child of eight years, giving at the same time copious quantities of such pure water as the non-sparkling water from Poland Springs, or any other spring water which contains a very small amount of organic and inorganic matter. By these means we flush the kidneys of toxic substances which in a concentrated form might produce serious renal irritation.

The second point of therapeutic importance is the condition of the throat. If the child is old enough to gargle its throat with a weak solution of chlorate of potash (3 or 5 grains to the ounce) four or five times a day, such a gargle is useful from the very beginning to the end of the attack. When the inflammatory changes in the pharynx are severe, the part may be cleansed with a spray of peroxide of hydrogen, or this drug may be applied by means of a cotton applicator, the throat being afterward cleansed by a spray of Dobell's solution. For the pseudomembranous pharyngitis which sometimes develops a similar local treatment is advisable, and, combined with this, both diphtheria antitoxin and antistreptococcic serum should be given. If the false membrane be due to the Klebs-Loeffler bacillus, diphtheria antitoxin is certainly indicated, and, as the streptococcus is always present in scarlet fever, and is probably responsible for the formation of false membranes in some cases, the use of serums designed to antagonize both of these poisons is manifestly rational.

For the relief of the intense burning and itching of the skin which is present in some cases, the child may be anointed with olive oil containing 0.5 to 1 per cent. of carbolic acid, or weak carbolized vaselin may be used. Sometimes a very distinct fall in temperature can be produced by allaying irritation of the skin in this manner.

Should the fever become high enough to deserve attention—that is, if it persistently remains above 103° or if it occasionally rises as high as 105°—the patient should be sponged with tepid water and alcohol, a small ice-bag being simultaneously applied to the head. Such a sponging, given early in the evening, will, by diminishing the irritation of the skin and quieting the peripheral sensory nerves, often cause the child to pass a comfortable night. The antipyretic coal-tar drugs are contraindicated in these cases, except under extraordinary circumstances.

If intense nervous irritation is present, 5 or 10 grains of the bromide of strontium or sodium may be given several times a day. Full doses of chloral have been highly recommended, but they are often contraindicated because of the irritant effects upon the kidneys and the depressant influence upon the heart. Should evidence of circulatory failure develop, small doses of an old brandy poured over shaved ice, or given in cool water, may be administered every two or three hours with advantage. Or, small doses, frequently repeated, of aromatic spirit of ammonia may be used in the same manner. If the circulatory failure is acute or sudden, either the aromatic spirit of ammonia or Hoffmann's anodyne should be used as rapidly acting diffusible stimulants.

Pain in the ear should be relieved by irrigating the external auditory canal with normal salt solution as hot as the child can bear it. In all these cases a careful examination of the ear-drum should be made twice a day to see whether there is any bulging due to accumulated secretion or suppuration in the middle ear, and if this is present paracentesis of the tympanum should be performed at once to relieve the pain and avoid danger of infection of the mastoid cells.

If evidences of septicæmia are present and the patient seems anæmic, either during the later stages of the attack or during convalescence, the tincture of the chloride of iron, in the dose of 5 drops three or four times a day, is advantageous, since it tends to combat the anæmia and the infection and also exercises a slight stimulant influence upon the kidneys. For the relief of persistent albuminuria after the attack is passed, the child should be prevented from taking excessive exercise, but, nevertheless, should live in the sunshine as much as possible, and may take either small doses of the tincture of chloride of iron or a very minute dose of the tincture of cantharides—say, ½ to 1 drop twice or thrice a day, well diluted; but the cantharides is contraindicated if the microscope shows in the urine the presence of red blood cells, indicating that the kidneys are still acutely inflamed.

In those cases of scarlet fever in which the rash fails to develop its full efflorescence promptly, and particularly in those cases in which the skin is mottled and marbled, indicating poor capillary circulation, it is exceedingly useful to immerse the child in a hot bath. In other cases the cool-warm pack may be used. This consists in stripping the child of its night-clothing and rolling it in a sheet which has been dipped in warm water, which, by the time it is wrapped around the child, has become considerably cooled by evaporation. As soon as the sheet is wrapped about the child, an ice-bag being in the mean time applied to the head, it is wrapped in a blanket, and in a few moments the heat of the child's body transforms the cool sheet into a warm pack. The primary effect of the cool sheet is to drive the stagnant blood out of the peripheral capillaries, and the effect of the warm sheet is to bring new blood into these vessels. By these means we are very frequently enabled not only to improve the circulation and develop the rash, but to diminish the toxic symptoms and relieve nervous stress. It is hardly necessary to add that exposure for any length of time to the cool sheet is to be avoided. The blanket is to be placed tightly about the child at the earliest possible moment after the cool sheet comes in contact with its body, so that the chilling of the surface will be only instantaneous. French therapeutists, and some other practitioners, have advised that in those cases in which cerebral symptoms are very marked and toxæmia is evidently profound, the child should be placed in a warm bath, and that cool water should be poured over its head, neck, and chest for a moment, in order to produce a certain amount of shock and rouse the flagging powers of the body. This method has been so highly endorsed by excellent practitioners that it cannot be condemned for theoretical reasons, but the author has never been brave enough to employ it.

Within the last few years several attempts have been made to produce an antiscarlatinal serum without very satisfactory therapeutic results. In the cases in which the author has directed its use, it has seemed to modify the throat symptoms, but otherwise it has not affected the progress of the disease.

MEASLES

Definition.—Measles is an acute infectious disease, usually epidemic, which most commonly attacks children and rarely occurs after the second decade of life. The skin during an attack is covered more or less profusely by a dusky red eruption of a maculopapular type. The eyes are congested and lachrymose, and the nasal and pharyngeal mucous membranes swollen and red. One attack usually confers immunity. Measles is sometimes called "*Morbilli.*"

Distribution.—Measles is met with in all parts of the civilized world. If by chance it is carried to a people who, by reason of isolation, have not been exposed in previous generations to its effects, it often develops in a malignant form and causes a great mortality. Perhaps the most noteworthy example of this is the case of the inhabitants of certain of the Fiji Islands, who, being exposed to the infection, fell ill and died by thousands, so that it is estimated that 20,000 deaths occurred in four months. The epidemic ceased only after every person on the islands had been infected.

The susceptibility of children in the first ten years of life to the infection is quite remarkable. If a large number who have not been rendered immune by a previous attack are exposed to the infection, nearly all fall sick. Smith and Dabney report an instance in which 110 children between eight and eighteen years of age were exposed, and only 2 were not taken ill.

Measles is much more prevalent in the spring and winter months than in the summer months, probably because the open-air life and free ventilation of the warmer season aids in preventing the exposure of susceptible persons to a concentrated form of the contagion.

Etiology.—Measles is in all probability due to a distinct micro-organism but so far it has not been isolated. Bacilli have been found in the blood and in the pharyngeal and nasal secretions of persons affected with the disease but no evidence has been adduced to show that they are its causative agents. The bacillus isolated and cultivated by Canon and Pielicke in 1892 produced no ill effects when injected into animals. Czajkowski and Borini have also obtained bacilli from the blood. A micrococcus which Lesage found in 1900, although highly virulent for rabbits, did not reproduce the disease. Protozoa have been found by Doehle and Pfeiffer (1892) in the blood of patients suffering from measles. Rosenberger and Wilson (1906) found in the blister fluid of 39 out of 41 cases a small hyaline body, containing a motile granule. Its relation to the disease is not yet clear.

The disease spreads with great readiness through the air and contact with the patient or his garments is not necessary for its transmission, although such contact, of course, provides the infection in more concentrated form. There is no doubt that the breath of a patient suffering from measles carries the infection, and so does the nasal and pharyngeal mucus, so that the expulsion of these secretions by coughing or sneezing may result in nurses or visitors becoming a means of transmitting the disease by their garments being contaminated in this manner.

Very short exposure to infected air is sufficient for infection, and even when careful precautions are taken to prevent the spread of the disease it not infrequently happens that all the other children in a house develop the malady, partly because it is infectious from the earliest period of invasion before its presence is recognized, but largely because of the ease with which it is conveyed by the air. This great diffusibility of the virus of measles is quite in contrast with the limited diffusibility of the poison of scarlet fever.

Although it is true that the diffusibility and activity of the infection of measles is exceedingly active while the disease lasts, it is also a fact that it speedily disappears after convalescence is established. Three weeks after the attack begins, the patient rarely transmits the disease, and by this time, with ordinary ventilation, the room and surroundings of the patient are usually innocuous. Any condition of ill-health which diminishes vital resistance very distinctly increases the susceptibility of an individual to infection, and in these instances the disease is prone to be severe.

The period of incubation of measles is usually from eleven to fifteen days, but cases are recorded in which the disease began one week afetr exposure.

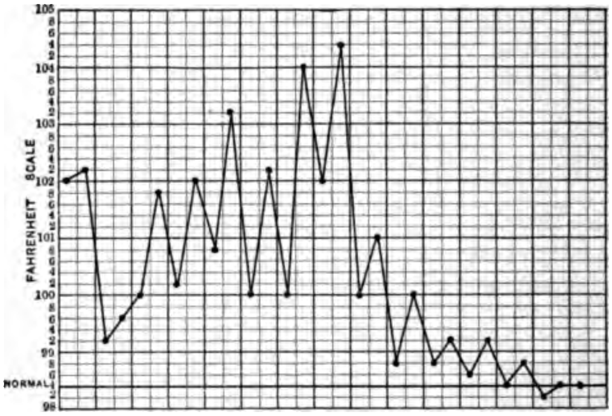
Prevention.—Measles is to be prevented by complete isolation of the patient, by the disinfection of all garments of the patient and nurse before they leave the sick-room, and by free ventilation, so arranged that the other rooms in the house are not exposed to a draught from the sick-room. After the attack has passed the patient should be given several hot baths to rid the body of all desquamating skin, and the scalp should be cleansed with special care.

Frequency.—Measles is one of the most common of the acute exanthemata and affects nearly all persons living in cities before they reach adult life. Indeed, it may be said to be the most common of all diseases in childhood.

Pathology and Morbid Anatomy.—There are no noteworthy changes produced in the various viscera by measles, if we exclude those ordinarily considered as complications and the changes in the mucous membranes of the respiratory and digestive tract, consisting of acute irritation and catarrh. With the onset of the disease these membranes become hyperæmic, and, it may be, dotted with an eruption much like that which is seen on the skin.

The pathological changes due to complications are chiefly those of bronchitis and bronchopneumonia, conditions which are exceedingly common in young children, and in patients who are poorly nursed and badly nourished, when suffering from acute infectious diseases.

FIG. 27



Showing initial fever with the subsequent fall and then a rise when the rash is well developed in a case of measles. Also shows an ending of the fever by crisis.

Symptoms.—Measles is usually ushered in by the symptoms of an ordinary cold or attack of *coryza*. There may be an initial *chill*, but this is often absent, the *fever* being the first additional symptom which becomes manifest. The patient's face looks flushed and, it may be, slightly swollen about the eyes and nose, and the conjunctivæ are injected, the general expression of the face being tearful. At this time, and later, in the disease photophobia may be marked. Sneezing may be noticeably constant, and an examination of the pharynx will reveal the fact that its mucous membrane is reddened and the hard palate dotted with a measles-like rash, which often appears here before it develops on the skin. Some cough may be present in the stage of onset as the result of the pharyngeal and laryngeal irritation, and headache may be complained of.

There is present in many cases upon the buccal mucous membrane a number of small, white-tipped, reddish spots first described by Filaton, but

PLATE III.

Fig. 1.

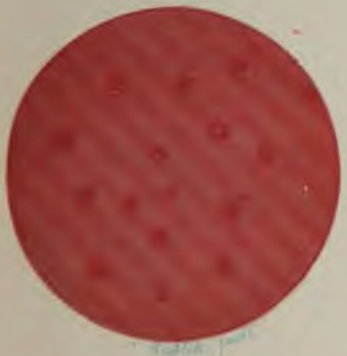


Fig. 2.

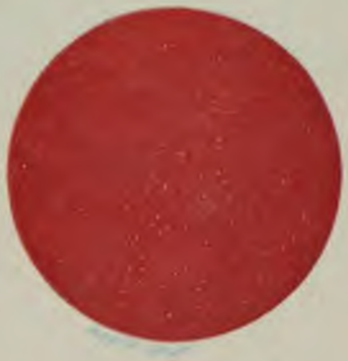


Fig. 3.

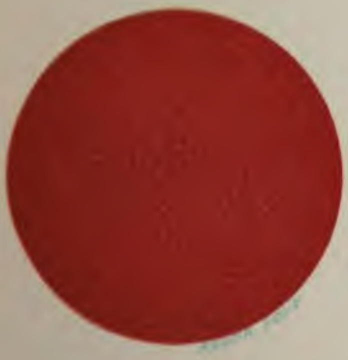
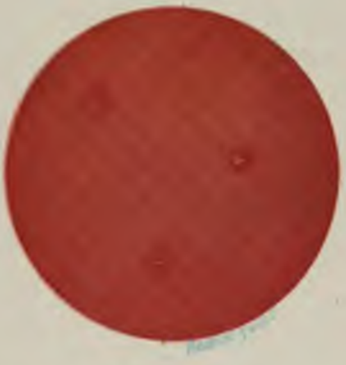


Fig. 4.



The Pathognomonic Sign of Measles (Koplik's Spots).

FIG. 1.—The discrete measles spots on the buccal or labial mucous membrane, showing the isolated rose-red spot, with the minute bluish-white centre, on the normally colored mucous membrane.

FIG. 2.—Shows the partially diffuse eruption on the mucous membrane of the cheeks and lips; patches of pale pink interspersed among rose-red patches, the latter showing numerous pale bluish-white spots.

FIG. 3.—The appearance of the buccal or labial mucous membrane when the measles spots completely coalesce and give a diffuse redness, with the myriads of bluish-white specks. The exanthema on the skin is at this time generally fully developed.

FIG. 4.—Aphthous stomatitis apt to be mistaken for measles spots. Mucous membrane normal in hue. Minute *yellow points* are surrounded by a red area. Always discrete.

more commonly called "Koplik's spots." (See Plate III.) When present they are pathognomonic of measles, but their absence does not negative the diagnosis of the presence of this disease.

The *fever* usually begins to rise with the onset of the catarrhal symptoms, increasing day by day till it reaches its acme of 103° to 105° on the fourth or fifth day from invasion, and remains fairly constant at about this level until the rash begins to fade, on the fifth to the seventh day, when the fever ceases abruptly or by lysis, reaching normal in a few hours or by the end of two or three days (Fig. 27).

The *eruption* of measles develops on the third or fourth day of the disease, and at first is most marked back of the ears and about the roots of the hair or on the forehead. The individual spots look like a flea-bite and are rather dusky red in appearance. By the end of twenty-four hours or at the expiration of the fifth day this rash is usually pretty well diffused all over the body, and the macular appearance of the eruption begins to become papular, so that it can be distinctly felt by the finger-tip of the physician. This rash varies greatly in its degree. Sometimes it is so profuse that every part of the body is covered; in other instances very considerable spaces of unaffected skin can be found between the groups of papules. It has been generally stated that the crescentic arrangement or grouping of the rash is diagnostic of measles. That this is erroneous the author is convinced, as he has frequently seen it occur in other morbilliform eruptions. When the disease is in its fully developed stage the skin of the face may be quite swollen and that of the neck and chest well covered by the eruption; but as the lower part of the trunk and the lower limbs become involved the rash on the face usually begins to diminish and slowly fades, leaving, for several days after it has entirely disappeared, a faint mottling of the skin with the desquamation of branny scales, which is scanty in some cases, but profuse in those who have had an intense eruption. The entire duration of the rash is from five days to one week, and the period of desquamation lasts for about the same length of time.

During the well-developed stage of the disease the patient nearly always presents some symptoms of *bronchitis*. This may be so mild as to be undemonstrable, or so severe as to threaten life. The thorax should be frequently examined, in order that the development of this complication may be recognized and its severe effects, as far as possible, avoided.

Variations.—It must not be thought, however, that measles always follows the course just described. All the acute infections present widely different symptoms in different epidemics and in different persons, and measles is no exception to this rule, for in some cases the systemic or constitutional disturbance is so slight as to be of no importance, whereas in others it is exceedingly severe. In strong, hearty children the course of measles is rarely grave if they are protected from cold and exposure, whereas in puny, badly nourished infants it is one of the most fatal maladies.

The following variations from the ordinary course of measles are met with:

A mild type, with a scanty rash and almost no constitutional disturbance, which runs its course without complications if ordinary care is exercised.

A severe type, in which nervous and constitutional symptoms predominate, in which the eruption may be exceedingly profuse, but is more commonly indistinct or poorly developed, perhaps because of poor circulation in the skin by reason of toxæmia.

Another severe type is known as hemorrhagic or "black" measles, because of the tendency to the occurrence of hemorrhages in the skin. Still another form is a respiratory type, in which the patient may suffer from great laryngeal and tracheal distress or from a serious bronchopneumonia. It is often said of these cases by the laity that the rash has been driven in by exposure to cold and is exerting its deleterious influence on the lungs. This is not exactly true, but it is, nevertheless, a fact that when we can, by means of a hot pack, restore the peripheral circulation and so indirectly cause the rash to be manifest, the symptoms of toxæmia and respiratory disorder often become decidedly less.

Rare cases are met with in which, after vomiting, purging, convulsions, and coma, death speedily occurs, even before the rash has had time to become well marked.

Complications and Sequelæ.—It has already been intimated that measles in itself is a disease which, in most individuals, with ordinary care, pursues a safe course and ends in recovery. While this is undoubtedly true, it is also a fact that it takes high rank among the acute infectious diseases which produce death, by reason of the complications which are prone to occur.

Of all these by far the most frequent and deadly is *bronchopneumonia,* a complication which is often severe in its course and which causes a great number of deaths when measles attacks young infants. The physical signs and symptoms are described in full in the article on that disease, but it is important to remember that in measles the disease is insidious and speedy in its onset, so that a pneumonia may be developed before the physician discovers it, unless he be on his guard and resorts to frequent examinations of the chest. Bronchopneumonia during an attack of measles in a child under one year of age is an exceedingly common and very grave complication of the disease. In children of five years or more this complication usually does not occur if the primary state of the health is fairly good and if careful nursing prevents exposure to "catching cold."

A second complication of far less importance than bronchopneumonia, both as to frequency and results, is *diarrhœa* and *vomiting* due to a catarrhal state of the bowels and stomach. It also is a complication which is due in a considerable proportion of cases to bad nursing and can generally be avoided by proper feeding and the avoidance of draughts. It not infrequently happens that these digestive disturbances are mild during the acute illness, while the patient is required to be prudent and quiet, and become pronounced when the acute illness is past and the attendants become careless as to exposure and feeding. This gastrointestinal disorder varies from a mild catarrh to a severe enterocolitis.

Another complication seen in many cases is a mild degree of *stomatitis,* which in poorly nourished children may become ulcerative. Even so severe and fatal a lesion as *noma* may develop in cases with very low vitality. Very rarely *gangrenous ulceration* of the ear, the labiæ, or the prepuce takes place.

So far as the *nervous system* is concerned, it may be said that it is rarely affected. In the stage of onset in very young children with poor resistance and an unstable nervous system there may be convulsions, but they are exceedingly rare. *Meningitis* as a sequel to measles is also very rare. Even meningitis due to middle-ear disease is rarely met with, for the otitis of measles, while not uncommon, is usually mild and rarely causes secondary lesions.

The eyes are usually inflamed and there may be a *mucopurulent conjunctivitis*, or, if the general health be poor, *keratitis* may prove troublesome.

So rarely are the heart and kidneys affected to any serious degree that these organs may be considered almost immune. A feebleness of the heart due to the infection and fever may be present for a time, and a transient albuminuria is often manifested, but both of these symptoms usually rapidly disappear if the patient is kept at rest.

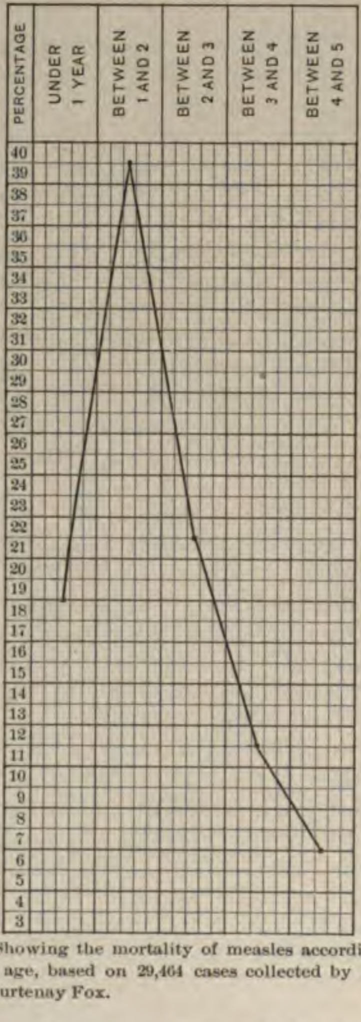
Measles is an infection which is not rarely complicated by other acute infections. *Diphtheria* may develop during its course, and *whooping-cough* is so exceedingly frequent that some relation between the two diseases has been thought to exist. When whooping-cough does occur as a complication the danger of bronchopneumonia is greatly increased. Still another sequel of measles is *tuberculosis*, probably because the catarrhal state of the mucous membranes offers a path for infection by the tubercle bacillus or because the devitalizing influence of measles permits an old focus of tuberculous infection to become active.

The persistence of a febrile movement in a case of measles after seven days should always arouse the suspicion of some inflammatory complication which should be most carefully searched for.

Diagnosis.—Measles must be carefully separated from a large number of conditions which somewhat resemble it. Many kinds of food, particularly shell-fish, produce a rash which looks remarkably like measles, but which usually lasts only a few hours. Antipyretic or other coal-tar products do likewise in some persons, and the physician should always enquire as to the use of these foods or drugs before stating that measles is present. Sometimes a morbilliform rash follows vaccination or precedes smallpox. The use of antidiphtheritic serum may also cause such an eruption. The contact of a caterpillar with the skin in some persons may cause a measles-like eruption which lasts only a few hours. None of these states, however, are accompanied by the appearance of Koplik's spots, by marked coryza, nor by the appearance of the rash on the mucous membrane of the soft palate. Fever, too, is usually absent. (For the diagnosis from Rötheln, see Rubella.)

Prognosis.—From what has already been said it is evident that the prognosis in a case of measles is dependent not on the fact that measles has developed, but rather upon the age of the patient, the vital resistance or the general condition of the system, and the surroundings as to sanitation and nursing. Given a poorly nourished infant in bad surroundings and with inefficient care, measles becomes one of the most fatal diseases to be met with, whereas in a case where these conditions are good the prognosis is fairly favorable. We find, too, that the danger of the disease decreases greatly with each year of life; so that children near puberty rarely die from this malady unless poorly nourished or badly neglected (Fig. 28). If bronchopneumonia develops, the prognosis must be guarded in direct proportion to the youth of the child. Thus, out of a series of 408 cases of measles complicated in this manner, 290, or 71 per cent., died. This, however, is an exceedingly high figure and by no means represents the death rate in a general run of cases in which all ages and conditions of patients are considered. Under these conditions the death rate for all cases is probably about 35 per cent. Thus, Holt speaks of an epidemic in the Nursery and Child's Hospital in New York in 1892, in which the mortality was 35 per cent., and in 9239 cases of measles occurring in France, principally in the hospitals of Paris, there were 3096 deaths, or a mortality of 33.5 per cent. It is, moreover, to be carefully borne in mind that hospital or asylum statistics are utterly worthless in determining the death rate for ordinary private practice, because most of these hospital cases are primarily in bad health or are brought to the hospital desperately ill from neglect. Including all cases in private practice, the mortality should not be over 5 to 10 per cent., and in many epidemics it is much lower, even in institutions and where good nursing is not to be had. Thus, in an epidemic in the Faroe Islands only 8 cases out of 1123 cases died, and at the Boston City Hospital only 5 were fatal out of 366.

Fig. 28



Showing the mortality of measles according to age, based on 29,464 cases collected by H. Courtenay Fox.

Treatment.—When measles runs a natural course, little or no medication is required; for, as it is a self-limited disease, it cannot be jugulated. The therapeutics of an attack of measles, therefore, consists in the prevention of complications and the relief of symptoms which are so prominent as to be distressing or perhaps even dangerous. In order to avoid irritation of the eyes and to lessen the suffering due to photophobia the sick-room should be kept dark. Light bed-covering should be employed, and heavy quilts which cause the child to perspire unnecessarily are to be tabooed. As a mild gastrointestinal catarrh is often present with the fever, food should be light, given at frequent intervals, and should consist chiefly in nutritious fluids, such as the various broths, milk, an egg boiled only one minute, and similar substances.

If the irritation of the conjunctivæ is marked, eye drops, composed of 4 grains of common salt and 4 grains of boric acid to an ounce of water, may be used several times a day; and if the cough is sufficiently constant to prevent sleep, it may be controlled by small doses of codeine, $\frac{1}{30}$ of a grain once, twice, or thrice in twenty-four hours, to a child of two years, or heroin may be used. Should the fever reach 105° there is usually no necessity of reducing it owing to its short duration, but the child's comfort can be much increased by sponging it with tepid water and alcohol, or even with water at 70°, using active friction at the same time. These cases do not need an immersion bath and it is not wise to give it to them. If the circulation has a tendency to fail, carbonate of ammonium in the dose of 2 grains four or five times a day may be given in syrup of acacia. For the relief of headache a small ice-bag may be applied to the head, provided that a nurse is at hand to prevent it from slipping down upon the neck, or about the ears, and also to prevent it from wetting the pillow. It should usually be wrapped in a towel to prevent the accumulation of moisture, and also to protect the head from too great cold.

In cases in which the rash is not well developed and the skin is dusky in hue, the brief use of a hot pack is very useful.

Should diphtheria arise as a complication antitoxin should be given.

After the disease has run its course, convalescence should be aided by the use of simple bitter tonics, the hypophosphites, iron, and arsenic, and, if malnutrition is present, cod-liver oil proves itself an exceedingly valuable remedy, since it improves the nutrition of the patient and exercises a most beneficial effect upon the mucous membranes. If the bronchitis is persistent and a considerable quantity of mucus is in the bronchial tubes, 3 grains of chloride of ammonium may be given in a teaspoonful of fluid extract of liquorice and a teaspoonful of water three or four times a day, and gentle counterirritation in the form of chloroform liniment or ammonia liniment may be applied to the chest. After the eruption has disappeared and desquamation has begun, the child should be bathed daily in order that its skin may be thoroughly rid of dead epithelium; and before the patient plays with other children the scalp should be shampooed several times, since not infrequently desquamation continues upon the head long after it has ceased upon the trunk.

For a long time after the rash of measles has disappeared the greatest care should be exercised that the patient is protected from exposure, as acute and chronic catarrhs of any or all the mucous membranes are very prone to develop under very slight provocation.

RUBELLA.

Definition.—Rubella is sometimes called "*rötheln*" or "German measles," "*rubeola notha*," "epidemic roseola," and "hybrid scarlet fever." It is a disease distinct from measles and scarlet fever, and is one of the mild acute infectious eruptive diseases of childhood. It rarely affects adults. Johann Seitz studied an epidemic involving 21 families and comprising 111 cases, and found that 4 per cent. of all adults were attacked. The ratio for children was much higher, being 64 per cent. Rubella occurs as a rule in epidemics, but sporadic cases are met with.

Etiology.—The micro-organism of this affection has not been isolated, but the disease is distinctly infectious and is contracted by one patient from another, not only by contact, but also by clothing and through the air.

Symptoms.—After a period of *incubation* lasting from ten to twelve days the stage of onset manifests itself by chilliness, general malaise, some running of the eyes and nose, and headache. As early as the first day of the illness the *rash* appears as a macular eruption which is red in hue, but is not scarlet. This is a so-called "rose rash." In some cases, however, this rash does not develop till the third day. The rash shows itself first on the face, then on the anterior surface of the thorax, and speedily covers the entire body. It can often be seen on the soft palate before it appears on the skin, in the form of bright rosy-red spots (Forchheimer's spots). The individual macules may remain separate or coalesce. In some instances, however, the skin has a diffuse redness like that of scarlet fever, but it is less scarlet. The macules last about three days and then fade gradually, being usually, but not always, followed by slight scaly desquamation. The skin is rarely as much stained after the rash disappears as it is after measles.

A noteworthy sign to be sought for is the *enlargement of the lymph glands* below the ears, in the lateral cervical region, and at the back of the neck. Sometimes the inguinal glands are also affected.

The *febrile movement* is usually very moderate, the temperature often not rising above 100°. The general symptoms may be so mild that the attention of the nurse is first called to the illness by the rash.

If the child is carefully nursed and clothed and properly fed, the malady pursues a rapid course to recovery. If, on the other hand, the child be feeble and exhausted, this disease may be more severe in its manifestation and be accompanied by otitis media, catarrhal pneumonia, or even albuminuria and jaundice.

Diagnosis.—Rubella is to be separated from true measles by the moderate character of the coryza, by the absence of Koplik's spots, the early swelling of the glands in the neck, and by the absence of bronchial irritation. From scarlet fever it is separated by the absence of high fever and of the well-diffused scarlet rash, which is not macular, and by the absence of the sore throat of that affection. While these differential points are of value in many cases, it is a fact that in some instances a diagnosis is most difficult until the case has been studied for some days, when the mildness of the symptoms and the brevity of the attack aid in deciding that neither measles nor scarlatina are present.

Treatment.—The treatment of rötheln consists in rest in bed and the use of spirit of nitrous ether and citrate of potash as diuretics and diaphoretics, and in attention to the bowels and kidneys. Exposure to cold should, of course, be avoided.

MUMPS.

Definition.—Mumps, or epidemic parotitis, is an acute infectious disease affecting the parotid gland and accompanied by mild systemic symptoms which may not be severe enough to demand notice. It occurs in the great majority of instances during childhood, between the fourth year and puberty, and one attack protects the patient from a second.

Etiology.—Mumps is usually conveyed by contact from one patient to another, but it may be carried by a third person or by garments to a susceptible individual. It is contagious from the beginning to the end of the attack, and it is probable that persons who have so far recovered as to have no visible swelling of the parotids can still transmit the disease. For this reason the patient should be kept separate from other children for a period of ten days after the swelling disappears. It is, however, a noteworthy fact that mumps is by no means so infectious as are the eruptive fevers, and many children escape the disease even when thoroughly exposed to it.

The period of incubation is uncertain. Sometimes it is brief, in other cases surprisingly prolonged. Holt, in 42 cases collected from literature, found it varied from three to twenty-five days. In all probability it is about fifteen days in the average case.

Pathology.—The chief change in mumps, and, indeed, the only one which is characteristic, is the swelling of one or both parotid glands. The swelling is due to a primary parenchymatous inflammation, followed by involvement of the connective tissue of the gland as well. Rarely the other salivary glands become swollen, and still more rarely the parotids suppurate. This result occurs only in children who are impoverished by other diseases, and is due to an invasion of the gland, through the duct of Steno, by pyogenic organisms.

Symptoms.—The chief symptoms of mumps, aside from the *swelling of the glands,* is *pain* in the parotid region, which is greatly increased by moving the jaw or by taking any sour material into the mouth. In susceptible persons there may be some feeling of malaise or wretchedness and the fever may reach 103° or 104° on the first day, although a temperature of 102° is more commonly met with.

The swelling of the gland is usually at its height by the third day and remains at this stage for two or three days more, when it begins to decrease and then gradually disappears. In some cases the degree of swelling is so marked that the tissues of the face and neck share in it to such an extent that the patient is unrecognizable. The swelling is bilateral in the vast majority of instances, but it often begins in a single gland.

Complications and Sequelæ.—While mumps is a very mild disease in many cases, it at times becomes severe, chiefly because of the complications which arise. These are more frequently met with in adults than in children. The most common of them is *orchitis,* which may be bilateral and severe enough to cause the patient intense suffering and force him to remain in bed.

Before the age of puberty the testicles are rarely involved, but after puberty orchitis is a frequent complication. Bich collected statistics on 862 cases of mumps occurring in young men between the ages of eighteen and twenty-five years, and found that 29 per cent. of the number were affected with orchitis. Granvier's record of cases occurring in the French army gives a percentage of 23. Usually only one testicle is involved. Thus, of 159 cases collected from various sources 152 were unilateral. The combined statistics of Grangier and Bich, based on 309 cases of orchitis, showed that atrophy of the testicle resulted in 176 cases, or 57 per cent. Active exercise seems to predispose to this complication, and it seems much more frequent in some epidemics than in others. Some years ago mumps appeared in an epidemic among the students of the Jefferson Medical College, and a very large proportion of those attacked developed metastasis to a testicle. The development of the orchitis is usually associated with a second rise of temperature and a general sense of illness which is in excess of that present at the onset of the primary illness. The swelling of the testicle lasts about a week, and after the acute inflammation has passed the gland may be enlarged for a long period of time.

Cases have been recorded in which *convulsions, meningitis,* and *arthritis* have developed as complications of mumps.

In young girls who have mumps, secondary swelling of the mammary glands, of the ovaries, or of the labiæ may develop, but secondary changes are far more rare among females than are those detailed as occurring in males.

Simonin, a French surgeon, has reported 10 cases of *pancreatitis* which occurred among 652 cases of mumps. The symptoms of pancreatitis appeared from the first to the twelfth day of the disease, but usually from the third to the sixth day, and lasted from two to seven days. The chief symptoms were epigastric pain and vomiting, but no glycosuria. Cuche has stated that he found epigastric tenderness present in 20 out of 26 cases of mumps.

Treatment.—The treatment of mumps consists in the use of mild alkaline diuretics and rest, for if the patient can be persuaded to avoid exercise and to use a light diet active medication is never needed. Sour foods and acid drinks are to be avoided, for when they are taken into the mouth they cause severe pain. If the febrile movement is marked and the pulse is quick 1 minim of tincture of aconite every two hours is useful for the first twenty-four hours of the malady. By decreasing the congestion in the gland the aconite not only moderates the inflammation, but also diminishes the pain. Local applications to the swollen parotids are usually not needful, but if any are employed they should be hot rather than cold. Should metastasis to the testicle occur, rest in bed is imperative, since taking exercise at such a time causes great increase in the swelling and pain. The scrotum should be supported by a bandage. Aconite in full doses and citrate of potassium are useful remedies when the swelling of the scrotal contents is severe.

WHOOPING-COUGH.

Definition.—Whooping-cough is sometimes called *Pertussis*, and is an infectious disease chiefly met with in childhood. It consists, as its name implies, in a respiratory disorder which is peculiar in two particulars. The patient in the well-developed stage of the disease is seized at varying intervals by a paroxysm of coughing which is so constant and violent that in a few seconds the quantity of residual air in the thorax is greatly decreased below the normal amount, producing in this way a sense of suffocation and flushing of the face or cyanosis. Immediately after the cough ceases the patient endeavors to take a deep inspiration to compensate for the excessive expiratory effort, when there is developed a narrowing of the glottic opening so that it is very difficult for the air to enter the larynx. This violent effort to draw air through a narrow opening produces a peculiar "whoop," which gives the disease its name. The name "whooping-cough" does not signify that the cough is whooping in character, but that there is a cough followed by a whooping sound.

Distribution and Frequency.—Whooping-cough is a disease which is found in all parts of the world, and is apt to occur in epidemic form, particularly during the months of March and April. It is least prevalent in September and October. As already stated, it is particularly prone to attack children; so that few persons reach adult years without suffering from an attack. If they do escape during childhood, they may suffer from it even in advanced old age. Even sucklings are attacked by it, and in this class of cases it is an exceedingly fatal malady. It is also a grave disease in old age.

Whooping-cough attacks both sexes with about equal frequency. Rosen collected 43,393 cases, of which 21,850 occurred in boys and 21,543 in girls. If the statistics of Goodhart, Comly, and Rilliet and Barthez are combined, it is found that in 4157 cases 1868 occurred in boys and 2289 in girls.

Etiology.—The cause of whooping-cough is certainly a micro-organism, and several investigators claim to have isolated it. Unfortunately no uniformity of opinion exists as to the specific organism of this disease. In 1887 Afanassjew, of St. Petersburg, isolated from the sputum of children affected with whooping-cough a short, slender, motile, anaërobic bacillus, cultures from which, when injected into dogs and rabbits, produced a disease similar to pertussis. This organism was found afterward by Szemteschenko, of St. Petersburg, and Koplik, of New York. In 1892 Ritter, of Berlin, described a diplococcus which he thought was specific, and more recently a bipolar bacillus resembling the *Bacillus influenzæ* has been considered the pathogenic agent by Czaplewski and Hensel (1897) and by Arnheim (1900). Jochmann, Krause, and Moltrecht, of Hamburg (1903), also have isolated an organism resembling the bacillus of influenza, but differing from the one described by Czaplewski and Hensel, which they have named *Bacillus pertussis Eppendorf*, and which they believe plays an important rôle in the production of whooping-cough, and also the bronchopneumonia which complicates it. Davis (1906), found this organism in the sputum of almost all of a large number of pertussis patients. Morphologically and culturally it is identical with the influenza bacillus which he believes it should be called. The evidence at hand does not prove it specific for whooping-cough. Leuriaux, of Brussels (1902), ascribes the disease to an aërobic bacillus ovoid in form and having rounded ends. This organism grows on all ordinary culture media and stains with aniline dyes and by Gram's method.

Whooping-cough so often occurs in close connection with an attack of measles that the two diseases must be regarded as nearly related.

The exact period of incubation is unknown. It probably varies greatly in different persons and in different epidemics. Sometimes it seems to be as short as two days; in others it apparently takes ten days, or even longer than this. The infection is perhaps conveyed by the air and certainly is transmitted by the sputum, either by the direct expulsion of particles of it into the face and air-passages of the child not as yet affected, or upon clothing or the food, so that it gains access to the respiratory tract. The infection is most marked during the acme of the malady, but is active at all times during the attack, and probably for a week or more after the cough has lost all characteristics of the disease. Children who have suffered from this disease should not come in contact with those who have not had it for three or four weeks after the last whoop is heard.

Pathology and Morbid Anatomy.—Primarily the only noteworthy change present in the thoracic organs during whooping-cough is a mild catarrhal state of the mucous membranes of the whole respiratory tract. Secondarily, the pathological results are far more serious in that the bronchitis and the great strain thrown upon the heart by asphyxia result in conditions which may destroy the patient, death usually ensuing in fatal cases from exhaustion due to excessive cough, lack of food, and lack of rest combined with bronchopneumonia, which in turn is also due to several causes of which lowered vital resistance and a feeble heart are important factors. Then, too, in the violent inspiratory efforts of the patient small particles of food or infected mucus may be drawn into the smaller bronchi and so produce local infection. As stated in the article on Bronchiectasis, this condition in its cylindrical form may be caused by pertussis. (For further pathological changes see Complications.)

Symptoms.—The symptoms of whooping-cough have already been described to some extent. Usually the patient develops what is apparently a slight cold in the head and thorax, followed by a *cough* which may be described as nervous or spasmodic. Perhaps the word "sudden" can best be applied to it in the sense that each coughing spell is sudden in onset. At first there may be only one or two coughs, but soon they come in series, which day by day increase in frequency and violence. Sometimes the *whoop*, which occurs at the end of the series of short, sharp coughs, does not appear for several days. It may never appear in the mild type of case, the patient suffering only from the paroxysms of cough which exhaust the chest of air to a considerable degree. When the whoop does come on it appears at the end of the repeated coughs, and is caused by the attempt to inspire air suddenly and forcibly through the narrowed glottis. The whole paroxysm, therefore, consists, first, of a series of coughs which increase in rapidity as one would count 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12 with increasing speed, and, secondly, in the long-drawn inspiratory whoop. Owing to the violence of the cough the face becomes suffused, the tears run, and the patient may even seem more or less convulsed. The frequency of the paroxysms varies very greatly in different cases and at different times in the twenty-four hours. Some patients cough but once or twice a day, while others are seized every few minutes. Usually the child is greatly frightened if the attack is severe, and often it soon learns to recognize the early signs of an approaching seizure and runs to its mother or nurse for help. The attacks are provoked by crying, laughing, eating or drinking, and by inhalation of dust-laden air. Between the paroxysms perfect quiet and respiratory comfort may be present unless complications arise. In the severe cases nose-bleed and ecchymoses of the conjunctiva may occur and blood may come from the ears and mouth. The convulsive efforts during the cough very frequently cause vomiting, and at times the urine or feces may be forcibly expelled, or they escape after an attack because of profound exhaustion and the relaxation produced by the asphyxia. A nodular infiltration, or an ulcer, at the frænum of the tongue is often produced by irritation of the projecting organ upon the lower incisor teeth. The circulation is usually not much affected save during the paroxysm, when it is labored, owing to the asphyxia. Between the paroxysms it may be rapid and feeble if the attacks are frequent and severe enough to strain and dilate the heart. Some clinicians assert that permanent cardiac feebleness and dilatation may result from this disease.

In severe cases in young children and in feeble individuals great asthenia may be produced by the violence of the spasm, the loss of sleep, and the loss of food from vomiting, which may occur at every paroxysm.

Inspection of the bared chest during the inspiratory part of each attack reveals in the stage of inspiration deep retraction of the intercostal spaces, of the episternal notch, and of the epiclavicular areas. The epigastrium is also retracted, for all the auxiliary muscles of respiration endeavor to aid in the drawing in of air. Auscultation of the chest, particularly over the posterior surface, almost always reveals bronchial rales, due to the bronchitis which is present in all cases, even if they be mild. Care should always be exercised that this bronchitis is not increased by exposure to cold and dampness, since it is exceedingly prone to develop into bronchopneumonia, particularly in young children and old persons. Indeed, it may be said that the high mortality of the disease is due almost entirely to this complication.

A number of clinicians, particularly Cima and Meunier, have shown that even in the very early stages of pertussis there is present a very extraordinary degree of leukocytosis. This leukocytosis is largely composed of lymphocytes, the polymorphonuclear cells being relatively decreased. As in most infectious diseases, a small amount of albumin is found in the urine in the majority of cases.

The duration of whooping-cough varies from six to eight weeks, more commonly the latter than the former.

Complications.—The complications of whooping-cough are chiefly connected with the respiratory tract. *Bronchopneumonia,* as just stated, is very common, and follows the bronchitis which usually is developed in the earlier stages of the disease. It is particularly apt to attack young children and to occur in the winter months. Sometimes a true lobar pneumonia develops.

In nearly all cases of whooping-cough a moderate degree of *compensatory emphysema* comes on because of the violent respiratory efforts of the patient, and rarely this strain on the tissues of the lungs results in the rupture of an air vesicle and the development of interstitial or interlobular emphysema. In other instances the quantity of air which escapes in this way is very large and infiltrates the tissues of the mediastinum, the subcutaneous tissues of the chest, and in extreme cases those of the entire body. Instances of this condition have been reported by Gelmo, Ferrell, and Bierbaum, and have usually proved fatal. Cases are also recorded in which *pneumothorax* has been produced. It is by no means uncommon, particularly among the children of the poorer classes, for whooping-cough to be followed by pulmonary tuberculosis, probably because of the lowered vital resistance of the patient. Another complication of importance, although it has been described as a symptom, is *vomiting,* which if it becomes constant is a serious condition, particularly in infants, since it may cause death from asthenia.

The *bronchial glands* are nearly always enlarged and may be so much increased in size as to cause dulness on percussion over the sternum. The area of cardiac dulness is increased by reason of the dilatation of the heart due to the strain thrown upon it in the attack of coughing.

Measles and whooping-cough are, as already stated, very commonly associated, but the whooping-cough complicates the measles more frequently than the measles complicates the pertussis. Sometimes in very young children the disease becomes so severe that the spasm of the cough seems to spread to all the muscles of the body and produce general convulsions. These cases are nearly always fatal.

Paralysis complicating whooping-cough is not common. It is usually in the form of a hemiplegia, and occurs either during the acute period of the disease or as a sequel. When it takes place during the paroxysmal period it is due in the majority of instances to meningeal or cerebral hemorrhage in all probability, although statistics as to this question are scanty. Twelve cases of cerebral hemorrhage due to whooping-cough have been collected by Townsend of which seven recovered, and Brown has reported a case in which he operated for the relief of cerebral compression due to this cause with excellent results to the patient. The literature of this subject has recently been analyzed by W. G. A. Robertson. Sometimes paraplegia or monoplegia has occurred during the stage of convalescence. The prognosis seems to be fairly favorable, indicating that the lesion producing these conditions cannot be permanent. Small *conjunctival hemorrhages* are not infrequent, and more rarely large extravasations of blood into the conjunctival tissues take place, amounting to ecchymoses. Still more rarely temporary *amblyopia* develops as a result of disordered circulation in the retina or possibly of an actual retinal hemorrhage.

Diagnosis.—The important points in the diagnosis of whooping-cough are the repeated and rapid coughs in series until the chest is almost emptied of air, followed by a sudden inspiration through the narrowed glottic opening. Some cases develop only the series of short coughs, and present no whoop afterward.

Prognosis and Mortality.—The prognosis in whooping-cough as in most infectious diseases, depends upon the age of the child, its general nutrition and vital resistance, and upon the care the child can receive. In general terms it may also be stated that the prognosis is not so good in winter as in summer, as fresh air is not so readily obtained and there is greater danger of exposure to cold in the winter months. In itself whooping-cough is not a fatal disease. Death is due to the complications which ensue, and if these can be prevented the patient always gets well. In very young children, however, it is almost impossible to prevent the development of bronchopneumonia, and this is a dangerous condition in proportion to the youth of the child. In London whooping-cough stands second as a cause of death from the infectious diseases in children under two years of age.

Hagenbach, of Basle, gives the following mortality statistics, which are based on the cases that came under his observation during a period of eleven years: under one year, 26.8 per cent.; between one and two years, 13.8 per cent.; between two and five years, 3 per cent.; between five and fifteen years, 1.8 per cent.

Holt states that the mortality for children under one year of age is 25 per cent.

Treatment.—It is vitally important that children who have whooping-cough should be put under the most favorable hygienic conditions as to sunlight, fresh air, and equable temperature. In the summer they do best out-of-doors when the weather is not too cool, if they are prevented from acting imprudently, as, for example, getting the feet wet. In winter they should be kept in a warm room, the temperature of which should be 70° night and day. The air of this room should also be moistened by liberating in it small quantities of steam obtained from a kettle of boiling water, from a croup kettle, or by dropping pieces of unslaked lime in a bucket of water. This is an exceedingly important measure if the room is heated by a furnace, since the air from the ordinary furnace is exceptionally dry and often laden with dust, and these two causes act as an irritant to the already irritated respiratory tract. When it is not possible to confine the child to a room which is heated evenly, a most excellent method of treatment, particularly in those cases where the paroxysms are frequent at night, is to place the child in a bronchitis tent. A bronchitis tent consists in throwing over a bed a large sheet which is supported several feet above the head of the child by means of broomsticks or poles, which are tied at each corner of the bed. This tent can be made quite attractive for children by decorating it. Into this tent, at the foot of the bed, may be discharged a small quantity of steam such as is given off from an ordinary kettle of water when it is kept constantly boiling. In this way the child's mucous membranes are not irritated by dry or cold air, but on the contrary are greatly soothed, and I have frequently diminished the number of paroxysms per day at least one-half by the institution of this plan of treatment, which has the additional advantage that it is prophylactic, and prevents the development of those serious complications like vomiting and bronchopneumonia, which are much aided in their development by repeated and violent paroxysms of cough. With a little attention a child may be kept in such a bronchitis tent night and day through the entire attack.

In the way of drugs there is no remedy so efficacious in diminishing the severity of the attack as small doses of antipyrin; that is to say, ½ to 1 grain of antipyrin every three or four hours to a child of one or two years, or 2 grains every three or four hours to a child of five or six years, care being taken that the drug does not too greatly relax the skin or depress the circulation. There is a widespread belief among the laity that quinine in small doses is not only a prophylactic against whooping-cough for other children in the family who have not as yet contracted the disease, but that it is also of curative value. Some physicians have used a spray of a weak solution of quinine in the throat with asserted advantageous results, but its value is doubtful, and its bitter taste makes its use impossible in a large proportion of cases.

The development of complications, such as bronchopneumonia, necessitates the institution of those lines of treatment which will be found suggested for that disease. For the relief of the individual paroxysms of cough several remedies may be employed, of which the best is probably chloroform. It is needless to say that this drug should be used with great caution, and the patient's parents and the nurse should be instructed never to use it on a cloth, but, when the paroxysm is threatened, to pour the remedy over the back of the hand and place the hand under the child's nose. Under these circumstances a sufficient quantity of the chloroform is often inhaled to relax the spasm, without producing any of the marked physiological effects which would certainly be obtained to an undesirable degree if the drug were poured on to a napkin. This method also prevents an overdose of chloroform being given, since the excess of the drug rapidly runs off the hand or evaporates. As the hurry of an approaching paroxysm often makes the attendant careless as to the quantity which is poured out of the bottle, the physician should insist that the chloroform be used in no other way than that which has just been described. If the paroxysms are too severe to be controlled in this way, nitrite of amyl may be occasionally employed.

An innumerable array of drugs have been recommended for the palliation and cure of whooping-cough. Suffice it to say that most of them are entirely useless. Even such powerful nervous sedatives as the bromides cannot act advantageously in many of these cases, and the use of more powerful ones such as chloral and opium are contraindicated for evident reasons. The physician should always remember that whooping-cough is a disease which is bound to run its course, uninfluenced in its duration by any treatment which he can employ. The most that the physician can do is to prevent complications, treat them if they arise, and endeavor to modify the frequency and severity of the individual paroxysms, being careful in so doing that the remedy is not worse than the disease, in the sense that it produces digestive or circulatory disorders which are distinctly disadvantageous.

INFLUENZA.

Definition.—Influenza is sometimes called *la Grippe*. It is a pandemic disease; that is, one which appears in widely separated parts of the world simultaneously. It is also highly infectious, and the infection is produced by the bacillus of Pfeiffer. Influenza of this type is to be separated, theoretically at least, from that condition sometimes called "common cold" or "coryza," which often causes somewhat similar symptoms in a milder form, although during the presence of an epidemic of *la Grippe* the differential diagnosis may be impossible. At the present time the term "influenza" is often employed when the physician is unable to reach a diagnosis, and as a consequence is greatly abused, particularly in the early stages of typhoid fever and tuberculosis.

Leichtenstern has divided the disease into two varieties, namely, true epidemic influenza (*influenza vera*) due to the bacillus of Pfeiffer, and endemic influenza due to the same cause and occurring for some years after an epidemic has been present. Both of these forms are to be separated from ordinary pseudo-epidemic influenza or an attack of ordinary cold in the head. A peculiarity of true influenza in its epidemic form is the large percentage of persons which it attacks within a short space of time, more than any other epidemic disease except dengue.

History.—At various times in the past great epidemics have broken out and raged over the entire world, and have been followed by long periods of immunity. Thus, when the great epidemic of 1889 occurred, only a few physicians, and they of advanced years, had ever seen a case, for the previous epidemic had occurred in 1847 and 1848.

Pandemics have occurred during the last century in 1830–33, 1836–37, 1847–48, and 1889–90. In 1889 the disease began in remote parts of Russia in October, reached Moscow in November, ten weeks later it got to Berlin, a month later to London, and soon after to New York and Philadelphia, and thence it spread all over the continent of North America. Within the next few months nearly the whole civilized world was affected by it. Since the last outbreak the disease has been endemic, but it is an attenuated form of the infection. An individual locality is rarely subject to an epidemic for more than two months, but sporadic outbreaks occur for a long period afterward.

Etiology.—It is interesting to note that the word influenza is derived from the Latin sentence *ab coeli occultes quadam influentia*—from some hidden influence in the sky. Influenza, if entirely dependent upon a micro-organism for its infectious character, must also be dependent upon certain telluric influences, at present unknown, which render the human race more susceptible to the effects of the germ at certain times or which render the germ more capable of producing infection at certain periods.

There are two chief factors involved in the production of an attack of influenza, namely, the presence of the bacillus, usually received directly from another patient by contact, or through the air; and, second, atmospheric states which are favorable to the growth of the germ or to the production of individual susceptibility. A third factor, always of importance in connection with infectious diseases, is the presence of pre-existing disease which decreases the general vital resistance of the patient.

The bacillus of Pfeiffer was first isolated by that investigator in 1892. The organism is small and non-motile, and can be well stained by Loeffler's methylene blue or by well-diluted watery solutions of carbol-fuchsin. It develops in myriads on the nasal and bronchial mucous membranes and in the secretions of those parts. A number of observers, and more particularly Ricciardi, have shown that the bacillus is readily distributed and spreads most actively by droplets of mucus. Even after the patient has recovered from an attack his nasal secretions may reinfect himself or other persons for a period of weeks, and therefore all handkerchiefs, towels, and pillowcases used by him should be boiled before being used by others. The room occupied by the patient should be fumigated with formaldehyde after his recovery occurs and before anyone else occupies it.

It is a noteworthy fact that during an epidemic of influenza other infectious diseases seem to be less common. This is particularly true of malarial fevers, if the statistics collected by Anders, of Philadelphia, are correctly interpreted.

Incubation.—The period of incubation is probably from twenty-four to seventy-two hours, but in some cases it seems to be longer than this.

Symptoms.—The onset of symptoms of epidemic influenza is nearly always sudden. A person feeling perfectly well may suddenly be seized by a sense of *chilliness* or a *severe rigor*, followed by severe aching pains in the back and in the legs. There is usually congestion of the nasal mucous membrane, so that the patient seems to have a severe cold in the head. The chill is quickly followed by *fever* which may rapidly rise to a point as high as 105°, although as a rule 103° is the more common acme. Associated with these early symptoms there is usually a sense of severe illness and a feeling of great wretchedness, so that the patient not only expresses himself as feeling very ill, but seems so to the physician.

About this time the symptoms are wont to be associated with additional ones indicating involvement of certain viscera. Most frequently the *respiratory system* is affected, and, in addition to more or less intense congestion of the nasal mucous membrane, an acute bronchitis develops; the physical signs in the chest being typically those of acute bronchitis with excessive, unproductive cough and a sense of thoracic soreness. When the nasal mucus is examined it is seen to be unusually thin, and if any bronchial mucus is expelled it is also of this character. As the disease progresses the sputum becomes greenish-yellow and thick.

The general state of the patient at this time is often one of *profound depression*, far in excess of that which usually accompanies such signs of bronchitis. The action of the *heart may be feeble* and the skin is relaxed and clammy, or it may be very hot and dry.

If convalescence is not soon established the disease often develops into a peculiar form of *pulmonary congestion* or *pneumonia*, in which the sputum may be blood-tinged and frothy or in which no sputum may appear. A peculiarity of this pulmonary involvement, in one of its forms, is the fact that it moves from place to place with remarkable rapidity. An area of impaired resonance which existed yesterday is clear to-day, and still another area of congestion develops elsewhere—a form of wandering congestion. When true pneumonia develops it may be croupous in type and be due to mixed infection by the bacillus of Pfeiffer and by that of true pneumonia, or it may be in the form of bronchopneumonia. The latter type is the more common, but both forms are apt to be serious and particularly so in the feeble, the aged, the very young, and in alcoholic or renal cases. Pneumonia and heart-failure due to an action of the toxin of the disease on the heart muscle are the chief causes of death in all epidemics.

Pleurisy followed by empyema is not very rare.

In studying a case of influenza accompanied by pulmonary signs the physician must always bear in mind the possibility of the presence of associated tuberculous infection, because an attack of influenza not only often predisposes to this disease, but in addition permits unrecognized foci of early tuberculous infection to become active.

In some cases of influenza the *heart* seems to bear the chief brunt of the attack, so that repeated attacks of syncope ensue. These instances are met with chiefly among patients who have persisted in remaining at work during the early stages of the disease, or who have had, previous to the attack, an impaired heart muscle. Thus, a heart dilated as the result of excessive exercise may succumb readily, or one in which early but hitherto unrecognized degenerative changes were developing may suddenly fail. Often the symptoms of influenza are chiefly gastrointestinal or nervous.

The *gastrointestinal form* of the disease may have its onset in severe diarrhœa and vomiting, with collapse and violent abdominal pain. In some cases the pain is entirely absent, and profuse watery stools are present. *Jaundice* may be present, due to an extension of the gastrointestinal catarrh to the common biliary duct.

In the nervous form the symptoms consist of profound *nervous* and *mental depression*, or in severe neuralgic pain which may or may not be due to neuritis. *Mental disturbances* in the course of an attack of influenza are by no means rare. Indeed, it may be said that no other acute infectious disease is commonly so complicated, or followed, by such a condition. Leichtenstern states that he met with fewer psychoses among 2000 cases of typhoid fever and 3000 cases of pneumonia than he found among 439 cases of influenza. These psychoses may be of the exhaustion type, but usually are due to a toxic state induced by the malady. The mental disturbance may develop during the stage of onset, the febrile stage, or the stage of decline or convalescence. The latter cases are usually of the exhaustion type. The prognosis in these cases as to the state of the mind is usually good unless there is a bad history as to heredity.

Very rarely *meningitis* develops, and still more rarely true encephalitis. Cases of cerebral abscess have also been ascribed to this disease. In rare cases a *toxic neuritis* develops, and this may be single or multiple.

Even paraplegia due to this cause may arise. Not only may this type of influenza affect the nerves of sensation and motion, but specialized nerves such as the vagus, thereby causing disturbances of the circulation such as paroxysms of tachycardia and bradycardia. In an analysis of 29,000 cases Lee found that 7000 were of the nervous type. In some instances the disturbance of circulation is due more to an influence exercised upon the vasomotor nervous system than to any direct effect upon the nerve supply of the heart, so that attacks of syncope come on from acute vascular relaxation.

Complications and Sequelæ.—It is difficult to separate the complications of influenza from the ordinary symptoms of the disease because the natural course of the disease presents such diverse manifestations in different organs. Without doubt pulmonary, cardiac, and renal disorders are the most common complications. In many cases death is due to an attack of pneumonia, which rapidly carries off the patient whose vitality is already sapped by the onset of *la Grippe*. In other instances the kidneys, which have been impaired before the attack, suffer from an acute congestion or true nephritis superimposed upon the subacute or chronic state, and so uræmia speedily comes on, with its helpmate, pulmonary œdema.

Patients with influenza develop *cardiac complications* in three chief classes: either they already have mild cardiovascular degeneration which enables the influenzal toxin to work havoc with the cardiac muscle, or they have dilated feeble hearts, or, again, as already stated, they persist in remaining at work after the attack begins and refuse to go to bed. These patients not only have serious cardiac difficulty during the attack, but very frequently suffer from cardiac weakness and distress for many weeks after convalescence should be well established. The man who persists in remaining out of bed when attacked by this disease literally "takes his life in his hand."

The German collective investigation of the epidemic of 1889–90, based on an analysis of 3185 cases, gave the following results as to the relative frequency of complications, which results, however, differ materially from clinical experience in America so far as the cardiac complications are concerned:

1. Diseases of the respiratory organs, exclusive of pneumonia, 48.76 per cent.

Of these complications pleurisy was the most frequent, being present in 27 per cent. of the entire number of cases. Pneumonia was present in from 6 to 8 per cent. of all cases.

2. Diseases of the nervous system, 45.77 per cent.
3. Diseases of the ear, 37.95 per cent.
4. Hemorrhages, 25.33 per cent.
5. Diseases of the heart and vascular system, 14.09 per cent.
6. Diseases of the digestive organs, 10.36 per cent.
7. Polyarthritis, 7.28 per cent.
8. Diseases of the eye, 7.03 per cent.
9. Albuminuria and nephritis, 4.52 per cent.

Diagnosis.—It is so easy to make a diagnosis of influenza during the presence of an epidemic that physicians are wont to be careless in examining the patient thoroughly, and so may overlook complications of importance or decide that the case is one of influenza when in reality the chills, the fever, the aching, and the prostration are due to an oncoming typhoid fever or an acute tuberculosis or malaria. All of these diseases, and also ulcerative endocarditis and sepsis, should be carefully excluded before a diagnosis of influenza is made.

Treatment.—Above all other things in the treatment of influenza is rest in bed. This is as true of mild as of severe cases and of the patient who is stalwart as of the patient who is feeble. A robust man who fails to rest almost always suffers from a severe attack or from sequelæ, such as cardiac disorder and giddiness, which may invalid him for weeks. Aside from rest in bed little medicine is needed except for the purpose of relieving symptoms which are troublesome. For the relief of the excessive pain in the back and limbs the coal-tar products have been employed by the ton. Although they give ease they are harmful if the doses are large, and often fail if they are used in moderate amounts. They tend to increase nervous and circulatory depression, and to decrease the ability of the patient to resist the infection from which he is suffering and the possible secondary infections which may occur. If the patient will rest they may be used moderately; if he will not rest they should not be used, for they not only do harm directly, but by diminishing discomfort they also enable and encourage him to remain out of bed.

A very useful drug for the relief of the aching and pains in the back and limbs is salicin in 5 grain doses every five hours in capsules. Many practitioners believe that this drug alone, or when combined with 2 grains of quinine, acts as a specific in the cure of the affection. Should the pain in the back be intense it may be relieved by the application of hot stupes or compresses or by rubbing with soothing liniments. A more ancient but nevertheless very useful remedy for this condition, particularly in the early stage of the malady, is Dover's powder in the dose of from 2 to 10 grains once or twice a day. At one time used as a matter of routine in all infections, it has fallen into an undeserved disuse.

Headache, if it be due to congestion, may be modified by the use of an ice-bag, or by the administration of 1 to 2 grains of caffeine with 5 grains of bromide of sodium or potassium every few hours. This formula can be given in the form of an effervescent granular salt without the use of the coal-tar products often added by manufacturers of headache cures. Hot foot-baths also decrease headache. Menthol pencils may be used locally for neuralgia or a spray of chloride of ethyl may be used locally for the same purpose.

As in all infectious maladies, it is of the greatest importance that the organs of elimination be kept active. The bowels may be first moved by a grain or two of calomel, followed in twelve hours by a Seidlitz powder, or, if constipation has been marked, they may be opened at once by citrate of magnesium. For the purpose of keeping the kidneys active 5 grains of citrate of potassium or of bicarbonate of potassium may be given every four hours in copious draughts of water if the urine is acid, or the same amount of benzoate of ammonium if the urine is alkaline. The latter drug is best given in capsules, and possesses the additional advantage of acting favorably upon the respiratory mucous membrane and upon the muscular pains. A hot compress or poultice applied over the loins will often establish renal secretion when it seems scanty.

Dryness and soreness of the mucous membrane of the respiratory tract, in the stage of onset, may be much relieved by telling the patient to inhale steam which may be medicated by the addition to the water from which it arises of a few grains of menthol or of equal parts of menthol, oil of eucalyptus, and oil of pine. In other instances the patient may add to the boiling water a tablespoonful of compound tincture of benzoin. The medicated steam may be taken directly from an inhaler or the vapor may be set free in the air of the room by the use of a bronchitis kettle. When the nasal mucous membrane is so congested and occluded that breathing is difficult and oppression is marked, adrenalin chloride, 1:5000, with chloretone may be sprayed into the nostrils or applied on pledgets of cotton. It loses its effect if applied too often, but it does not do so as rapidly as does cocaine, nor is it dangerous in its systemic effects.

For the relief of the congestion of the respiratory mucous membrane, when the illness has lasted for several days and the secretion is thick and tenacious, chloride of ammonium in 5 grain doses four times a day may be administered combined with codeine or heroin to relieve cough, or terpin hydrate may be used with the same sedatives in the form of the well-known elixir of terpin hydrate with heroin. For the persistent cough of convalescence, oil of sandal-wood in 5 minim doses four times a day is very useful.

Circulatory and nervous stimulants are not to be used unless there is distinct evidence of their need. Alcoholic drinks are as a rule to be excluded, unless the patient uses them habitually when well, when they have to be given, preferably in the form of an old brandy or good whiskey. Great care must be taken that the patient does not overuse them in his endeavor to make himself feel stronger. For acute circulatory failure aromatic spirit of ammonia or Hoffmann's anodyne are the remedies of choice. When the failure of the circulation is associated with nervous depression the use of strychnine is indicated, but it is greatly abused and should not be given day after day except as a tonic in convalescence, as it loses its power, is not a true stimulant but a nervous irritant, and often causes great irritability if not employed skilfully.

As influenza is a disease which produces great prostration, a diet which is easily digested and nutritious is essential for the maintenance of strength, particularly in the very young, very feeble, and very old. Animal broths, oysters, and predigested foods are useful, and they may be fortified with advantage by barley-gruel, the digestion of which may be aided by the use of taka-diastase. Indeed, the various vegetable gruels with taka-diastase are in many cases better than the animal broths. Arrowroot and milk-toast and eggs are also useful.

Prophylaxis.—There can be no doubt that much can be done to prevent the spread of *la Grippe* from one person to another by isolating the ill and by forbidding healthy persons to occupy the sick-room after it is vacated, until it is thoroughly disinfected. This is particularly advisable when the old and feeble are about the house and when persons who are still weak from one attack are exposed. Every effort should be made to keep the malady out of the non-medical wards of hospitals, insane asylums, and almshouses. Patients in these institutions when taken ill should be isolated.

All rooms, clothes, and books used by patients suffering from influenza should be disinfected as carefully as if the patient had suffered from some more fatal malady.

DENGUE.

Definition.—Dengue is an acute infectious, but non-contagious, usually epidemic fever, which is probably dependent for its development upon the presence of some specific organism the exact nature of which is still obscure, although McLaughlin and Graham believe that they have succeeded in isolating it. The disease is characterized by two febrile attacks with severe pains in the muscles and joints. Because of these latter symptoms it is often called "breakbone fever," and from the peculiar gait caused by this condition "dandy fever." A large number of other popular names have been given it, such as "three-day fever," "bouquet fever," or sometimes, as a corruption of the last name, "bucket fever."

History and Distribution.—The earliest accurate description of dengue that we possess is that of Brylon, who described the outbreak of 1779; later the celebrated epidemic in Philadelphia, in 1780, was described by Rush. Since then it has occurred in a considerable number of epidemics in various parts of the world, such as Batavia, Spain, India, Bermuda, Brazil, the West Indies, and in various parts of the Southern United States. Within twenty years it has also visited Turkey, Greece, Fiji, and Tripoli. It is, however, distinctly a disease of warm climates, and, so far as I know, has never been met with north of Philadelphia. The disease spreads from point to point along lines of travel, being carried by infected individuals and perhaps by clothing.

A peculiarity of dengue is the rapidity of its spread and the few people in a community who escape its attack. In this respect it surpasses epidemic influenza. No age sex, or race escapes, and in an incredibly short time after the first case is seen a multitude may be down with it. As Manson well says, it "bursts" upon a place. The spread of an epidemic is always arrested by the appearance of cold weather. High altitudes are also unfavorable to its spread.

Pathology and Mode of Propagation of Dengue.—H. Graham, of Beyrouth, has observed in the blood of dengue patients an unpigmented, oval-shaped protozoon, having amœboid movements, and frequently showing a sharply pointed extremity such as is seen in the parasite of the so-called Texas fever. This parasite is of lighter color than the red blood corpuscles in which it constantly changes its position. Dr. Graham also conducted experiments for the purpose of determining whether the disease is propagated by mosquitoes, as a result of which he came to the conclusion that the *Culex fastigans* carries the parasite from the sick to the well.

In several instances Graham placed persons suffering from dengue in apartments in which all mosquitoes had been destroyed by chlorine gas, and allowed healthy individuals to associate with the sick. In no case of this kind was the disease contracted. In addition to this negative evidence Graham offers positive evidence which he obtained by allowing mosquitoes which had bitten affected persons to bite two healthy individuals who resided in a district where no cases of dengue were present. Both of these men developed the disease, one on the fourth and the other on the fifth day after they were bitten. They were kept under mosquito nettings until they had completely recovered and the infected mosquitoes were all killed. No other cases of dengue occurred in the village where these experiments were made. The same organism which was found in the blood of dengue patients was also found in the blood and the stomach of *Culex fastigans*, even up to the fifth day after the insect had bitten an infected individual.

The organism multiplies by sporulation in the stomach walls and veno-salivary glands of the mosquito. Inoculation of a human subject with a solution of the salivary glands of an infected mosquito produced, on the third day, a chill and high fever followed by a typical attack of dengue. As yet Graham's observations have not been confirmed.

Symptoms.—Dengue is characterized by a train of symptoms which is quite remarkable. In the first place, the *suddenness of its onset* is noteworthy. A patient may be in perfect health at one hour and sick in bed with well-developed symptoms the next. In any event the *onset is sudden*, and sometimes it is ushered in by a *chill* or by pains in the limbs. *Fever* rapidly develops and may reach as high as 106° or 107°, but usually the acme is 103° to 105°. There is *intense headache* and the *pains* in the limbs are so excruciating that the term "breakbone fever" is well applied. The discomfort of the patient is increased by the pain caused by moving the body. The *tongue is usually heavily coated*, and nausea and vomiting may be distressing symptoms.

With the onset of the fever there develops a rash which is of the nature of *erythema*. In from one to three days, usually two days, the fever suddenly ends by crisis and simultaneously the patient not only sweats freely, but also has free *diuresis*, *diarrhœa*, and *nose-bleed*. This nose-bleed, by relieving the cerebral congestion, greatly decreases the headache, and the rash rapidly fades.

In other instances the fever gradually falls by lysis, but this is less common than crisis. The fever having fallen to normal the patient, still feeling weak, is able to be about, although he suffers from twinges of pain in the joints and muscles, which impress upon his mind the fact that he is as yet ill. After a remission of several days, usually from two to four, the *fever returns* with some violence, but it is rarely as severe as in the primary paroxysm, and it usually lasts only a few hours. With the appearance of this secondary fever a roseolous rash develops, and with its development the patient may have a return of his bone and joint pains to a very severe degree. Although the fever soon disappears the rash lasts for several days and may end in a slight desquamation. Taking it all in all, the secondary attack is usually much milder than the first.

The rash of the second attack is *roseolous*, and is peculiar in that it is usually first seen on the hands, both the palmar and extensor surfaces, and thence rapidly spreads to the entire body. The spots are as large as a pea, circular in appearance, dusky red, and perhaps elevated. As the disease progresses they may coalesce, leaving patches of healthy skin between. This rash is more apt to be profuse and to coalesce around the joints than elsewhere. The roseola fades as it begins, first on the hands, then on the arms and body, and lastly on the legs. The *desquamation* may last for weeks, but it is so fine that it may be overlooked. The skin never peels as after scarlet fever. In some instances the patient passes on to rapid convalescence after the terminal or roseolous rash fades, but in others he remains miserable for a long time from wandering pains in his joints or in the soles of his feet. The muscles are sore on pressure and stiff on moving after a long rest, and debility may be persistent. In some instances insomnia or furunculosis delays complete recovery.

In certain epidemics there is sufficient degree of swelling and redness about the joints to suggest the presence of acute rheumatism.

Relapses of dengue occur not infrequently.

Diagnosis.—Dengue may be separated from rötheln, which it resembles during the period of its secondary rash by the lymphatic swellings of the latter disease. The differentiation is also accomplished by the sudden severe onset and the pain in the joints. It is distinguished from scarlet fever by the lack of sore throat and the peculiar scarlet hue of that disease, and from syphilitic roseola by the absence of a history of venereal infection, and the fact that associated symptoms of the early secondary stage of syphilis are absent. On the other hand, it is to be recalled that many syphilitics, with the onset of the roseola of that disease, suffer from a chill and general wretchedness with pains in the bones. Influenza is separated by the absence of catarrhal symptoms and by the presence of the rash in dengue. Acute articular rheumatism and malarial infection are two other diseases which must be borne in mind when the diagnosis of an individual case is in question.

Prognosis.—The prognosis in a case of dengue is always favorable if the patient, prior to the attack, is in good health, and not debilitated by some other malady or old age. Death may be said not to be known as a result of this malady in ordinarily healthy persons. Convalescence, after a severe attack, is, however, very often quite slow, and if the patient is living in a hot climate recovery may not be complete until a change of residence is made.

When dengue attacks the aged and feeble, or very young children, it sometimes indirectly causes severe illness and death by predisposing the patient to other infections so that there develops a severe bronchitis or bronchopneumonia, or some other evidence of acute infection. In such cases the prognosis depends chiefly upon the character of the secondary ailment.

Treatment.—In discussing the treatment of this disease it is to be recalled that it presents very different degrees of severity in different cases. In many persons the symptoms are so mild that the patient seems scarcely at all ill, and in others the manifestations are so severe that convulsions and unconsciousness may be present. In the mild cases no drugs are needed, but in the severe cases active treatment may be essential. In general terms it may be stated that the treatment of the patient suffering from dengue consists in absolute rest in bed from the earliest stage of onset till the conclusion of the second stage of fever. Indeed, the longer he will consent to rest in bed after the fever develops, the more rapidly will complete convalescence be established.

So far as drugs are concerned, there are no specifics for this disease, which, if permitted, will usually run its own self-limited course to recovery. When the pains are intolerable they may be controlled by moderate doses of morphine given hypodermically or by the use of acetanilid or phenacetin. A gentle antipyretic and sedative mixture, containing 5 grains of potassium citrate and 30 minims of sweet spirit of nitre in a dessertspoonful of water, is useful to keep the kidneys active. An ice-bag may be applied to the head to relieve the cephalalgia, and if the face is very much flushed and the head throbs a hot foot-bath is advisable. Sometimes a hot bath is useful to develop the rash and relieve the pains in the body and limbs. In these cases the salicylates may also be used for the same purposes, 20 grains of sodium or strontium salicylate, or of aspirin, being given every three or four hours.

When the circulation is strong and full McLaughlin asserts that large doses of tincture of gelsemium serve to quiet the excited pulse and to relieve the neuromuscular pains. The dose he recommends, namely, 20 to 30 minims every three or four hours, seems to the writer much too large and capable of causing serious depressions; but as McLaughlin has had large experience with the disease, his views demand respectful attention. The fever is rarely sufficiently high or prolonged to require treatment. Should it require attention tepid spongings are usually sufficient to control it within safe limits; but should it reach as high as 105° or more, then it must be reduced by cold spongings, or even by the use of the cold bath, with active frictions. Should nervous symptoms be very manifest and convulsions be threatened, chloral should be given in the dose of 5 grains by the mouth, or 10 grains by the rectum, if the patient is a child, and bromide of sodium used to aid it in its sedative action.

The patient should be urged to drink freely, if his stomach will retain liquids, in order to keep his kidneys active in eliminating the poisons of the disease. When the stomach is not retentive a pint of cold water may be given by the rectum every eight hours. Should diarrhœa be troublesome it can be best controlled by giving castor oil to cleanse the bowels, and following it by opium.

CEREBROSPINAL FEVER.

Definition.—Cerebrospinal fever, sometimes called "cerebrospinal meningitis," "spotted fever," or "petechial fever," is an acute, often malignant, infectious, but rarely contagious disease, due to the diplococcus of Weichselbaum, which is sometimes called the meningococcus or the *Diplococcus intracellularis meningitidis*. It is characterized by a rapid course, rigidity of the neck, retraction of the head and the formation of inflammatory exudates under the membranes which cover the brain and spinal cord. It is to be clearly understood that a number of pathogenic micro-organisms are capable of producing inflammation of the pia arachnoid, and consequently all the symptoms of true epidemic cerebrospinal meningitis. Such cases are not instances of this disease, but rather are to be considered as sporadic cases of meningeal infection. Indeed, it is a noteworthy fact that the sporadic cases of cerebrospinal meningitis which are due to the pneumococcus, are prone to be more virulent than those due to the specific organism just named. While the epidemic form has been proved to be always due to the *Diplococcus intracellularis meningitidis*, it is not correct to call all cases of cerebrospinal meningitis instances of cerebrospinal fever.

History.—No definite description of this disease is to be found in medical literature prior to the nineteenth century. In 1805 the first case was described by Vieusseux, in Geneva, Switzerland, where several deaths took place from the disease. In America it first appeared in Medfield, Massachusetts, in 1806. During the next ten years the malady broke out in different parts of Europe and America, but disappeared after 1816 till 1822, when it reappeared in France. In 1828 it broke out in Ohio. It was not, however, till 1839 that it became sufficiently prevalent in any one place to cause a very large number of deaths. In that year, at Versailles, it ravaged the town and garrison and produced a mortality of nearly 75 per cent. Scattered epidemics have since occurred in the United States at intervals of every few years, and it is constantly present in scattered cases in the central part of the State of New York. (Elsner.) A noteworthy point in connection with the disease is the fact that it suddenly appears simultaneously in widely separated areas, and without any dependence upon lines of travel. Thus during a recent period of twelve months many cases occurred in New York, but none in Philadelphia, which is only ninety miles away. Certain atmospheric influences may make this possible, but its cause is not definitely understood.

Etiology.—There can be no doubt that cerebrospinal fever is due to the diplococcus already named, but the same anatomical conditions and a similar clinical picture may be produced by other bacteria, for example, the pneumococcus and other pyogenic cocci. In cases of cerebrospinal meningitis which have appeared sporadically and presented all the signs of the epidemic disease the streptococcus, the staphylococcus pyogenes, the pneumococcus, the gonococcus(?), and even the bacilli of influenza and typhoid fever have been found as apparently the only cause of the affection. A similar acute serofibrinous meningitis may accompany pyæmia or septicæmia, or may be due to injury, with infection or extension of infective processes from the frontal, ethmoidal, sphenoidal, or mastoid sinuses, middle or internal ear, or other parts of the envelope of the brain and cord. Such forms of meningitis are often called consecutive, incidental, or secondary, and are to be distinguished from the epidemic malady.

Infection probably takes place through the respiratory passages, particularly in the nose.

Climatic conditions undoubtedly exercise some influences, for the disease confines itself almost entirely to the colder parts of the temperate zone, but this is not to be taken as indicating that it is a disease of the winter months. On the contrary, it appears about equally frequently in winter and summer. While it is true that unhealthy surroundings favor all diseases, it is also true that they do not seem to greatly influence this malady, for it occurs on high and on low land, when it is dry and when it is wet, on hill and in marsh, with equal frequency. As Stillé says, "It has passed by large cities reeking with all the corruptions of a soil saturated with ordure and populations begrimed with filth, to devastate clean and salubrious villages and the families of substantial farmers inhabiting isolated spots."

The disease affects children and young adults far more frequently than persons in advanced life. It is slightly contagious, but cases of undoubted transference from one patient to another occur. The occurrence of the malady in a number of persons living in the same district is usually due to the fact that they have all been exposed to the same cause.

Prevention.—We know of no method of preventing epidemic cerebrospinal meningitis, but physicians and others who are attending cases, should wash the nasal mucous membrane with normal salt solution to aid in preventing infection. Cases should be isolated.

Frequency.—From what has already been said, it is evident that this disease is met in epidemic form, but is comparatively rare. Many practitioners never meet with a single or sporadic case in a long career; whereas, others may be so unfortunate as to meet several outbreaks.

Pathology and Morbid Anatomy.—In fulminating cases death may occur before the meningeal exudate forms; in these the meninges may exhibit no exudate, showing only intense hyperæmia and œdema, but the membranes and cerebrospinal fluid are usually rich in the specific organism. Death in such cases seems to depend on the toxic action of the serous bacteria-laden exudate.

The characteristic lesion of this disease is an acute inflammatory exudate of the pia-arachnoid enveloping the brain and spinal cord. These membranes become infiltrated, and the surface appears to be covered by a white or creamy-white exudate, which is most conspicuous in the sulci. The ventricles may contain a cloudy, opaque, or even distinctly purulent fluid. The inflammatory exudate is most copious at the base of the brain and on the dorsal surface of the spinal cord, particularly in the lower thoracic and lumbar regions. When the disease affects children the lateral ventricles are often found at autopsy to be distended with purulent fluid, but in adults this condition is not likely to be marked. In the early stages of the disease the diplococcus is found in large numbers in the leukocytes contained in the exudates, but when death occurs late in the course of the malady the germ may be demonstrated with difficulty, if at all.

In addition to the lesions in the meninges the nerves and ganglia exposed to the toxic action of the exudate undergo inflammatory and degenerative changes. The involvement of these nerves may leave irreparable damage, manifested by blindness, deafness, or other phenomena dependent upon the structures involved. Secondary alterations in other parts of the body may be present. These are due to the toxins of the disease or to the presence of the micro-organisms in the affected areas. Thus, we find petechiæ in the skin and mucous membranes and somewhat similar punctate extravasations of the blood in the endocardium. Not rarely multiple abscesses are found scattered through the body and multiple suppurative arthritis may be present. Hyaline and granular degeneration of the voluntary muscles is also demonstrable, and the heart muscle, kidneys, and liver may manifest necrotic, degenerative, or inflammatory changes. Occasionally there is found, associated with the meningitis, croupous pneumonia, ulcerative endocarditis, and otitis media. These pathological conditions are characteristic of the severe forms of the disease.

In some cases the lesions are much more moderate in that hyperæmia, or intense congestion, of the pia mater only is seen, although the sulci between the convolutions of the brain may contain fibrin or pus.

Incubation.—The period of incubation is from one to four days.

Symptoms.—The symptoms of epidemic cerebrospinal meningitis may be grouped into five classes—viz., the moderate, the malignant, the intermittent, the typhoid, and the chronic form.

In the *moderate form*, after an unknown period of incubation, the patient suffers from a sudden chill, which may be preceded by headache and dizziness. The *headache* rapidly becomes very severe and is accompanied by severe pain in the back and down the back of the thighs, the muscles of which are often tense or fixed. The fever which follows the chill is usually moderate, rarely exceeding 102°, and it presents no characteristic curves. On the contrary, it is exceedingly irregular and does not show any constant morning and evening variations. Very rarely hyperpyrexia may develop.

As the disease develops the tenseness of the muscles of the legs extends to those of the back, neck, and arms, and, finally, they may become almost rigid, and contracted to such a degree that the patient develops opisthotonos. The abdomen is rigid and scaphoid. Not rarely spasmodic movements of the muscles of the face develop as the result of irritation of the roots of the cranial nerves, and by reason of this same cause *strabismus*, *ptosis*, *amaurosis*, and *diplopia* may be present. The conjunctivæ are usually reddened.

Delirium is a very frequent symptom, and is sometimes so severe as to be maniacal. From this state the patient may pass into coma.

The *pulse* and *respiration* are not greatly affected, except that as the disease progresses they may become feeble. Toward the end of the attack, if it be fatal in its nature, Cheyne-Stokes breathing may develop and the pulse become rapid and small.

An *eruption* develops on the skin in about one-half of the cases. When it appears about the mouth it is herpetic, but on other parts of the body it is usually petechial, although herpes of the skin of the trunk and about the genitals may appear. At times a general erythema may be present or in its place an urticaria is developed.

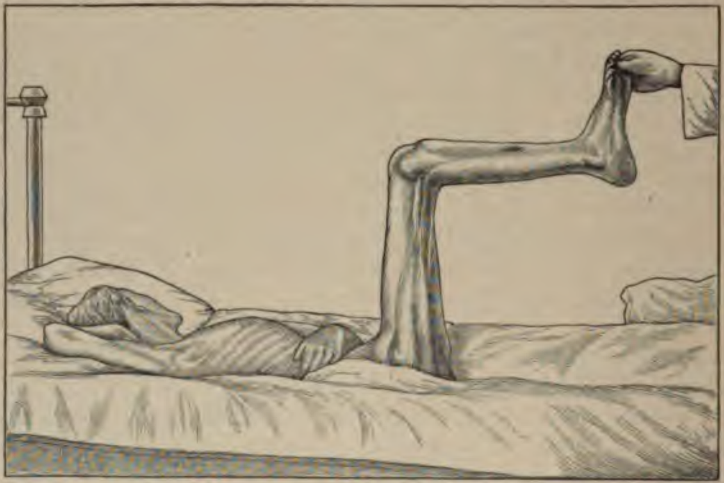
The presence of *arthritis* has already been referred to. It appears in about 20 per cent. of the cases, and, as it is septic in nature, it may cause serious changes in the joints and result in permanent deformity if the patient survives.

The *blood* shows no marked changes, save that the inflammation of the meninges results in a leukocytosis of the polymorphonuclear cells.

As an almost constant symptom, mention should be made of "Kernig's sign," which consists in the fact that in inflammatory processes in the membranes of the cord it is not possible to extend the leg on the thigh when the thigh is at right angles to the plane of the body. Rarely this sign is unilateral (Fig. 29).

In the *malignant type* of the disease the onset is remarkably sudden. The patient is seized by a chill, followed by headache, unconsciousness, and death. Convulsions occur more commonly in children than in adults. The fever may be absent, the pulse slow, the breathing labored, the urine greatly decreased in amount and loaded with albumin, and the stupor profound. The patient in such an instance is probably overwhelmed by toxæmia, so that death may ensue in a few hours.

FIG. 29



Kernig's sign, showing the strong contraction of the flexors on attempting to extend the leg. (After Osler's case.)

In the *intermittent form*, which is probably due to the *Streptococcus pyogenes*, or *Staphylococcus pyogenes* alone, or to association of those organisms with the specific coccus of Weichselbaum, the fever intermits, as in malarial fever, but the intermittence is irregular, as in sepsis, and is not distinctly periodic, as in malaria. The *typhoid form* is characterized by symptoms of apathy, feebleness, and abdominal disorders.

The *chronic form* consists in the prolongation of the ordinary type, with special symptoms, such as headache, pains in the nerves, vomiting, and progressive emaciation, with secondary arthritic changes and increasing inability to move the limbs. Here, again, it is probable that the maintenance of the illness is due to septic organisms rather than to Weichselbaum's coccus.

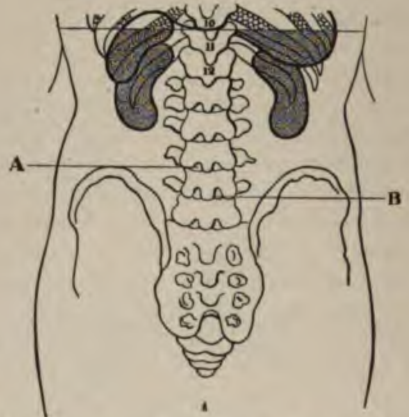
While for the sake of description these several types of the disease have been named, it is, of course, true that it may manifest various degrees of severity in the same case at different periods. Some cases which seem quite severe at the onset gradually ameliorate and pass into the chronic or subacute form. The fact that the malady presents widely different types is well illustrated by the seemingly exaggerated, but nevertheless correct, statement of Hirsch that the duration of epidemic cerebrospinal meningitis may be between several hours and several months. N. S. Davis has stated that its duration in his experience varied from twenty-four hours to twenty-eight days. I have seen death occur in eighteen hours.

Complications and Sequelæ.—The complications and sequelæ of epidemic cerebrospinal meningitis are very numerous. During the attack *croupous pneumonia* not only often develops and aids materially in producing a fatal issue, but acute pleurisy also is not uncommon. So, too, inflammation of other serous membranes, such as the pericardium and the endocardium and the synovial membranes, is often met with, because the coccus has an affinity for these membranes in all parts of the body. In the *nervous system* the most common sequelæ are blindness or impaired vision due to optic nerve atrophy, ptosis due to oculomotor paralysis following neuritis or to changes arising from the inflammatory exudate at the point where the nerves leave the membranes, and deafness arising from the effects of the acute inflammation or infection upon the auditory nerves. Sometimes the *deafness* arises from an otitis media due to the specific coccus. Aside from chronic nasopharyngeal disease and scarlet fever, this disease is responsible for deafness in larger proportion of cases than any other malady.

Diagnosis.—While it is true that in a majority of cases the diagnosis of this disease is readily made, it is also a fact that many other diseases may produce symptoms which so nearly resemble those of epidemic cerebrospinal meningitis that it may be absolutely impossible to make a differentiation. In the first place, it must not be forgotten that cerebrospinal meningitis is, as its name implies, an inflammation of the cerebrospinal membranes, and this change may be produced by a host of causes, none of which have any true relationship with the true epidemic form of the disease. As already pointed out in this article, and in that on typhoid fever, the bacillus of Eberth may cause a train of symptoms and morbid changes which is identical with that due to the diplococcus of Weichselbaum, yet such a case would not be one of epidemic cerebrospinal meningitis. It is evident, therefore, that cases of retraction of the head, rigidity of the limbs, and twitchings of the face should not be called true cerebrospinal fever unless the specific diplococcus can be demonstrated, or unless the disease can be found to be present in other patients in the vicinity. In the midst of an epidemic of typhoid fever the development of cerebrospinal symptoms should be credited to this infection rather than to the specific fever now under discussion. If any doubt exists as to the true nature of the affection, it should not be forgotten that herpes is very rare in typhoid fever and in typhus fever, but is common in true cerebrospinal fever. Both these fevers run a course which is marked by a natural limit; whereas, epidemic cerebrospinal meningitis does not begin to decline after the lapse of a definite course, but is exceedingly irregular in its duration.

Croupous pneumonia is the infection, above all others, which is capable of misleading the physician in his diagnosis of cerebrospinal fever. It has already been stated that pneumococcus is often found to be the cause of inflammation of the meninges, and in children in particular the cerebrospinal symptoms may be so well developed that unless the physician examines the lungs very carefully, he may diagnosticate cerebrospinal meningitis when in reality the true cause lies in the lung. It would seem that two types of cerebrospinal symptoms develop in pneumonia, namely, those due to the secondary meningeal infection with the pneumococcus and those in which there is no true infection, but simply irritation produced by the toxæmia of the pneumonia.

FIG. 30



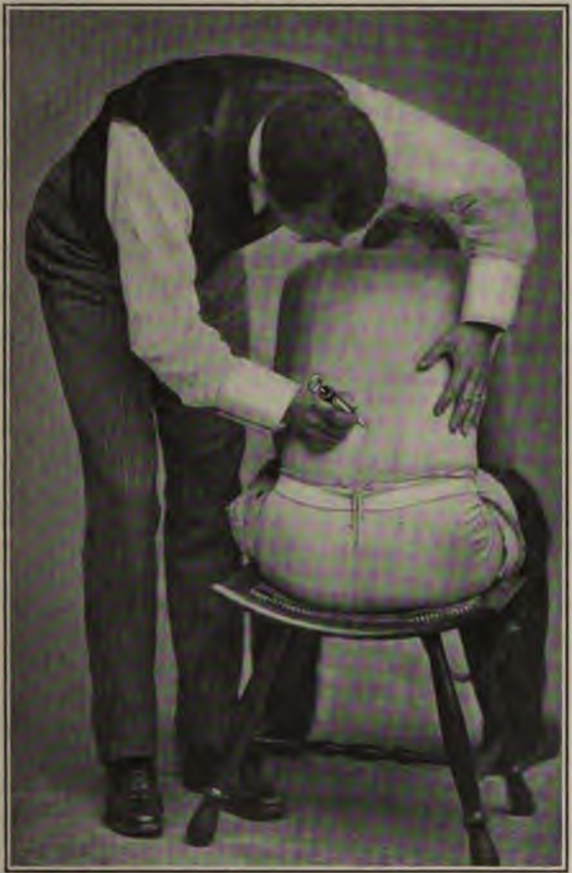
A, space between the third and fourth lumbar vertebræ which can be used for puncture; or *B*, the space between the fourth and fifth lumbar vertebræ.

Tuberculous meningitis is practically never so sudden in onset as is the true epidemic form, and careful physical examination of the patient will usually reveal a primary tuberculous focus if meningeal tubercles are present. When the inflammation is tuberculous the leukocyte count is not materially increased, whereas, in the specific type it may vary from 9000 to 26,000.

When cerebrospinal symptoms develop in the presence of an epidemic of influenza, the differentiation between true cerebrospinal meningitis and that due to influenza may be impossible, although the fact that the case is single points to the influenza bacillus as the true cause rather than that the attack is a sporadic case of the disease now under discussion. The cerebrospinal symptoms of influenza are rarely so severe or so persistent as those due to epidemic cerebrospinal fever.

The greatest aid that we have in differential diagnosis is by means of lumbar puncture. This operation consists in inserting a large hollow needle between the third and fourth or fourth and fifth lumbar vertebræ, a little to the side of the median line and just below the spinous process. The needle as it enters should be directed upward and inward. In children the fluid is reached when the needle is inserted about 2 cm., and in adults when it has reached the depth of from 4 to 6 cm. As soon as the membrane containing the fluid is punctured it flows from the needle in drops, which should be caught in a sterile test-tube in such a way that the fluid does not run down its side. If the infection is due to the specific organism, the pressure is greatly increased, so that the fluid may escape with a spurt. This fluid is clear if tuberculous meningitis is present, but cloudy if the diplococcus of Weichselbaum is the cause of the illness. Under these circumstances, too, the faint trace of albumin found in the normal fluid is very distinctly increased. The careful staining of a single specimen or a more exhaustive bacteriological examination may reveal the presence of the diplococcus.

FIG. 31



Introduction of needle between the last two lumbar vertebræ. The syringe is used as a convenient handle for the needle, and is unscrewed after the puncture is made.

Demonstration of the meningococcus in the nasal mucus is an important addition to other signs, but a number of observers, and more recently Lord, have shown that meningitis may occur without the specific coccus being present in the nose, and also that a meningococcus rhinitis neither preceded nor followed by meningitis is not of exceptional occurrence.

Prognosis.—The prognosis of true epidemic cerebrospinal meningitis is always grave, but its rate of mortality varies in wide limits, namely, from 20 to 75 per cent. In children under two it is almost always fatal, and before puberty its mortality is very high. The most violent cases usually meet death by the fifth day, but it is not to be forgotten that many others reach the fourteenth day before death occurs. Then, again, it sometimes happens that after several days of severe symptoms the general aspect of the case improves, but the favorable signs only persist for a few hours and then the symptoms return with renewed vigor. Further than this, patients who seem about to recover not rarely suffer from a relapse which may prove fatal. Koplik asserts that the character of the spinal fluid is of great prognostic value. If it is thick and purulent the outlook is bad; but if it is of a straw color and clear it is good.

Treatment.—The treatment of true cerebrospinal fever is not very satisfactory. We know of no remedies which exercise any true curative influence, and all the physician can do is to keep the patient during the acute stages in a quiet, darkened room, and give bromides in sufficiently large doses to prevent convulsions of sufficient violence to exhaust the patient. Chloral is an even more powerful and useful drug for this purpose, being given in the dose for an adult of 20 grains by the mouth, or 60 grains by the rectum, in starch-water. Unless the nervous symptoms are very severe, it is a great mistake to attempt to overcome moderate rigidity or twitchings by full doses of nervous sedatives, because they in no way influence the progress of the disease, and simply give the stomach the task of absorption and the kidneys the burden of elimination. The idea that quinine is of value is probably erroneous. The doses usually advised are too small to act in any way as specifics, and if large doses are given they serve to distinctly increase congestion of the meninges and in the middle ear. Pain is to be relieved, if excessive, by the use of morphine in adequate doses, as much as ½ a grain being used if needed, particularly at night, to give rest and sleep. The morphine, or deodorized opium, had better be given by the mouth when this mode of administration is possible, as hypodermic injections may cause abscesses.

Cold, in the form of an ice-bag, may be applied to the head for the relief of headache. Blisters have been applied to the nape of the neck in the hope of influencing the effusion at the base of the brain, but they are of little value. It has been claimed by some practitioners that a very liberal use of antidiphtheritic serum exercises a curative influence.

Mention has already been made of lumbar puncture for diagnostic purposes. When headache, high temperature, rigors, or stupor are marked, the relief of the pressure upon the brain and spinal cord by this means may give temporary relief, but that it aids the patient permanently is very doubtful. The amount of fluid withdrawn should equal 40 to 50 c.c.

Very recently several clinicians have reported good results from the injection into the cerebrospinal fluid of an antiseptic substance. The method they employ is as follows: after withdrawing about 50 c.c. of cerebrospinal fluid by lumbar puncture they inject an equal quantity of normal salt solution (0.9 per cent.) through the same needle which has remained *in situ* after the withdrawal of the fluid. They next inject into the spinal cavity through the same needle about 10 c.c. of a 1 per cent. solution of lysol. After this is done the needle is withdrawn. It is claimed for this plan that it causes a fall of the fever, which, however, returns after one or two days. The same procedure should be repeated at intervals of two days until the cerebrospinal fluid when withdrawn is limpid and clear.

Relief from the severe pains in the limbs and back may be obtained in some cases by immersing the patient for long periods of time in a hot bath of plain or salt water at 99° or 100°.

The fever is rarely high enough to need treatment. If it is above 105°, the ice-bag and the use of cool spongings with frictions may be resorted to.

In all cases the diet should be one which is easily swallowed and easily digested, and everything should be done to support the system. This is particularly necessary in the prolonged types, in which marked emaciation is often present.

CROUPOUS PNEUMONIA.

Definition.—There is no condition of the lungs which is so apt to be confused in the mind of the student as that designated pneumonia. This is because the word "pneumonia" is used by some medical men to designate a single disease affecting the lung and by others as signifying any state in which, as the result of an inflammatory process, a part of the lung becomes congested or consolidated. The latter is the better use of the word, and when the physician desires to state that a lesion representing a definite infection is present he should specify the type of pneumonia by employing an adjective to qualify the noun—*i. e.*, he should speak of the various forms of pneumonia as croupous or lobar pneumonia, catarrhal or lobular pneumonia, and of tuberculous pneumonia. The term "pneumonia," while commonly used to signify croupous pneumonia, means nothing more definite than consolidation of the lung.

Croupous Pneumonia is sometimes called Lobar Pneumonia, Pneumonitis, Lung Fever, or Fibrinous Pneumonia.

Croupous pneumonia is an acute infectious disease depending for its existence, when in its typical form, upon the activity in the body of the specific organism known as the *Micrococcus lanceolatus*, sometimes called the pneumococcus of Fraenkel. As the result of this infection, there takes place in the lung an acute inflammation accompanied by the exudation into the air vesicles of an adhesive, croupous, or fibrinous exudate, which produces consolidation of the lobe or lobes affected. In addition to these changes the patient suffers from a greater or less degree of toxæmia, due to the poisons made by the infecting micro-organisms and from the changes produced in the tissues of other organs than the lungs by the growth of the micrococcus or by its toxins. This disease is also characterized by the fact that it usually lasts about nine days and ends by crisis, although this crisis may occur as early as the third day or even earlier in very rare instances.

Etiology.—The development of croupous pneumonia is dependent upon many causes, some of which we do not know. These causes are those external to the body which produce conditions in the individual favorable to the growth of the specific germ, and internal causes which exert similar influences. The importance of these conditions is shown by the fact that the pneumococcus is periodically or continuously present in the oral secretions of a large percentage of healthy human beings. Although the organism is capable of rapidly increasing in virulence, this cannot be held to account for all cases of infection, as even the most virulent strains are sometimes found in normal persons. A valuable contribution to the various phases of the pneumococcus question, including the communicability of the organism from person to person, is the work done under the auspices of the Department of Health of New York City, *Journal of Experimental Medicine*, August 25, 1905, vol. vii., No. 5, pp. 401-632.

So far as season is concerned, there can be no doubt that the summer and autumn are the months in which the fewest cases occur. Thus, the combined statistics of Seitz, in Munich, and Jürgensen for six large German towns, and of Sturgis for Westminster Hospital, London, show that in winter the incidence is 31.7 per cent.; in the spring, 34.6 per cent.; in the summer, 15.1 per cent., and in the autumn, 18.5 per cent. The following chart is based upon 35,828 cases occurring in hospitals in the United States, Germany, and Austria, and 19,000 cases occurring in the Confederate army during the year 1862, collected by Joseph Jones.

Exposure to cold was thought for many years to be a cause of croupous pneumonia, but we now know that this only acts as a predisposing cause which decreases the general systemic, or local, powers of resistance to infection; in other words, it is prone to affect all persons whose vital resistance is diminished. Living in poorly ventilated rooms is a predisposing cause, as is prolonged physical or mental strain, or any condition which saps vitality. A very interesting illustration of the effect of fatigue, bad air, and exposure in the production of croupous pneumonia has been recorded by Connell, of Leadville, Colorado, who reports the common occurrence of the disease in miners and others who go on long railway journeys for a day's outing and live during that time in badly ventilated railway cars.

In many cases of acute or chronic disease, death results not from the primary malady, but from the superimposed croupous pneumonia which attacks the feeble individual, who may be just about to touch the shores of convalescence. In these cases it is a true "terminal infection."

Croupous pneumonia is also a disease peculiarly apt to attack those of advanced years, and a very large proportion of deaths among the aged is due to this cause, such patients seeming to possess little resistance to its attack. This inability to resist the infection depends upon at least two causes—viz., a feeble heart muscle which cannot meet the circulatory demands of the disease nor resist the depressant effects of its toxins; diseased kidneys, or kidneys impaired in function, whereby toxic materials cannot be speedily eliminated, and as the general result of which the vital resistance of all the tissues is diminished, so that not only the *Micrococcus lanceolatus* is permitted full sway, but the patient is also placed in a favorable condition for the growth of other infecting micro-organisms which aid in producing a fatal issue. It is because of these facts that pneumonia so frequently attacks those who are already in ill health, or who are suffering primarily from some other malady, and it is for these reasons that it so often ends in death. Acute and chronic alcoholism greatly predispose to croupous pneumonia, and it is a singularly fatal disease in persons addicted to alcohol.

Fig. 32

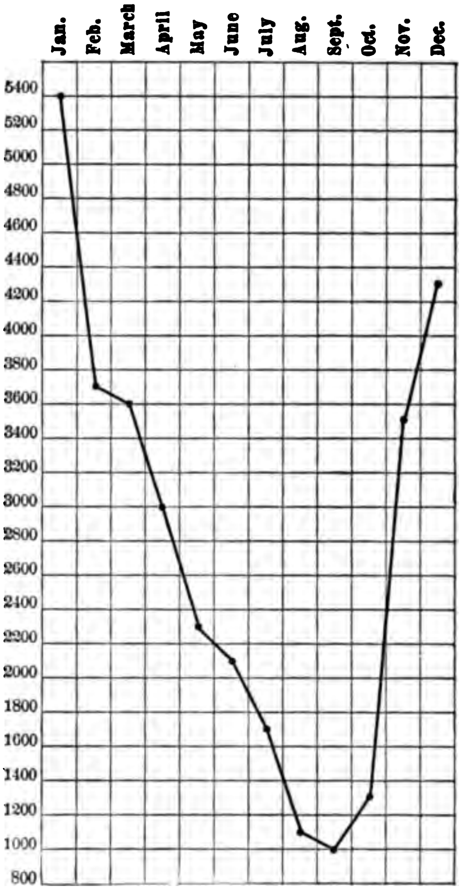


Chart showing the seasonal incidence of croupous pneumonia.

Sometimes an injury to the chest wall will be followed by acute croupous pneumonia, probably because the trauma to the lung renders it susceptible to infection. Numerous experimental observations have confirmed this clinical fact, which may be of great importance from a medicolegal standpoint, as well as from the purely clinical aspect. Without doubt local injury renders a part peculiarly susceptible to infection by any pathogenic micro-organisms which may enter it, and as the pneumococcus is a constant inhabitant of the mouth in healthy persons, a source of infection is ever present.

There can be no doubt that the disease is capable of being spread from one patient to another. On several occasions I have seen pneumonia contracted by the wife, or daughter, of a patient who was engaged in nursing him, and repeatedly it has occurred that the introduction of a case of pneumonia into a ward of a hospital has resulted in the development of the disease in other patients. Thus, out of eleven women suffering from typhoid fever on admission to my wards in the Jefferson Medical College Hospital, no less than eight suffered from croupous pneumonia after the introduction of a single case of this disease.

Unlike many of the acute infectious diseases, one attack does not protect against another, but rather predisposes the patient to subsequent attacks.

Distribution.—Croupous pneumonia is met with in all parts of the world, but it is more common in the temperate than in the tropical zones. In the

United States the census for 1900 shows that its greatest mortality occurs in the great Northwestern States east of the Rocky Mountains, in which district it causes 120, or more, deaths per 1000 deaths from known causes. Only a few areas in the States east of this area have so heavy a mortality, even if large cities like New York, Philadelphia, and Chicago are included.

Frequency.—Statistics as to its frequency are to some extent vitiated by the fact that in many health reports the difference between the various forms of pneumonia is not specified. There can be no doubt, however, that it is one of the most common and most fatal of all acute infectious diseases, and that its frequency and mortality are increasing. The United States census for 1900 shows that during that year the total mortality from pneumonia was 105,971, of whom 58,340 were males and 47,631 were females. The proportion of deaths was 106.1 for each 1000 deaths from all known causes. Its average mortality is about 1.5 to 2.3 per 1000 persons living.

At times croupous pneumonia may occur in epidemic form and cause an extraordinary increase in the death rate of a given district. Thus, the mortality from this disease in Chicago, as shown by Reynolds in his official report covering the period from January 1 to June 1, 1903, became remarkably high. There were 2891 deaths from pneumonia, as compared with 1321 from consumption and 1238 from all other communicable, contagious, or infectious diseases, including diphtheria, erysipelas, influenza, measles, puerperal fever, scarlet fever, smallpox, typhoid fever, and whooping-cough. This is an excess of 382 pneumonia deaths over the deaths from all the other preventable diseases—1570, or 118.8 per cent., more than the deaths from consumption, and 1653, or 133.5 per cent., more than those from the other specified diseases.

Rivière found 65 cases of croupous pneumonia in 260 cases reported as cases of "pneumonia" occurring in infants under two years. He believes that the disease is more frequent in infancy than in early childhood.

Croupous pneumonia occurs with the greatest frequency between the ages of forty and fifty years, but it is also very common between fifty and sixty. The mortality is in direct proportion to the age of the patient. It affects males far more frequently than females, the proportion being as high as 88 per cent. in the former to 12 per cent. in the latter (Kerr). This proportion in favor of men is probably too high for the average, but it serves to emphasize the fact stated, and is approximately correct. The reason probably lies in the greater exposure of men to cold and wet and to their abuse of alcohol.

The relative frequency with which croupous pneumonia affects the right and left lung, as based on many thousand cases collected by Meltzer in Russia; Jürgensen, Moellmann, and Brach in Germany, and West and Pye-Smith in England, is for the right lung, 51.4 per cent.; left, 39.4 per cent., and for both lungs, 9.2 per cent.

In 495 cases examined at autopsy, and collected by Fowler, Osler, Kerr, and Steven in this country and England, the disease was unilateral in 83 per cent. It is unilobar in the proportion of about 50 per cent. The disease affects a lower lobe in nearly 75 per cent. of the cases.

Prevention.—At the present time we have no means of directly preventing development of this disease. It is hardly necessary to state that the sputum of the patient should be received into a spit-cup containing some suitable disinfectant, or into a cloth which should be speedily burned. A patient suffering from croupous pneumonia should not sleep in the same bed with a person who is in health, and should be isolated as much as possible.

Pathology and Morbid Anatomy.—In studying croupous pneumonia it must not be forgotten that the disease is, at least in some cases, a general infection with the *Micrococcus lanceolatus*, the morbid changes being chiefly manifested in the lungs, just as in typhoid fever they are chiefly manifested in Peyer's patches. The pneumococcus is found in the blood during the progress of this disease with great frequency, now that proper methods for its discovery are employed. Thus, Prochaska has found it in the blood in 38 out of 40 consecutive cases, and Rosenow has isolated it in 77 out of 83 cases, and has discovered it in the blood as early as twelve hours after the initial chill. On the other hand, the mere presence of the pneumococcus in the blood of a patient does not necessarily mean that pneumonia is present, for it has been found in the blood in cases of tonsillitis, otitis, arthritis, and in pulmonary œdema. Parker and many others have even described cases of purulent peritonitis due to this organism.

While it is true, as already stated, that croupous pneumonia is, in its typical form, due to the *Micrococcus lanceolatus*, it is also a fact that lobar pneumonia or consolidation of the vesicular portions of a lobe or lobes may arise from infection by other micro-organisms. Such an occurrence is, however, rare, the non-specific infection resulting usually in abortive changes in the pulmonary parenchyma, or running a course at variance with that commonly pursued by the true infection.

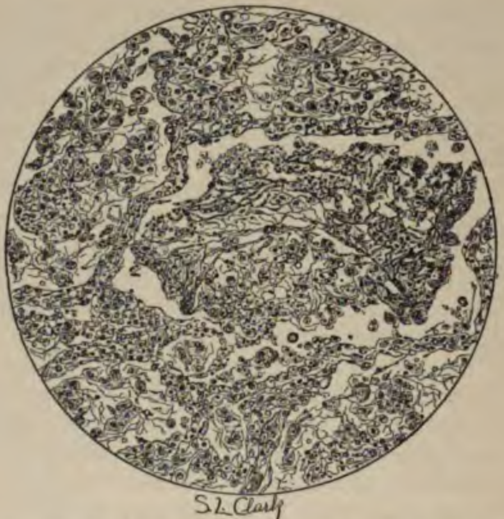
ENGORGEMENT STAGE.—The first change taking place in the lung in croupous pneumonia is a hyperæmia of the intervesicular tissues of the lobe or lobes about to be consolidated. This engorgement rapidly becomes more marked, and is accompanied by the exudation into the air vesicles of white cells (apparently transitional leukocytes) and red blood cells and serum, with fibrinous material, which speedily becomes solidified, so that all that part of the lung which is affected may, in the course of a few hours, be devoid of air and impervious to its passage, except in those bronchial tubes which are of some size.

STAGE OF RED HEPATIZATION.—The lung is now said to be in the stage of red hepatization (Fig. 33), since the exudate is red from blood-coloring matter, and the consistency of the organ to touch and on section resembles that of fresh liver; hence it is said to be hepatized or liver-like.

When a cross-section is made of the solidified lung the surface is seen to be granular because of the protrusion of the exudate from the air spaces. In some instances the cut surface is found not to be uniformly solid, probably because the process is less marked in some places than in others. This appearance of the lung on section is also largely modified in young children and in greatly enfeebled individuals, in whom the degree of solidification may be much less marked. If the exudate is examined microscopically, it will be found to contain not only shreds of fibrin, red and white cells, and desquamated epithelial cells from the walls of the vesicles, but large numbers of pneumococci as well. That the amount of extravasation is in many cases extremely large is shown by the fact that a lung may increase in weight by six or seven pounds.

STAGE OF GRAY HEPATIZATION.—Following the stage of red hepatization there ensues the stage of gray hepatization. At this time the acute inflammation in the lung has passed by and the system is beginning the task of clearing away the results of the disease, which is accomplished by the cells which have been extravasated undergoing fatty degeneration and granular change while the fibrin undergoes softening. During this stage of resolution the exudate is gotten rid of by absorption and expectoration. Finally, the air cells are freed from the exudate with which they were filled, the epithelial lining is reproduced, and recovery results.

FIG. 33



Lung; croupous pneumonia, stage of red hepatization. The centre of the microscopic field is occupied by an air vesicle containing a mass of exudate composed of a network of fibrin, red blood cells, and a few leukocytes.

UNUSUAL CHANGES.—In rare instances the normal process of resolution is not followed, and in its place organization of the materials which have been extravasated takes place to some degree, new connective tissue is proliferated into the air vesicles from their walls, and fibrous bands containing bloodvessels extend throughout the lungs. Simultaneously a similar growth takes place in the interstitial tissues, and so the lung gradually becomes consolidated by overgrowth of fibrous tissue.

Flexner and others have urged the view that unresolved lobar pneumonia is due to the fact that, owing to some disproportion between the leukocytes and other constituents of the exudate, or other causes as yet undiscovered, the normal process of autolysis is not carried out, and so the exudate undergoes organization instead of resolution.

In other instances which are far more rare the process of resolution is supplanted by the development of abscess or gangrene of the lung, which conditions are probably due to secondary infection of the lung by the streptococcus pyogenes, or staphylococcus pyogenes, or other bacteria capable of producing such lesions. Sometimes the process of fatty change and death of the extravasated cells is so rapid that on section of the lung the vesicles exude a purulent matter looking like true pus, which indeed it may be, but this in no sense is an abscess of the lung.

Associated with the changes in the lungs we find adjacent organs involved by direct extension of the inflammatory process or by the infection itself. The most common of these is inflammation of the bronchi (bronchitis), which is practically always present. After bronchitis in frequency comes inflammation of the pleura, due to direct extension from the underlying lung and to infection of the pleura by the specific organism of the disease. Nearly always this is manifested by the formation of a plastic fibrinous exudate on the serosa and an abnormal amount of fluid in some part of the pleural cavity, which fluid is often serous and not infrequently purulent. (See Pleuritis.)

Sometimes the pericardium is similarly affected, and even the endocardium may be infected by the specific germ. (See Pericarditis and Endocarditis, under Complications.)

Reference is made elsewhere to the meningitis which sometimes develops.

It is a great mistake to view the lesions just described as representing all the pathology of croupous pneumonia. It is true that these changes are the most evident, but it is not to be forgotten that the toxæmia of the malady exerts a very great influence in producing symptoms and lesions during life which are not so readily seen, but are equally important in their influence on the patient. The muscular fibres of the heart and the epithelial cells of the kidneys undergo albuminous degeneration, and similar changes occur in the liver. When the heart is opened after death we frequently find its cavities, particularly those of the right side, almost filled by firm clots, part of which may have formed so long before death as to be of the "chicken-fat" type. The liver is often found to be greatly engorged with blood, because of the impeded circulation in the vena cava, produced by the difficulty with which the right side of the heart empties itself. The bronchial lymph glands also show by the swelling of their cells and by their distended sinuses that they have endeavored to prevent the entrance of the micrococcus and its toxins into the general system, for in them may be found broken-down cells, red cells, pneumococci, and phagocytes containing cells or organisms.

My colleague, Coplin, has shown that definite changes take place in the intercostal muscles in the course of pneumonia and pleurisy. 1. Granular degeneration or cloudy swelling of the muscle fibres, which is probably a part of the general action of the toxic bodies circulating in the blood. 2. The muscle fibres are dissociated, œdema is present, but there is little fibrin-containing substance. Groups of muscle fibres and bundles show changes that cannot be differentiated from the hyaline degeneration described by Zenker as occurring in the muscles of the abdominal wall in typhoid fever. 3. In addition to the changes already described, leukocytes become abundant, bacteria are often present, and, finally, if the inflammatory process is chronic, there is an overgrowth of fibrous tissue and fatty infiltration of the muscle.

Incubation.—The incubation period of croupous pneumonia is forty-eight hours.

Symptoms.—Before describing the symptoms met with in cases of croupous pneumonia, it is essential to emphasize the fact that in no other infectious disease are the manifestations of illness so variable. These variations depend not only upon the virulence of the infecting germ and the susceptibility of the patient, but upon his habits, age, and general state of health. In some cases the malady develops as a frank, open inflammation of the lung. In others it is so insidious as to be overlooked, except by the most careful physician. In certain cases the course of the disease is markedly sthenic, in others profoundly adynamic. In still others the progress is so mild that the patient is never seriously ill, and in some instances it springs like a tiger upon a seemingly healthy man and destroys him.

The symptoms of croupous pneumonia may be divided into three stages for readiness of description—namely, those of onset, those of the well-developed stage, and those of convalescence.

STAGE OF ONSET.—The patient, usually an adult, is seized after, or without, a brief period of general malaise, with a *chill*, followed by a well-developed fever, The chills may be repeated and may vary from a slight feeling of creepiness to a severe rigor of sufficient force to shake the patient severely, and to last for over an hour. The *pulse* is quickened, but not as much so as we would expect from the sharpness of the onset, and at first may be small, but soon becomes full and bounding if the patient has been previously in good health; the *respirations* are also markedly increased in rate per minute. More or less severe *pain* may be felt in the chest on the affected side. The degree of pain, however, varies greatly, some patients bitterly complaining of it, while others seem to have little or no suffering, probably because in the latter cases the inflammation of the lung is so deeply situated that it does not extend to and involve the visceral layer of the pleura. It is important to bear in mind the fact that this pain not infrequently is referred by the patient to another part of the body. I had a case admitted not long since to my wards, on the statement of a well-known physician that she had appendicitis, when she was really suffering from a pleuropneumonia of the right lower lobe. Children are very prone to refer the pain to the epigastrium.

If the patient is very feeble it sometimes happens that the onset of the malady is insidious and no pain is felt. This is especially apt to be true when the disease complicates chronic alcoholism, renal disease, or other grave malady.

The *temperature* usually makes a sharp and decided rise, immediately after or during the chill, to 103° or 105°, and in some cases even higher than this, and remains high throughout the disease, the variation in the morning and evening temperature not being more than a degree or a fraction thereof. (See Fig. 34.)

The *face is usually flushed*, particularly over the cheek bones, and it is a noteworthy fact that this flush is usually most marked upon the cheek of the same side as the lung involved. The expression of the face is apt to be somewhat anxious, the skin dry and hot, and a moderate degree of cyanosis may be seen in the capillaries of the lips and finger-tips, and about the nose.

Violent *headache* may or may not be present. A more or less active *delirium* may also develop at this time, and the patient may be quite restless unless the pain in the side makes it more comfortable to lie quietly in bed. An incessant *unproductive cough* is often an early symptom of onset.

The *physical signs* of the disease in the thorax in the stage of onset are not, as a rule, well marked. Inspection may reveal some impairment of expansion upon the affected side; palpation may evince some increase in vocal fremitus; auscultation will show in many cases fine crepitant rales, increased bronchial breathing or tubular sounds, increased loudness of vesicular breathing for a few hours, and often some exaggeration of the normal respiratory sounds on the sound side. Indeed, this increase in the harshness of the breath sounds over the normal side, due to the increased activity on the part of the healthy lung, to compensate for the impairment of the diseased lung, may mislead the beginner in physical diagnosis into thinking that this is the lung diseased. Percussion may also reveal some impairment of resonance over the affected area.

DEVELOPED STAGE.—The developed stage of the disease is characterized by certain conditions and physical signs, some of which are almost pathognomonic of the malady. The peculiarity of the *pulse* is that it is quite slow as compared to the rapidity of the respirations. Usually when high fever is present the pulse rate is as high as 110 or 120, or even higher, while the respirations are about 24, but in pneumonia of the croupous type the pulse rate is sometimes only 90, while the *respirations* are as high as 30 per minute. More commonly, however, the respiratory rate mounts to as high a point as 40 or 50 per minute, while the pulse reaches 110 to 120, the relative proportion being 1 to 3, while in health it is usually about 1 to 4.5.

A second peculiarity of this stage is the *rusty* or *bloody sputum*, which is still more characteristic in that it is sticky and tenacious, and therefore difficult to expectorate, and so adherent that even when a spit-cup is filled with it the vessel can be held nearly upsidedown without losing its contents.

A third characteristic of croupous pneumonia at this stage is the development of single or multiple fever blisters, or spots of *herpes*, upon the lips or about the edges of the nostrils.

Dyspnœa may or may not be present. If present it depends upon the fact that so much of the lung is involved that respiration is difficult, or it is due to feebleness of the heart from engorgement of its right ventricle by the blood which cannot pass readily through the diseased lung; or, again, it may be dependent upon actual impairment of the power of the heart as a result of the action of the toxin of the disease upon its muscular tissues and nerve centres. Dyspnœa in croupous pneumonia may, therefore, be due to pulmonary, cardiac, or toxic causes. *Cyanosis* may be very marked, and not uncommonly the jugular and other superficial veins can be seen to be full and distended.

A peculiarity of the dyspnœa of pneumonia is the fact that the patient does not seem capable of resting quietly, but continually moves about, making exertions which seem scarcely compatible with so much shortness of breath.

Delirium of an active type is common in this stage, and it may be difficult to keep the patient in bed, particularly if he is an alcoholic.

During the second stage of croupous pneumonia the *pulse* may become hobbling or dicrotic, the heart sounds tumultuous, and the dyspnœa severe. In other instances the pulse seems voluminous, but nevertheless is very easily compressed to the point of extinction, while the sounds of the heart reveal the fact that that viscus is laboriously endeavoring to fill vessels which, because of their relaxation, fail to offer the normal resistance to its action. In still other instances, if the heart is markedly affected by the toxæmia of the disease, the heart sounds will be feeble and difficult to differentiate, and the pulse be very small and easily extinguished by pressure. In still other cases auscultation over the area of the pulmonary valves at the third left interspace will reveal accentuation of the pulmonary second sound or a murmur due to incompetency of these valves under pressure, while later on the labored action of the heart is shown not only in the signs named, but also in the pulsating jugular veins, which are distended and full, indicating great venous engorgement, as the result of the obstruction of the flow of blood out of the right ventricle, or because of inco-ordination of the auricular and ventricular contractions, as the result of the formation of a heart clot or from toxæmia.

The *physical signs* of croupous pneumonia in the well-developed stage are quite characteristic in typical cases. *Inspection* shows an even greater impairment of expansion on inspiration on the affected side than in the stage of onset, and palpation reveals, when the patient speaks, a distinct increase in vocal fremitus over the part of the lung which is diseased. *Auscultation* gives a harsh inspiratory sound, prolongation of expiration, and a large number of fine crackling or crepitant rales in the same area, so fine that they may not be heard by the careless examiner. They sound very much as does that noise which is produced by moistening the tip of the forefinger and thumb with saliva, pressing them together, and separating them, or, again, as does the sound made by the hair which grows over the examiner's ear when it is rubbed between the finger and thumb. Ordinary vesicular breathing over the area diseased is absent, and in its place is heard bronchial breathing, which is caused by the air in the bronchial tubes, which produces a sound which is transmitted through the consolidated lung unmuffled by the vesicular murmur usually present. Auscultation while the patient speaks will also show a distinct increase in vocal resonance. That is to say, the sound of the voice will be transmitted through the chest-wall with a greater degree of clearness than in health. While auscultation is being performed in cases which have a delicately developed chest, as in youths and children, it is often noted that the movement of the anterior chest-wall under the ear is not uniform, but undulating, one part expanding at an appreciable interval before the other.

Percussion, a most valuable aid in the diagnosis of this disease, reveals, if the lesion in the lung is near the surface, marked impairment of resonance amounting to dulness, but it is a fact well worth remembering that if the lesion in the lung is deep seated, and not near its surface, the percussion note over the area diseased may not be impaired or dull, but hyperresonant, or, as

Samuel West has said, "boxy" in character. Usually hyperresonance is demonstrable all over the lung, except where it is consolidated, and is also to be found upon the healthy side of the chest, owing to the increased amount of air which is in these parts to compensate for the area of consolidation; but careful examination will reveal the fact that the hyperresonance over the consolidated area, or in its immediate neighborhood, has a different tone from that in the healthy and compensating lung, the "boxy" note just named. I have frequently been able to determine the presence of deep-stated pneumonia by the presence of this sign. By the aid of careful auscultation and percussion it is usually, but not always, possible to definitely determine the exact area of the lung which is involved.

While in the majority of cases these positive signs of croupous pneumonia may be found in a more or less well-developed form, it is not to be forgotten that negative signs may be as valuable in making a diagnosis. That is to say, there may be absence of any one or all of the signs just enumerated, and a total absence of vesicular breathing. In such cases, therefore, the physician must exercise care lest the loud and exaggerated breath sounds of the healthy part of the chest mislead him into thinking that that portion is the one which is diseased.

In certain instances, in which the action of the heart is very labored, its sounds distant, and the pulse is small and insufficient, careful examination may reveal a pericarditis with effusion, which, by its pressure, interferes with the movement of the cardiac muscle. This question as to whether there is pressure by *pericardial effusion* is by no means readily determined, because it frequently happens that there is a marked degree of cardiac dilatation present at this time, which naturally increases the area of cardiac dulness downward and to the right. Further, as it is the right ventricle which is most apt to be engorged, the area of cardiac dulness may be abnormally great in this direction. Again, it not infrequently occurs that the compensatory fulness of the healthy lung, if the disease is on the left side, pushes the heart downward and to the left, or, on the other hand, if the right lung is diseased, the unusual expansion of the left lung causes an extension of pulmonary resonance to the right, and so increased area of cardiac dulness is very effectually masked.

Patients suffering from croupous pneumonia should always be turned on the side when the back is to be examined, as it is dangerous, because of the state of the heart, for them to sit up in bed.

The *urinary flow* during an attack of croupous pneumonia is usually diminished, so that the passage of about twenty ounces of urine in twenty-four hours may be taken as the average. This urine is usually highly concentrated, and contains, as does the urine in most febrile diseases, an increased amount of urea and an excess of amorphous urates which are deposited on standing. It also contains, very constantly, a moderate amount of albumin, but the chief peculiarity is its *scanty content of chlorides*, which may be entirely absent. If the albumin be large in amount, or casts are present, the probability is that the kidneys were diseased before the onset of the pneumonia.

During the course of croupous pneumonia the function of the *alimentary* *canal* is rarely seriously disturbed, although loss of appetite because of the fever may be a marked symptom. The most important change in any part of the digestive system, if it may be so called, is seen in the tongue, the state of which is noteworthy, because it gives some idea of the general state of the patient. It is, of course, prone to be dry and somewhat coated, caused by the rapid breathing through the mouth, and because of the fever; but if it be exceedingly dry and red, narrow and pointed at the tip, it possesses a more positive significance as to the general state of the patient than if it be broad and moist.

Sometimes when pneumonia is very severe and particularly when toxæmia is marked, an excessive degree of *tympanites* develops, which is of evil significance, in that it shows a diminution in vitality and causes interference with the action of the lungs and heart by pressure. I have seen this most commonly when the disease has affected those addicted to the excessive use of alcohol.

The *nervous symptoms* of pneumonia are quite various and depend more upon the previous habits of the patient, the location of the lesion, and the degree of toxæmia than upon any other causes. Delirium varies in degree from mind wandering, as the patient is about dropping off to sleep, to active mania, during which it may be very difficult to keep the patient in bed. The severity of the delirium depends largely upon the age of the patient and his habits. Alcoholic patients nearly always have delirium in a well-marked degree, and in this class of patients it is grave from a prognostic point of view in direct proportion to its constancy and severity.

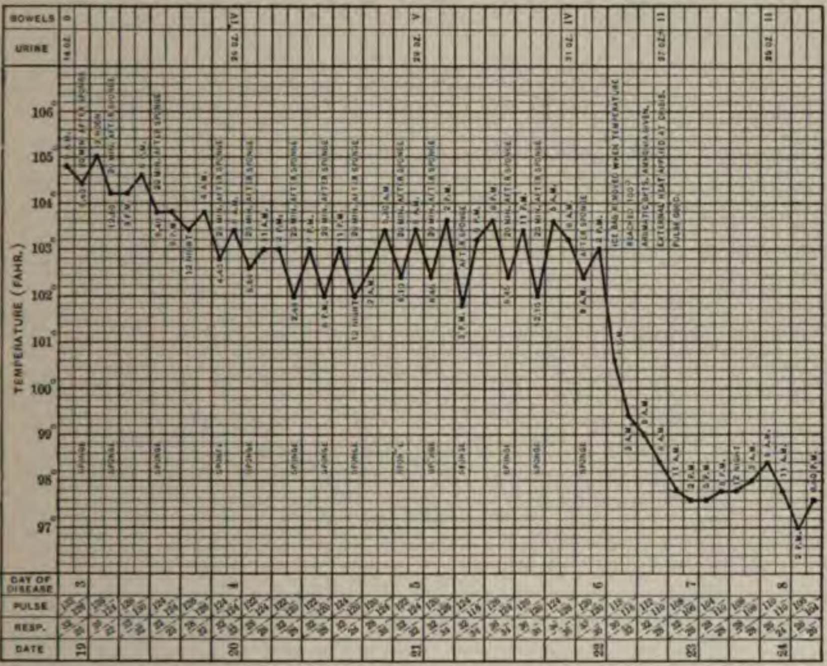
The type of the delirium also varies very greatly in the strong and in the weak. In those who are adynamic from some previous disease or from bad habits, it is often of a low muttering type, resembling that sometimes seen in toxic cases of typhoid fever, while in other instances it may be violent, as already described.

It is a noteworthy fact that delirium is particularly prone to affect those who suffer from pneumonia at the apex of the lung, and I have frequently seen in children, who had pneumonia at the apex, a delirium in which there seemed to be a constant fear of falling, so that the child clutched its mother every time it was moved. Holt's experience, on the other hand, leads him to believe that the portion of lung involved has little influence upon the production of nervous symptoms, and without doubt the recent advances in the study of pneumococcus infection tend to show that the toxæmia and not the portion of lung involved is responsible not only for the marked nervous manifestations, but also for the dyspnœa and great acceleration of the respiration. A peculiarity of the delirium in many alcoholic cases is that they labor under the delusion that they are lying in a coffin, and in their constant efforts to escape greatly exhaust themselves. This form of delirium is exceedingly grave from a prognostic standpoint. Delirium is also very much more apt to be marked in those patients who suffer from toxæmia than in those in whom a very considerable area of the lung is involved, but who have, nevertheless, comparatively slight signs of poisoning by the toxin of the pneumococcus.

Very rarely, in young children, the onset of the disease is characterized by convulsions instead of by the ordinary chill.

Insomnia is a very constant symptom of croupous pneumonia, and may become so persistent as to require medicinal measures for its relief, particularly if it be accompanied by great restlessness.

Fig. 34



A chart of croupous pneumonia in a girl of six years, showing the little effect produced by sponging upon the temperature and the characteristic crisis on the sixth day.

The *skin* in an ordinary case of croupous pneumonia is usually hot and dry; but if the toxic element in the case is very marked, it may, as death approaches, become cold and clammy and even bedewed with sweat. In toxic cases, too, it is not infrequently somewhat *jaundiced*. If this jaundice is associated with *hæmoglobinuria* the prognosis is almost certainly fatal. On the other hand, in some instances jaundice occurs apparently as the result of the action of the toxin upon the liver, and this type is not so grave.

Profuse sweating nearly always occurs at the time of crisis. The frequency with which herpes appears about the mucous membranes and skin of the mouth and nose has already been mentioned.

Stage of Resolution.—As the disease approaches the period of crisis, and sometimes not until this event has taken place, it will be noted that the rapidity of respiration as compared to the rapidity of the pulse more nearly approaches the normal ratio.

The first change which can be noted in the physical signs in the chest is the development of fine moist rales, which indicate the early stages of resolution. These rales, when they are first heard, are fine and crepitant, and closely resemble those heard in the stage of onset; for this reason they are called *râles redux*.

The rales in the chest become more and more coarse and moist in character as convalescence is carried on, and the speed with which nature in an otherwise healthy individual clears away the exudate is quite extraordinary, although usually for several weeks after a sharp attack of croupous pneumonia, involving the surface of the lung, impaired resonance on percussion and some prolongation of expiration with harsh inspiration can be demonstrated.

The *critical fall of temperature* is often preceded by a sharp rise, but when the fall occurs it takes place with extraordinary speed, the patient being afebrile or with a subnormal temperature within a few hours, or even within one hour (Fig. 34). Sometimes this critical state is accompanied by a profuse sweat, and even collapse may develop, with urgent dyspnœa, due to vasomotor palsy and vascular relaxation.

When the fall is quite gradual, extending over a day, it is called a *protracted crisis;* this very commonly occurs in children.

Often the day after crisis the temperature returns to slightly above normal, and sometimes an apparent crisis fails to reach the normal and the fever rises again. Such a *pseudocrisis* is rarely seen after the fifth day.

The critical fall of temperature, as has already been stated, usually occurs on about the seventh or eighth day of the disease, but it may occur as early as the third day (Fig. 35). In feeble persons and in children the disease sometimes ends by lysis.

Complications.—The complications of croupous pneumonia are quite numerous. Of these the most frequent is undoubtedly *pleurisy*. Indeed, it may be said that in almost every case of croupous pneumonia a certain amount of inflammation of the pleura exists. As an illustration of this fact, the statistics of Kerr are of value. Out of 171 cases which came to autopsy from croupous pneumonia, no less than 118 showed acute pleuritis. Of these, 74 were acute fibrinous pleuritis, 38 serofibrinous pleuritis, and 6 acute empyema. In Osler's 103 autopsies pleuritis was present in all but 2 cases. The pleuritis is due to the extension of the inflammatory process to the visceral layer of the pleura and to infection of the pleural membrane by the pneumococcus or by some other organism which is associated with it. (See Pleurisy). The inflammation of the pleura manifests itself by an excess of pain in the area involved, by a friction sound on auscultation, and later, it may be, by the outpouring of a considerable amount of fluid which may be serous or purulent. When the fluid is serous it is often absorbed with a rapidity only equalled by the absorption of the croupous exudate in the lungs. In other instances it persists and actually increases in quantity, relief only being obtained when the physician performs paracentesis. In 4523 cases of croupous pneumonia, occurring in twelve large hospitals in the United States and England, pleural effusion is stated to have occurred in 233 cases, a percentage of 5.15.

FIG. 35

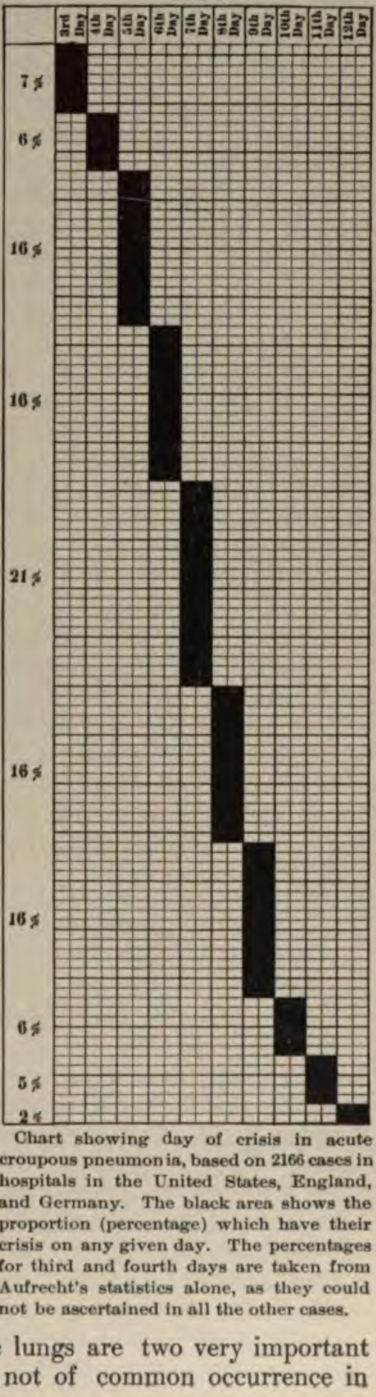


Chart showing day of crisis in acute croupous pneumonia, based on 2166 cases in hospitals in the United States, England, and Germany. The black area shows the proportion (percentage) which have their crisis on any given day. The percentages for third and fourth days are taken from Aufrecht's statistics alone, as they could not be ascertained in all the other cases.

In still other cases the effusion is purulent from the beginning, and in this way an *empyema* is formed. Like all collections of pus, recovery can only be reached in the majority of these cases by giving vent to the accumulation. The presence of the pus is usually manifested by a return, or maintenance, of the febrile movement seen in the early stages of the disease, accompanied, it may be, by the ordinary manifestations of septic poisoning, such as chills, sweats, and irregular temperature. On the other hand, all evidences of the presence of pus may be absent, owing to the non-absorption of toxic matters through the pleural membrane. In 10,076 cases of croupous pneumonia collected principally from the official reports of hospitals in the United States, England, and Germany, empyema is stated to have occurred in 208 cases, a percentage of 2.06.

In all cases in which speedy recovery from croupous pneumonia does not take place and where marked impairment of resonance persists upon the diseased side, pleural effusion or empyema should be strongly suspected, and the tests for the purpose of determining these complications be instituted. Sometimes the presence of a pleural effusion is not suspected because it produces no symptoms until, by the increase in its quantity or the taking of moderate exercise by the patient, it produces dyspnœa by interfering with respiratory movements. (See articles on Pleural Effusion and Empyema.) It is a noteworthy fact that if the empyema be due to the pneumococcus the prognosis is more favorable, both as to complete recovery and to speediness of cure, than if it be due to some other infecting micro-organism.

Hydropneumothorax has occasionally been recorded as a complication, but it is very rare.

Gangrene and abscess formation in the lungs are two very important and serious lesions which, fortunately, are not of common occurrence in connection with cases of croupous pneumonia. Eisendrath has analyzed 96 recorded cases of pulmonary abscess, gangrene, and bronchiectasis following croupous pneumonia. When the totals are computed as to percentage of recovery, the result is quite striking, especially in the more acute cases. Of 25 cases of acute single abscess, 96 per cent. recovered and 4 per cent. improved; of 28 cases of acute gangrenous abscess, 71.4 per cent. recovered, 7.2 per cent. improved, and 21.4 per cent. died. Of 14 cases of chronic simple abscess, 42.8 per cent. recovered, 21.4 per cent. improved, and 35.8 per cent. died; while in 26 cases of chronic putrid abscess with bronchiectasis 50 per cent. recovered, 15.3 per cent. improved, and 34.7 per cent. died.

Eisendrath found, from his review of the subject, that the symptoms usually came on after the crisis and consisted in a post-critical rise in temperature, which then became remittent in type. The sputum became purulent, and there was a distressing cough, accompanied by expectoration of pus in large quantities. If the abscess cavities do not communicate with a bronchus there is but little expectoration. There is in all cases emaciation, loss of appetite, and a rapid decline in strength. If the abscess becomes chronic there may be recurrent attacks of fever, with profuse expectoration.

Physical examination in these cases is rather disappointing. The lesions are most frequently in the lower lobes, and this is of some aid in diagnosis. There are no typical physical signs, owing to the fact that the cavities, be they due to abscess, gangrene, or bronchiectasis, may be near the surface, or quite deeply situated, and may or may not communicate with a bronchus. Dulness, decreased respiratory murmur, decreased vocal resonance, and decreased fremitus are present in the majority of cases, but bronchial breathing may be heard. The most reliable sign is the presence of large, moist rales, not infrequently metallic in character. Another striking feature is the variability of the physical signs, so that dulness and then tympany may alternate at the same spot. Clubbed fingers develop quite early, as do also symptoms produced by pressure on the heart, liver, and spleen.

Gangrene must be suspected when there occurs a rise of temperature, a few days after the crisis, and the breath becomes fetid. The sputum is also fetid and divides itself into three characteristic layers. (See Gangrene of the Lung.)

The frequency of hæmoptysis in cases of gangrene is due to the fact that the vessels are more apt to pass freely through the cavity, owing to the more rapid destruction of tissue.

In *bronchiectasis* following pneumonia the sputum may be fetid at times, but the odor is not so penetrating as in gangrene and there are no elastic fibres. There is usually a history of long-continued expectoration of large quantities of pus. This, however, is not characteristic, for the same history may be true of chronic simple abscess.

Pericarditis is not a very infrequent complication of pneumonia. In the majority of instances it is of such mild degree that it does not jeopardize the patient's life; but in other instances, when the effusion which follows it is profuse, it may, by mechanical pressure, produce great cardiac disability. When the accumulation is extensive, a definite increase in the area of cardiac dulness is usually demonstrable. Not rarely, however, the presence of this complication may be unsuspected during the patient's life. Thus, Thayer was only made acquainted with the presence of pericarditis in one of his cases of croupous pneumonia when the autopsy disclosed a thick layer of pyogenic membrane over the visceral pericardium, with a large quantity of pus in the pericardial cavity. Some statistics seem to show that pericarditis varies in frequency in from 5 to 16 per cent. of all cases, but in 21,383 cases of croupous pneumonia collected by me principally from the official reports of hospitals in the United States, England, Germany, and Austria, pericarditis is stated to have occurred in only 266 cases of croupous pneumonia, a percentage of 1.24. (See Pericarditis.)

Endocarditis is a rare complication, occurring much less frequently than pericarditis. In a considerable number of cases the pneumococcus is responsible for the lesion. It often affects the aortic valves, and it is generally of the ulcerative type. In 14,510 cases of croupous pneumonia collected from several series of cases reported by German, English, and Swedish physicians, and from official reports of hospitals in the United States, England, Germany, and Austria, endocarditis is stated to have occurred in 106 cases, a percentage of 0.73. Norris in 500 cases found it recorded five times, while Sears and Larrabee, in Boston, found it 9 times in 940 cases. Aufrecht, in 1500 cases, met with endocarditis only once. Out of a total of 5738 cases of croupous pneumonia von Brach found less than 0.2 per cent. complicated by endocarditis, and less than 0.5 per cent. of them complicated by pericarditis. Preble, from an exhaustive study, places the average at 1 per cent. in all cases and 5 per cent. in fatal cases, and these figures are probably correct. Osler found 16 instances of endocarditis in 100 fatal cases. Preble believes that while pneumonia is more common in males than in females, endocarditis due to this infection is most common in females. (See Endocarditis.)

Two apparently distinct types of *meningitis* are found as complications of croupous pneumonia; one appearing at the onset of the disease, the other during the active or post-critical stage. The former variety is seen most frequently in children, and is probably symptomatic; it is rarely fatal, and therefore its pathology is somewhat uncertain. On the contrary, meningitis developing during the course of the well-developed infection is generally the result of meningeal infection and is very frequently associated with endocarditis. (See Cerebrospinal Meningitis.)

Numerous cases are on record of croupous pneumonia in children which at the onset simulated meningitis, cerebrospinal meningitis, and even hemiplegia. But the subsequent appearance of local physical signs, the pulse and respiration ratio, and the crisis, marked by a sudden fall in temperature about the eighth day, have confirmed the diagnosis of croupous pneumonia. The favorable termination in many of the reported cases has not permitted an adequate pathological investigation, although meningitis due to the pneumococcus is well recognized.

Disturbance of the nervous system over and above the signs of meningeal irritation or true meningeal inflammation may occur. *Hemiplegia* in croupous pneumonia was recorded by Huxham; later it was described by Charcot, Lépine, and Vulpian as *hemiplegie pneumonique*. It may occur early in the course of the disease, or may not develop until the period of convalescence. Such a paralysis has been observed in cases as early in life as the eighteenth month and as late as the seventy-sixth year.

Pierre Boulloche has collected 56 cases of paralysis resulting from croupous pneumonia. In this analysis the type of paralysis was found to be nearly always hemiplegic. In advanced years death nearly always ensued upon this complication, while in the young the mortality was very much lower, recovery being the rule. In one case occurring at the age of fifty-eight years, hemiplegia, with aphasia, developed during the course of the disease, but ended in recovery.

In some instances the *paralysis is monoplegic*, and this is well illustrated by a case described by Boulloche in a patient thirty-two years of age, who, from the onset of the disease, was delirious and who presented a typical right-sided croupous pneumonia. Paralysis of the right arm and right side of the face was discovered upon the sixth day of the disease. Movements of the right leg were entirely retained. There was aphasia, but no loss of consciousness, neither was there any disturbance of sensibility; twelve days later the fever had subsided, the aphasia had diminished considerably, and the muscles of the face were less drawn. Sensation in the pharynx returned and a day later the aphasia disappeared. The facial paralysis passed off; the relative strength of the two arms showed only a decrease of 10 degrees in the affected side, and at the expiration of twenty days the monoplegia had entirely disappeared.

Transitory aphasia is a complication reported by Chantemesse. This observer has found that aphasia usually occurs about the second or third day of the disease, that it is ordinarily preceded by headache and giddiness, even to the verge of syncope; in some cases numbness or a sensation of pricking in the right side of the face and right arm is experienced; in other cases it may set in abruptly without loss of consciousness or become manifest after a typical apoplectiform seizure. The characteristics of the speech impairment do not differ from those dependent upon an organic lesion of the third frontal convolution upon the left side of the brain. The paralysis may involve the entire right side of the body, but usually only the inferior portion of the right side of the face, the right half of the tongue, and the right superior extremity are affected; as a rule, sensation and the reflexes are not altered. In pronounced cases the paralyzed parts may be the seat of increased redness and an œdema, which is more or less circumscribed and increased by heat. The phenomena persist commonly for from a few hours to a few days, and seem in no way to influence the primary disease.

It is doubtful whether the clinical picture and pathology of these cases of transitory aphasia differ in any particular from many of the cases already described as hemiplegic. They probably represent the cases in which no lesion is found postmortem.

Softening of the brain has occurred in some cases. In one case, reported by Suckling, it was due to thrombosis of the basilar artery, and thrombosis of the circle of Willis; with plugging of the superficial arteries of the left hemisphere. While these lesions have been found as the causative agents in producing hemiplegia, there are also cases on record in which the autopsy has been negative. In other words, hemiplegia with lesions and hemiplegia without lesions occurs. In the former case hemiplegia results from either meningitis or softening, or is due to thrombosis or embolism. In the second class the paralysis is like that of diphtheria—that is, of the toxic type. It is important to remember that it is possible for hemiplegia to develop in pneumonia without there being any relationship between the two conditions.

The fact that these marked nervous manifestations sometimes come on early in an attack of croupous pneumonia emphasizes the importance of examining the chest in all cases of paralysis, not only because pneumonia is competent to produce hemiplegia or other localized palsy, but also because these conditions are quite competent to produce secondary pulmonary lesions. In other words, pulmonary lesions may be the cause of hemiplegia, and hemiplegia may be the indirect cause of croupous pneumonia.

Neuritis, occurring chiefly as a sequel to croupous pneumonia, has been described by several observers. These cases resemble those described by Boulloche as paralysis with muscular atrophy, coming on during the period of convalescence.

Until Weichselbaum isolated the pneumococcus from the pus aspirated from the synovial sac of joints involved during the course of croupous pneumonia, the occurrence of *arthritis* was considered a coincidence, but since 1888 arthritis and osteoarthritis have been recognized as being not rarely due to a pneumococcus infection. Herrick has collected 52 cases from the literature of the subject, including some of his own, but it is interesting to note that in 2292 cases of pneumonia collected by me, treated by various Swiss and German physicians, only 2 cases of arthritis occurred.

In regard to the frequency with which different joints are involved in this complication, the following quotation from Herrick's paper is of interest: "In 23 of 52 cases the upper extremities alone were involved; in 18 cases the joints of the lower extremities alone; in 11 there was involvement of joints of both the upper and lower extremities. These figures show a slight preponderance in favor of limitation to the upper extremity, but so slight that little or no significance can be attached to it. In fact, the knee seems to be the joint oftenest affected, being involved in 22 of the 52 cases, in 3 of which both knees were affected, so that out of a total of 84 joints the knee makes up 25, or about 30 per cent. The involvement of other joints was as follows: the sternoclavicular, eight times; the shoulder, twelve times; the elbow, nine times; the wrist, eight times; the metacarpophalangeal, twice; the hip, three times; the knee, twenty-five times; the ankle, three times; the metatarsophalangeal, three times. The arthritis was monarticular in thirty-two instances, or in 61.5 per cent. of the cases. The joints thus solitarily involved were: shoulder, ten times; knee, nine times; wrist, five times; elbow, twice; sternoclavicular, four times; and the hip, ankle, metacarpophalangeal, and metatarsophalangeal, each once. Of the remaining cases there were involved: two joints, nine times; three joints, four times; four joints, once; more than four, three times." These figures bring out the fact that the larger joints are more often affected than the smaller ones.

The process in subacute cases is sometimes highly destructive to the joint.

It is a noteworthy fact that the prognosis as to life is grave, the mortality amounting to 65 per cent., chiefly because this lesion is associated, as a rule, with affections of the serous membranes elsewhere, and particularly in the endocardium.

Venous thrombosis is an exceedingly rare complication of pneumonia. Steiner could find only 38 cases recorded, and reports 3 of his own. In 27 of these the thrombosis occurred during convalescence. In 1 case it occurred at the time of crisis and in 4 during the course of the disease; and in the cases collected by him the lower extremities were always involved. The left lower extremity was involved in 16 cases; the right in 10, and both legs in 7. The more frequent involvement of the left extremity is attributable in this disease, as in typhoid fever, to the greater length and obliquity of the left common iliac vein and its passage beneath the right common iliac artery. Adding Steiner's 3 cases to the 38 which he found in the literature, making 41, we find that recovery occurred in 25, death in 9, and that no definite information is given of 7.

Gangrene of a limb due to arterial thrombosis or embolism has been recorded by Zuppin, Benedict, Grimm, and Nielsen.

Parotitis, while a rare complication of croupous pneumonia, may occur, and not infrequently goes on to suppuration. Most of the cases so far reported have not been due to the pneumococcus, but to the staphylococcus or streptococcus.

Otitis media is quite a common complication of croupous pneumonia in children, the infection taking place through the Eustachian tube.

A *relapse* in croupous pneumonia is practically never met with, but recurrence is very common.

Duration of Croupous Pneumonia.—It is important to remember that while croupous pneumonia often runs a course of from seven to ten days, it not infrequently reaches its crisis at a much earlier period. As already pointed out, crisis may occasionally occur as early as the third day, and by no means infrequently takes place as early as the fifth. While it is true that early crisis usually occurs in comparatively mild attacks of the disease, it is also a fact that the patient may seem seriously ill throughout the whole course of these cases of comparatively short illness.

Varieties of Croupous Pneumonia.—Croupous pneumonia varies much in its character with the condition of the patient that is attacked. I have already mentioned the type which occurs in persons who are addicted to the excessive use of alcohol. In other individuals the disease is accompanied by such marked symptoms of adynamia that the patient seems to be suffering from typhoid fever, so far as his general symptoms are concerned. This form is known as typhoid pneumonia, in that it is typhoid in character; but this term does not necessarily imply that typhoid infection is associated with that by the pneumococcus. On the other hand, it sometimes happens that patients suffering from typhoid fever also have a pneumococcic infection of the lung, and this, of course, is another form of so-called typhoid pneumonia. True croupous pneumonia also occasionally, although rarely, complicates malarial fever, acute articular rheumatism, and pulmonary tuberculosis. Sometimes, too, it occurs as a sequel to the administration of ether as an anæsthetic.

This is probably due primarily to the chilling and irritation of the lung by the drug, and secondarily to the inhalation of pneumococci from the mouth, where, as already stated, they are almost constantly present even in healthy persons.

Diagnosis.—Croupous pneumonia is to be carefully differentiated from acute tuberculous pulmonary infection, from lobular or catarrhal pneumonia, from infarction of the lung, accompanied by bloody expectoration, due to cardiac disease, from pleurisy with effusion, and from chronic inflammation of the pleura, with marked thickening of that serous membrane. Finally, it is to be separated from hypostatic congestion due to cardiac feebleness arising in the course of acute diseases or chronic ailments.

The differentiation from acute pneumonic phthisis may be quite impossible until the development of profuse sweating, a feeble and rapidly acting heart, and the appearance of yellow elastic tissue and tubercle bacilli in the sputum takes place. From pulmonary infarction it is to be separated by careful examination of the heart, which may reveal valvular lesions, and by the fact that in infarction the onset of pulmonary disorder is instantaneous and the sputum contains bright blood. From pleural effusions it is differentiated by the development of the physical signs of that condition. (See Pleurisy, with Effusion.) Hypostatic congestion of the lungs is discovered by the character of the sputum, which may be blood-stained, although it is usually serous, by the fact that the lesions are usually bilateral, and also by the fact that the heart is primarily very weak. Catarrhal or lobular pneumonia is recognized by the absence of the typical rusty sputum, by the history of the presence of some primary disease prior to the onset of the pneumonic consolidation, and by the wide distribution of the lesions and the more diffuse physical signs.

An important aid to the diagnosis of croupous pneumonia is the increase in the number of the polymorphonuclear white cells, the so-called leukocytosis of croupous pneumonia. In this disease in most instances the increase in these particular white cells causes a leukocytosis of from 18,000 to 20,000.

The blood serum of these cases is capable of causing agglutination of the pneumococcus and the degree of agglutinative power seems to be greatest about the time of crisis, but there are technical difficulties about the test which render it of little value in diagnosis.

It is of the greatest importance that the severe pain sometimes described as being in the belly at the onset of pneumonia is not mistaken for that due to appendicitis. Cases frequently occur in which pain due to thoracic disease is thought to be abdominal, particularly if the base of the lung is involved. The presence of pain on pressure over McBurney's point, of some fixation of the abdominal muscles, and of a high leukocyte count may be so misleading as to lead the physician to operate for disease of the appendix.

It is characteristic of croupous pneumonia that the chlorides in the urine are greatly decreased.

The physician should always be on his guard lest he overlook a "central" or deep-seated pneumonia, which presents no marked physical signs.

Prognosis.—The prognosis in croupous pneumonia is always to be governed by the recollection of the fact that its mortality in adults is usually high, and again by the condition and habits of the patient. It is to be remembered that the prognosis in a case of croupous pneumonia is grave in direct proportion to the years of the patient. In young children, unless it is complicated by some grave accident, the disease has a very low mortality. By

Fig. 36

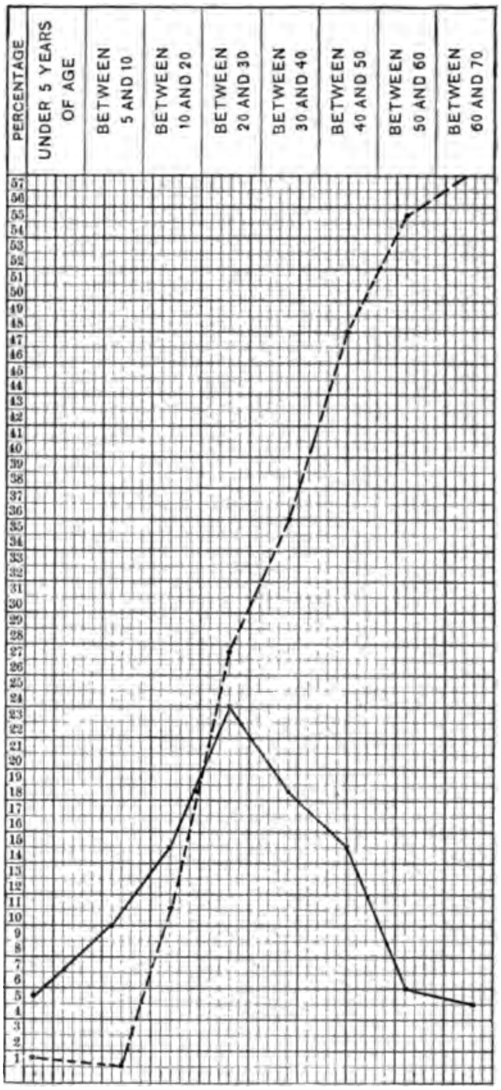


Chart showing the morbidity and mortality of croupous pneumonia at different ages, based on 86 cases in the Presbyterian Hospital, New York, and Guy's Hospital, London. Solid line, morbidity; dotted line, mortality.

far the greater number of children recover, whereas in advanced years the disease is exceedingly fatal (Fig. 36). As an illustration of how low the mortality may be when young, healthy persons are affected by the disease and come under skilful treatment early in its course; Osler states that in 40,000 cases occurring in the German army the mortality was only 3.6 per cent.

If the mortality percentage is based upon the total number of deaths from this disease, it may be stated to be as high as from 25 to 40 per cent.; but if, on the other hand, those cases which would naturally fall victims to its ravages are excluded, the mortality is probably only about 10 per cent., if we accept the large statistics of Townsend and Coolidge, who excluded patients over fifty years of age and those who were delicate or suffering from some other disease primarily present. In private practice the mortality varies from 6 to 18 per cent.

Aside from advanced years the other causes which render the prognosis especially grave are renal disease, with secondary cardiovascular lesions, alcoholism, and diabetes. Indeed, these three states contribute a very large proportion of the number of cases which suffer from this malady, and also the largest proportion of deaths in the statistics.

It is stated by some authors that any history of previous ill health distinctly increases the danger from croupous pneumonia. While this may be true in certain cases in which vitality is greatly depressed, it is also a fact that pneumonia in chronic invalids frequently runs a comparatively mild course unless the cause of their ill health be renal or cardiac disease, whereas it may speedily produce death in robust, powerful, muscular men, who frequently succumb to its ravages far more rapidly than more lightly built and apparently delicate individuals. Indeed, the physician of experience dreads the onset of this disease in powerful, well-developed men much more than when it attacks those who are less given to active exercise and feats of physical strength. Stout persons also seem much more susceptible to the lethal influences of the disease than those who are lean. This probably depends upon two causes: first, the heart and lungs may be overweighted by fat, and, second, such persons usually contain in their tissues a large amount of serum, in which, perhaps, specific micro-organisms find an opportunity to grow and to prepare their toxic product in large quantity.

Cases of croupous pneumonia characterized by moderately high fever do not possess the unfavorable outlook of other diseases which suffer from hyperpyrexia; that is, a temperature in the neighborhood of 106°. On the other hand, it not infrequently happens that cases running a temperature course varying from 101° to 102° are more severe as to toxæmia than those which range in the neighborhood of 103° or 104°, or even 105° for a short time. If, with the drop in temperature which occurs at crisis, the general condition of the patient does not markedly improve, the prognosis is bad. If in place of the ordinary rusty sputum it is of the color of prune-juice, it is usually considered that the disease is malignant.

An important prognostic point in any given case is the degree of toxæmia which is present. In other words, the prognosis depends not so much upon the area of lung which is involved as it does upon the quantity of toxic material which the infecting micro-organisms seem to be producing. Again and again death occurs in apparently otherwise healthy individuals who present a small area of consolidated lung and almost no typical signs of pneumonia, but who are apparently overwhelmed by great toxæmia.

An absence of leukocytosis in a case of croupous pneumonia usually possesses an evil import, since it seems to indicate a degree of toxæmia with which the system of the patient finds it difficult to deal. Indeed, in some fatal cases the leukocyte count may not only not be increased, but very much diminished.

As to the prognostic value of finding the pneumococcus in the blood, there is much difference of opinion. Some observers assert that its presence is of evil import, while others think it of little significance, unless the infection is manifestly severe. The latter view is probably correct.

Treatment.—The treatment of a case of croupous pneumonia varies greatly with the condition of the patient who is suffering from the disease. When it attacks the stout and robust, the only duty of the physician, in a large number of instances, is to watch the patient's symptoms; to insist upon rest in bed in a well-ventilated and quiet room, and to administer a sufficient quantity of bromide, Dover's powder, or morphine to relieve pain, if that symptom is excessive. If, on the other hand, the patient is one who has been addicted to the use of alcohol in excess, whiskey or brandy should be given him in amounts varying with the quantity which he had been accustomed to ingest daily. Not only does his system require the effects produced by this drug, but its use is also necessary to prevent the rapid development of delirium tremens, which is a most fatal complication in these cases. An active stimulation is also usually required in many cases of croupous pneumonia in which the patient is just recovering from some other severe infection, such as typhoid fever.

It is, however, a fatal mistake to think that every patient suffering from this disease should be stimulated. The physician should always bear in mind the important rule not to meddle with the course of the disease unless symptoms are so pressing as to require interference. There can be no doubt that one of the best stimulants in the average case of croupous pneumonia is alcohol, in some form which will agree well with the stomach. The dose of this drug in the form of whiskey or brandy must depend upon the needs of the individual. Rarely will any patient require more than 8 to 12 ounces in the twenty-four hours, and many will do best on much less than this. Valuable adjuvants to alcohol are the aromatic spirit of ammonia, given in the dose of 30 minims, well diluted, every two or three hours; and should any sign of acute cardiac failure develop, Hoffmann's anodyne, in the dose of 1 or 2 drachms, in water, every hour or two, is an invaluable remedy.

For the condition of acute cardiac weakness, the value of strychnine should also be borne in mind. Under these circumstances it is often invaluable, and if need be may be given in full dose, frequently repeated, by a hypodermic needle, until the patient rallies. Usually $\frac{1}{40}$ to $\frac{1}{20}$ grain, repeated once or twice, at an interval of two or three hours, approximates the proper dose. At the present time it has become fashionable for physicians to administer strychnine as a cardiac stimulant throughout the whole course of pneumonia. This is an abuse of a good remedy. Strychnine is not a direct cardiac stimulant. It increases the activity of the heart by rallying the nervous system and acting as an indirect whip to the circulation. If its use is persisted in it soon loses its so-called stimulant effects, and is apt to produce a condition of nervous irritation, particularly in the aged, which may be quite distressing. Its constant use deprives the physician of a valuable remedy for meeting critical moments in the course of the disease.

The value of digitalis for the purpose of combating cardiac failure in acute croupous pneumonia has been questioned. It is a well-known fact that digitalis loses a large amount of its power over the heart in the presence of high fever; and fever is nearly always a marked symptom in this disease. It is also coming to be a well-recognized fact that digitalis is of little value in those cases in which the heart muscle has undergone degenerative change, and the toxæmia of pneumonia often produces such alterations in the muscle fibre of this viscus. In cases in which there is marked vascular relaxation and cardiac dilatation, I have known it to do good when given in a few large doses, particularly if strychnine and atropine were simultaneously administered. Although it is a drug which contracts the bloodvessels, the vasomotor dilatation or relaxation of advanced pneumonia is often so marked that digitalis seems to be unable to raise the arterial pressure, and I am convinced that in many instances death occurs more largely because of the relaxed condition of the bloodvessels than by any direct effect of the disease upon the heart. When I use digitalis, therefore, I am in the habit of prescribing 5 or 10 drops of a physiologically tested tincture every eight or six hours, and the same quantity of tincture of belladonna every three or four hours, in order that the belladonna may increase the tone of the vessels. This treatment, however, is rarely instituted before the fifth or sixth day, or at the approach of crisis.

If cardiac failure is the result of cardiac dilatation due to the obstruction of the flow of blood through the lung, digitalis may be advantageous, but when the cardiac weakness is due to toxæmia it is probably of little value, and if a clot has formed in a cardiac cavity it is manifestly useless. Sometimes when the fever is high and digitalis fails to act, it is well to aid its effect by quieting the heart through the application of an ice-bag placed upon the præcordium. I have also known the reduction of temperature by the local application of the ice-bag and by cool sponging of the body, with friction, to be followed by the manifestation of a distinct digitalis influence. If moderate doses of 5 or 10 minims of a physiologically tested tincture, three or four times a day, fail to produce good effects under these circumstances, I feel quite confident that larger ones will not be of any value.

Should sudden collapse come on, a hypodermic injection of strychnine and atropine should be given, and it may be wise to introduce under the skin, by hypodermoclysis, a pint of normal saline solution, containing 1 drachm of a 1:1000 solution of adrenalin chloride. The normal saline solution, under these circumstances, cannot do much good directly because the relaxation of the bloodvessels is so great that even if it is absorbed its influence will not be felt, but it forms a reservoir from which the adrenalin chloride will be slowly absorbed and so raise arterial pressure by stimulating the walls of the bloodvessels when the toxæmia of the disease has perchance paralyzed the vasomotor centre. When the skin becomes relaxed and bedewed with sweat, atropine is often a life-saving drug.

In cases in which the heart is laboring, where there is evidence of dilatation of its right cavity with pulsating jugulars and other evidences of venous stasis, free venesection may be practised with advantage, and sometimes gives wonderful relief; but in cardiac failure without these signs of venous obstruction, venesection is practically of no value whatever.

The value of inhalations of oxygen gas is problematical. I always employ them because they seem to give comfort both to the patient and his friends. The oxygen should not be given through an inhaler, but be allowed to escape, through the opening of the rubber tube or glass nozzle, about the lips or nose of the patient, for the ordinary individual who is suffering from dyspnœa in this disease will not permit one of his nostrils to be blocked or his mouth closed by such an inhaler, as his desire for ordinary air is too great. If the dyspnœa is due to toxæmia, the oxygen is probably useless. If it is due to a large area of the lung being incapacitated by consolidation, it is conceivable that oxygen can do great good.

The value of saline infusion also depends upon the degree of toxæmia which is present and upon the activity of the kidneys. If, in a given case, the urinary secretion is scanty and toxic symptoms develop, a pint of normal salt solution may be given by hypodermoclysis every six or eight hours for twenty-four hours with advantage. If, on the other hand, the pneumonia complicates renal disease, and there is any tendency to œdema of the subcutaneous tissues, this method of treatment may be disadvantageous, in that it tends to increase the dropsy, and perhaps increases the tendency to pulmonary œdema. As marked toxæmia is usually associated with renal inactivity, this method of treatment should be borne in mind. Direct infusion of a saline solution into a vein is probably not advisable in the majority of cases, since it is usually absorbed with sufficient rapidity from the subcutaneous tissues.

The treatment of the fever during the course of croupous pneumonia is not of as great importance as it is during the course of a more prolonged malady, like typhoid fever. Indeed, there is some evidence to show that fever within moderate bounds may be an effort on the part of the organism to protect itself from the infecting germs. If the temperature does not exceed 102.5° to 103°, antipyretic measures need not be instituted, although sponging the patient with tepid or cool water three or four times a day will control the temperature somewhat, allay peripheral nervous irritation, keep the skin clean, and often produce sleep. These spongings are, therefore, useful in the ordinary case of pneumonia with a temperature of 103° or more, but they are not to be carried out with the same vigor, either as to the activity of the rubbing or degree of cold, as is employed in typhoid fever, for the temperature, as a rule, does not resist the cold, and if it is applied too freely the patient may be thrown into collapse by a sudden fall of fever. Nearly every case of acute pneumonia will be benefited if an ice-bag is kept applied to the head, and if the action of the heart is very rapid when the fever is high an ice-bag over the præcordium, as already stated, is often advantageous.

The administration of antipyretic drugs to patients suffering from pneumonia is absolutely inexcusable. In the first place, antipyresis by drugs is rarely if ever needed. In the second place, there is overwhelming clinical and experimental evidence to show that the use of these drugs materially diminishes the vital resistance of the patient, decreases the ability of his blood to convey oxygen to his tissues, reduces its ability to destroy infecting micro-organisms, lowers vascular tone, depresses the heart, and is altogether evil in its influence, probably also diminishing the elimination of toxic materials by the kidneys, and certainly giving these organs the additional labor of eliminating the antipyretic drug, which, perchance, may be irritating to them.

Quinine is employed by some practitioners with the idea that it possesses antipyretic power, and there is no objection to its use in small doses; large doses, which produce cinchonism or irritation of the stomach, are valueless, and may do harm by irritating the stomach, producing cerebral congestion and meningeal irritation, or irritating the kidneys.

When croupous pneumonia is of the typhoid type and asthenia is marked, valuable results can be obtained very frequently by the hypodermic injection of ½ to 1 grain of camphor, dissolved in sterilized olive oil. This injection may be given once, twice, or thrice in twenty-four hours for one or two days, but ought not to be continued too long; first, because it rapidly loses its effects if used too frequently. and, second, because in these doses there may be some danger of camphor poisoning. Camphor is to be regarded as a remedy for an emergency, and is to be reserved for critical periods.

If great mental and nervous excitement is present and persistent, life can often be saved by the administration hypodermically of ⅛, ⅙, or ¼ grain of morphine. This will often produce several hours of desired sleep, from which the patient awakens much refreshed and perhaps free of the delirium which before the administration of the morphine was an annoying symptom, in that it produced physical exhaustion through the constant activity of his body and mind.

The employment of nitroglycerin in the treatment of pneumonia is limited to those cases which have a high arterial tension. The drug, under these circumstances, is of great value in that it diminishes the work of the heart by removing the *vis a fronte*. If, on the other hand, vascular spasm does not exist, the drug is useless, for it is not, as some have thought, in any sense a direct cardiac stimulant.

The question of the employment of circulatory sedatives in the early stages of acute croupous pneumonia is one which has been widely debated, particularly in this country. There are many excellent practitioners who consider that full doses of veratrum viride or aconite in the earlier stages of croupous pneumonia are advantageous. Statistics, or, to speak more correctly, wide personal experience on the part of many physicians, seems to justify the use of this drug in some cases, namely, in those instances in which the physician sees the patient during the first hours of the attack, and if the patient is a strong, sthenic individual, with a full, bounding pulse, and great flushing of the face. Under these circumstances the relaxation of the general vascular system produced by the veratrum viride and the quieting of the excited heart seems distinctly advantageous. Whether such treatment in any way aborts, or jugulates, or diminishes the violence of the subsequent attack is difficult to determine. In a few instances of acute croupous pneumonia and acute pleurisy, seen in the very early stages, I have noted good results from such treatment. But in the vast majority of instances the physician does not see the patient for nearly twenty-four hours, by this time the disease is well started on its way, and the symptoms of great circulatory excitement have usually passed by, so that circulatory sedatives are distinctly contraindicated.

As the rapid development of the signs of circulatory depression can be aided by the administration of sedative remedies, the use of chloral and the bromides as nervous sedatives in the course of croupous pneumonia is usually inadvisable. Chloral in particular is contraindicated, because of its well-known depressant effect upon the heart and its irritant action upon the kidneys.

The diet should be liquid and consist of milk, with a little pancreatin and bicarbonate of soda, to aid in digestion, and of animal broths and gruels made of wheaten grits, oatmeal, rice, or barley, the digestion of these starchy foods being aided by the administration of small quantities of taka-diastase or pancreatin. I am quite convinced that we too infrequently resort to these cereal fluids in the treatment of diseases of this nature, since they possess much nutritional value and, if their digestion is aided, agree with the vast majority of patients, and enable us to change the diet so that the patient does not become tired of any one particular kind of food, which is a great advantage.

Care should be taken in cases of croupous pneumonia that the patient receives an adequate amount of water to drink, so that the kidneys may be well flushed with fluid in each twenty-four hours; but it is important that only small amounts of fluid be taken at a time, as distention of the stomach may cause fatal cardiac embarrassment. The bowels should also be moved each day in the early stages of the attack by full doses of calomel, and in the later stages by salines, or, if the patient is too weak for the use of these purgatives, by a rectal injection of water or of glycerin and water.

The administration of expectorants in croupous pneumonia is useless until the stage of resolution is reached. Even then they are probably of little value in clearing up the exudate in the vesicular portions of the lung. But the chloride of ammonium, the oil of sandal-wood, guaiacol, and terpin hydrate often prove useful at this time in aiding in removing the symptoms of chronic bronchitis which exist, a state which results in the formation of a good deal of thick, tenacious bronchial mucus, which the patient may have difficulty in expectorating.

Excessive cough in all stages of croupous pneumonia is best controlled by the administration of Dover's powder, codeine, paregoric, or the newer drug, heroin. In the stage of resolution cough sedatives should not be administered unless the physician is certain that the cough is in excess of the needs of the patient in getting rid of the materials in his chest which should be gotten rid of in this way.

Meningeal symptoms are to be treated by the application of cold to the head, and sometimes it is wise to apply a blister to the nape of the neck.

DIPHTHERIA.

Definition.—Diphtheria is an acute infectious disease, which chiefly affects children under puberty. It is due to the Klebs-Loeffler bacillus, and is characterized primarily by an acute local inflammatory process which affects, as a rule, the pharynx, larynx, or nasal mucous membrane, and which is peculiar in that it is associated with the development of a false membrane due to a fibrinous exudate. From the spot upon which this condition develops the general system becomes affected, not by the micro-organism of the disease, but by the poisons or toxins produced by the specific organism at the site of primary infection. Other infections may occasionally cause the production of a false membrane, but the discovery of the presence of the Klebs-Loeffler bacillus determines that the affection is diphtheria. All cases in which a false membrane develops on a visible mucous membrane should be considered to be cases of diphtheria and treated as such until proved to be non-diphtheritic, because in this way the spread of the disease is prevented and the use of the specific remedy, antitoxin, will save life if the disease is present and do no harm if it is not.

In the great majority of cases the disease primarily affects the pharyngeal mucous membrane, or the mucous membranes immediately adjacent thereto, and from this area spreads to the nose or larynx, where the results of its development are very fatal. The specific inflammation and false membrane may, however, develop on any exposed mucous membrane, and even upon the true skin if the epiderm be removed intentionally or by accident.

It is possible for bacteriologists to find the Klebs-Loeffler bacillus in cases of sore throat in which there is no false membrane and no systemic symptoms of diphtheria, and in some of these instances even local disturbances may be absent because of the resistance offered to this infection by some persons. These cases are not to be considered instances of diphtheria, although they are entirely capable of conveying the completely developed disease to others.

On the other hand, cases are not rarely seen in which the physician finds a shaggy false membrane on the throat associated with signs of great systemic toxæmia, and in which the bacteriologist fails to find the specific micro-organism of diphtheria. This condition is called diphtheria by the physician and pseudodiphtheria by the bacteriologist. The streptococcus is probably responsible for some cases of the latter type, while in other patients the pneumococcus causes a similar effect. These instances are met with most commonly as complications of scarlet fever or more rarely of measles, and also occur as manifestations of severe tonsillitis or angina.

History.—Diphtheria has been recognized for many centuries as a disease, but it was not until the clinical observations of Bretonneau, of Tours, that its separate identity was established under the name of "diphthérite." He classed all cases of "putrid sore throat," "cynanche maligne," and "suffocative angina" under this one heading, and much more recently those cases heretofore called "membranous croup" have also been very properly put in the class called "diphtheritic." This sweeping classification is not scientifically justifiable, as has just been pointed out, but from a clinical standpoint it is proper because in the majority of instances the false membrane is due to this cause.

Distribution.—Diphtheria is a disease which occurs in nearly all parts of the world, but is much more prevalent in the temperate zones than elsewhere. It occurs in epidemics and in sporadic cases, and is endemic in nearly every large city. While common in cities, it is even more common in country districts. No special influence upon its development is known to be exercised by bad drainage, although such drainage may, by diminishing vital resistance, very greatly increase susceptibility to the malady.

It is a disease of the poor rather than of the rich, and when it occurs in the well-to-do it is usually sporadic and its source can often be traced to some single exposure. The reason for this does not lie so much in greater susceptibility of the poor as in greater exposure to the infection, for when the children of the well-to-do are attacked they succumb as readily as their otherwise less fortunate fellows.

Diphtheria occurs much more frequently between the ages of two and five years than at any other time of life (Fig. 37).

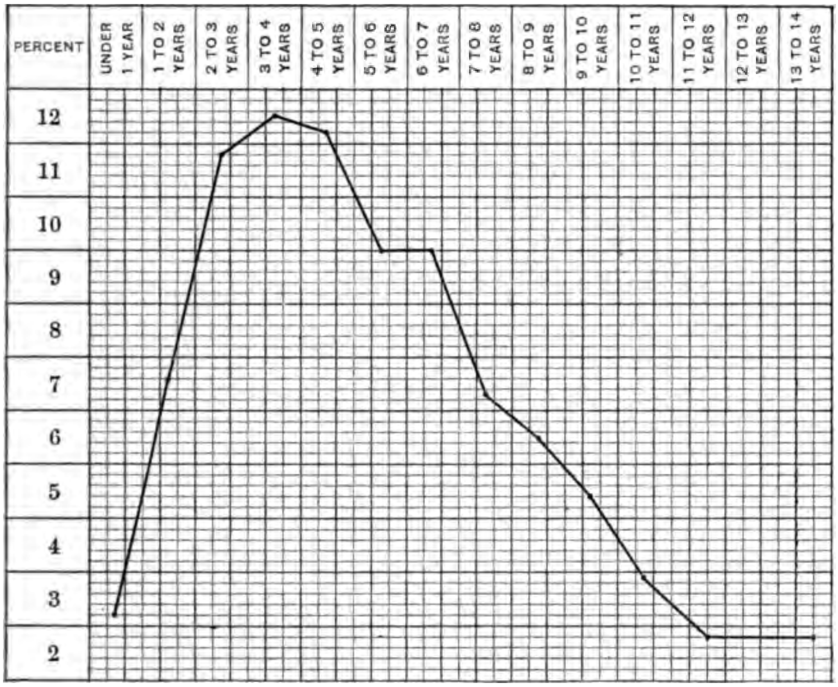
Etiology.—Diphtheria is due, as has already been stated, to a specific bacillus first described by Klebs in 1883, and later isolated by Loeffler. This micro-organism is from 1.5 to 3.5 or rarely 4.5 micromillimetres in length, and from 0.3 to 0.8 in breadth. It usually appears singly, in groups of two or three, but true chains are said not to occur; the organisms may lie side by side or at an angle. They are slightly curved with straight, rounded ends, sometimes branched, and commonly beaded or barred. They do not give off spores, and flagella are absent. They may contain highly refractive bodies which cause them to stain irregularly. The best stain is that of Loeffler, the oval bodies in the organism staining more highly than the rest of the bacillus. They are grown best in Loeffler's blood serum, but develop in all the laboratory media. The organism is non-motile and almost purely aërobic.

All cases of diphtheria are due to the entrance into the body of this specific bacillus originally derived from some patient ill with the disease. The transfer is made in a multitude of ways. Sometimes it is by the clothing, by books, by foodstuffs, or drinks, or drinking-vessels, by pencils, or by the coughing of an individual, who may have the bacilli in the throat, in such a way that the infectious agent is driven into the respiratory passages or mouth of the other person. Convalescent patients may in this manner act as disseminators of the disease long after they are apparently entirely well, and healthy persons with the bacilli in the mouth may also carry the infection.

Thus it is entirely possible for a nurse who has been in charge of a case of diphtheria to carry in the crypts of the tonsils the specific micro-organism, to have no sign of the disease, and yet infect a child or adult whom she may care for soon after leaving the first case. It is evident, therefore, that while the infection is not carried by the air, as in smallpox, it is very easy for a patient who sneezes or coughs to distribute the infectious agent broadcast by its falling on neighboring substances which act as agents of conveyance. These are some of the causes that result in the rapid spread of the disease in tenement houses, schools, and other public places where children are congregated.

As the specific bacillus possesses great vitality, the relationship between cause and effect may not be readily discovered. Thus, if the bacilli fall on a garment they have been found to remain capable of producing the disease six months later, and they have been found in the throat many months after perfect health has been established. So, too, the dust of the room may carry the infection, and even the hair or beard of the physician may do likewise, if the patient expels any secretion upon it. Finally, as already intimated, milk may act in this manner, and cheese made from contaminated milk may even convey the bacillus. Pet animals, such as cats and dogs, also act as distributors, and rodents, such as rats and mice, may do likewise.

FIG. 87



Showing the age incidence of diphtheria, based on 3360 cases collected from various sources.

While all the diseases of birds, cats, and calves characterized by the formation of a false membrane are not communicable to man, the possibility of a true diphtheritic infection, in domestic animals, cannot be denied.

There are a number of causes existing in the patient which exercise a predisposing influence in connection with this infection. Some of these are at present obscure and probably depend upon a lack of anti-bodies in the blood and tissues, but others are equally active, readily recognizable and in many cases remediable. There can be no doubt whatever that chronically enlarged tonsils, overgrowth of the so-called pharyngeal tonsil and chronic catarrh of the nasopharynx very materially increase the susceptibility of a child to diphtheria. For this reason these conditions should not be allowed to exist in otherwise healthy children. Further than this the crypts of the tonsils when diseased may harbor the Klebs-Loeffler bacillus, until a time when the system of the patient is favorable for its growth and then develop rapidly, or on being expelled cause the malady in another individual.

None of the pathogenic organisms seems to possess a greater degree of variance in virulency than the one under discussion. In some instances it fails to exert any malign effect beyond a local influence, and even this may amount to nothing more than a sore throat. In other cases it attacks the patient with a virulence which is perfectly terrifying.

It is not probable that sex has any influence as a predisposing cause. Statistics vary, however, some showing that a greater number of cases occur among boys than among girls. That these differences are merely fortuitous is exemplified by the fact that of 22,005 cases collected from various sources 11,006 occurred among boys and 10,999 among girls, a difference of only seven cases.

Pathology and Morbid Anatomy.—In studying the pathology and morbid anatomy of diphtheria it is essential to remember that the disease is primarily local and secondarily systemic; that the local area of infection is the site at which the specific organism multiplies and produces local changes by its growth, and at the same time elaborates a toxin which, being absorbed, acts on distant parts and so endangers life. The bacillus enters the blood stream in a relatively constant percentage of cases.

LOCAL LESIONS.—The local change produced by the growth of the bacillus is now well understood, for a large number of researches in Europe and America have given us clear conceptions of it. Of these researches by far the most noteworthy is that carried out by Councilman, Mallory, and Pearce in Boston.

The poison produced by the specific bacillus in the mucous membrane of the throat results in the death of the tissues and in this necrotic mass the bacilli then very rapidly develop. The epithelium in many cases manifests more or less proliferation, becomes hyaline and necrotic, eventually fragmenting and disintegrating. The inflammatory exudate which permeates the mucosa and even the submucous stratum is very rich in fibrin elements, and when brought in contact with the necrosing structures forms a fibrous reticulum entangling within its meshes the cells and bacteria. The membrane formed by the coagulation necrosis and hyaline degeneration of the cells may be so transparent as almost to escape detection during life (hyaline type), or it may be granular or fibrillar. The necrosis may extend into the superficial epithelium only or penetrate the submucosa, in some instances involving the parenchyma of the tonsil or even the submucous muscular structures. This necrotic membrane is subject to a number of important changes. It may disintegrate and form a mass of shreddy detritus on the surface or it may be thrown off by being elevated by the exudate which forms beneath it. In the latter process a very thick, false membrane formed of consecutive layers may be produced. The membrane always develops on a necrotic surface, but it may extend a short distance over the surrounding mucous membrane.

The depth of the destructive process is not very great in the majority of cases, but in rare instances it has become deep enough to erode the carotid artery. In many, if not all, such instances other organisms play an important part in the spread of the necrosis and add to the intoxication their own poisonous products. It is important to bear in mind in diphtheria that the infection is "mixed" in the vast majority of cases; that is, the false membrane not only holds in its meshes a multitude of the specific bacilli but many other micro-organisms as well, many of which possess a power for evil, as, for example, the streptococcus.

The membrane is closely attached to the tissues beneath and is stripped off with great difficulty except when it develops in the larynx and bronchi, when it is dislodged quite readily.

The false membrane may develop on any mucous membrane or upon any wound or abrasion, but it most frequently appears on the tonsils. According to Lennox Browne, of London, the relative frequency of its appearance is as follows:

Above the larynx, 84.1 per cent.:	841 cases.
Fauces (including tonsils) alone	672 cases.
Nose alone	2 cases.
Fauces and nose	165 cases.
Mouth or lips alone	1 case.
Hard palate alone	1 case.
Involving larynx, 15.9 per cent.:	150 cases.
Larynx alone	4 cases.
Larynx and fauces	100 cases.
Larynx, fauces, and nose	46 cases.

The growth of the membrane, whatever its site, varies greatly in rapidity, in the area covered, and also in its thickness, but the virulence of the systemic infection is not always in direct ratio to the size of the diphtheritic patch. On the other hand, the degree of secondary infection depends largely upon the particular surface involved, and if it develops in the nasopharynx the toxæmia is apt to be profound. In many severe cases the accessory nasal cavities are infected, particularly the antrum of Highmore.

Visceral and Systemic Lesions.—The action of the toxin of diphtheria is chiefly expended upon the heart, the nervous system, and the kidneys. The *heart* suffers from an acute myositis or inflammation of its interstitial and muscular tissues, and this may be followed by conversion of the muscle fibres into hyaline masses. In a large proportion of the cases in which sudden death from heart-failure occurs the cause lies in the effect of the poison upon the nervous mechanism of the heart, possibly to a greater degree than its effect upon the myocardium. In some of these cases, however, death is due to thrombi in the heart cavities (see Sequelæ), or, again, portions of these thrombi are swept out of the heart and produce embolism in the coronary arteries, in the pulmonary vessels or in the general arterial system. As would be expected from the effects upon the heart, just described, the bloodvessels are also affected. An acute arteritis often occurs and affects particularly the intima.

The *nervous system* is involved chiefly in its peripheral portions. The nerve trunks suffer from acute toxic neuritis and less commonly autopsy reveals hemorrhage into the spinal cord and its membranes as a result of the vascular action of the poison. A much more common spinal lesion is, however, an acute anterior poliomyelitis, that is, involvement of the cells in the anterior horns of the gray matter. Sometimes, too, the posterior nerve roots in the cord may be also affected. The special cranial nerves are also involved in many instances and loss of function in the oculomotor, vagus, hypoglossal and spinal accessory fibres takes place. It is, however, interesting to note that while paralysis, due to peripheral diphtheritic neuritis, may be absolute and widespread, it usually gets well unless the function of some vital part is so interfered with that death speedily ensues. The brain is very rarely affected.

The *kidneys* are more or less affected in all cases of diphtheria. In some there is only a mild albuminuria produced by the irritative effect of the toxin upon the renal epithelium. In more severe cases an acute toxic nephritis develops. This nephritis primarily is parenchymatous, involving the Malpighian tufts and the tubules, but it speedily becomes diffuse. Hyaline degeneration also takes place in the renal vessels, as elsewhere in the body.

The *spleen* is enlarged, markedly congested, and minute hemorrhages are to be seen beneath its capsule. The liver may be found, on cross-section, to be dotted with small areas of coagulation necrosis.

As the infection is most marked in the throat the *cervical lymph glands* are usually infiltrated and the poison may also cause enlargement of the lymphatics in the mediastinum and in the retroperitoneum. The inflammation of the lymph nodes, however, rarely ends in suppuration or extensive necrosis.

The *lungs* are often the site of bronchopneumonia resulting from a complicating pneumococcus infection, but true croupous pneumonia is a rare complication. When great dyspnœa is present because of laryngeal stenosis compensatory emphysema may develop. In the laryngeal form the membrane may extend to the smaller bronchi.

The *blood* is affected very deleteriously by the poison of diphtheria, so that a great diminution in the number of the red cells takes place with a corresponding fall in hæmoglobin. A leukocytosis occurs except in the very malignant forms of the disease. Myelocytes are said to be present in severe cases, and Engel states that in those cases in which they appear in as high a percentage as 2 per cent. death occurs.

Symptoms.—After a period of incubation varying from two to seven days the disease has its onset in the form assumed by most acute infections, namely, with general *malaise*, *chilliness*, and *fever*, the temperature often reaching 102° or even 103° in the first twenty-four hours. The severity of these symptoms varies greatly. In some cases they are so mild that the child is scarcely thought to be ailing, and the physician at his second visit is shocked on examining the throat to find distinct local lesions. In other cases the disease is fulminating in its onset. I have seen a small patch of membrane on a tonsil within twelve hours involve the larynx and necessitate tracheotomy, the membrane involving the external edges of the wound in less than twelve hours more. In nearly every case there is some complaint of *sore throat,* or of difficulty in swallowing arising from this cause. The pharyngeal mucous membrane is reddened and upon the tonsil or tonsils is seen a *tiny patch,* which is the beginning of the *membrane,* but which may be due to the exudate thrown out by a follicular tonsillitis. This membrane rapidly spreads and may extend to the pillars of the fauces, the pharynx, nasopharynx, and the uvula. It is grayish or light mouse color in hue, and in many cases speedily becomes shaggy and dirty looking. If the physician attempts to remove it it is found to adhere to the mucous membrane, and it can be taken off by only tearing it loose, so that a raw, bleeding surface is exposed over which a false membrane speedily reforms for reasons given when discussing the pathology and morbid anatomy of the disease.

There is nearly always some *enlargement of the glands* at the angle of the jaw.

The degree of *systemic disturbance* depends in every case upon the virulence of the infecting bacillus and the rapidity with which the toxin is absorbed.

Some patients who present on examination a large area of membrane suffer slightly in a comparative sense. The fever does not rise above 102° or 103°, the pulse does not go above 100 or 110, and the general state of the patient is favorable. In other instances from the very onset the general systemic state is bad, even when the local changes may seemingly be slight. Even in severe cases, however, the fever is not prone to be high, and often it never rises above 101°.

The *nervous symptoms* consist in restlessness, sometimes in delirium, and rarely convulsions come on. As the disease approaches a fatal issue, the child becomes apathetic and it may be difficult to rouse it.

The *circulation* is feeble and irregularity of the pulse is a very frequent symptom. In White's recent exhaustive study of 946 cases this irregularity was present in 60 per cent. The younger the patient the greater the frequency of irregularity of pulse. Endocardial murmurs, systolic in point of time, occurred in 94 per cent.

Albuminuria is a very constant symptom in these cases, appearing as early as the third day. The albumin may appear in considerable quantities, but the urinary flow is in many cases not greatly increased, although the presence of granular and hyaline casts shows that a true nephritis is present. Dropsy is uncommon.

In cases which are not complicated or treated by antitoxin, and which spontaneously recover, the membrane ceases to grow by the fifth or sixth day, and gradually separates at about the seventh or tenth day, leaving at its former site a bright-red surface which bleeds easily. The nasal false membrane persists longer than that in the pharynx, and often comes away in one mass.

After this period convalescence gradually goes on, the patient being profoundly weak and anæmic and in great danger of sudden death from heart-failure if any sudden change in posture is made. A great many cases thought to be on the high road to recovery meet an unexpected fatal ending at this time.

SPECIAL FORMS.—There still remain to be described the special symptoms connected with those cases of diphtheria in which particular portions of the respiratory mucous membrane are involved, or in which the disease presents conditions which may be considered aberrant. In *nasal diphtheria* the false membrane may be so hidden by the swollen turbinated bodies that it is overlooked until it extends well forward into the nostril, when it may completely occlude the nares. Only a careful rhinoscopic examination will reveal this form in its early stages. Because of the importance of instituting treatment in all cases of this disease at the earliest possible moment the nasal cavities should always be examined at the same time the throat is investigated, and any signs of nasal obstruction taken as of importance. A valuable sign of nasal diphtheria, but one which unfortunately does not manifest itself until the disease is well advanced is a nasal discharge which may excoriate the upper lip.

It is never to be forgotten that nasal diphtheria is a very malignant type of the disease in nearly every instance in which it occurs, and it is particularly prone to affect infants or very young children.

Laryngeal diphtheria manifests itself chiefly by the marked respiratory obstruction which it produces very shortly after the pathological process begins in the mucous membrane of the larynx. Hoarseness on speaking, or crying, and a harsh cough of a metallic sound, sometimes called "brassy," develops. Following these symptoms it is noted that there is slight inspiratory stridor which is accentuated at intervals by what seems to be associated laryngeal spasm. This is followed by persistent stridor, harsh breathing, and manifest unrest and respiratory anxiety. The child may grasp its throat with its hands as if endeavoring to remove the obstruction, and as it becomes livid, partly from mechanical failure of respiration and partly from toxæmia, it often grinds its teeth and looks from side to side for relief, presenting at the same time signs of profound toxæmia. Its pallid skin may be bedewed with sweat. As the disease advances the child becomes more and more limp, and struggles less and less for its breath. In children old enough and strong enough to cough violently in an effort to dislodge the membrane it often happens that they expel pieces of false membrane, and in some instances they may expel complete casts of the larynx. The fever in this type of diphtheria may not be at all high after the larynx has become infected, but, as would be expected, the pulse is usually exceedingly rapid and small.

Laryngeal diphtheria rarely occurs without extension to the pharynx, so that at the time of death the membrane usually covers a wide area. When the pharyngeal and laryngeal symptoms are very marked there is usually great enlargement of the cervical glands.

Bronchopneumonia, due to the inspiration of septic material, is a frequent complication of this type.

Like nasal diphtheria, laryngeal diphtheria has a very high mortality, partly because it causes suffocation and partly because it is associated with toxæmia of a grave type. Sometimes laryngeal diphtheria results in diphtheria of the bronchial tubes.

Diphtheria of the conjunctiva may occur as a complication or as a primary lesion.

Complications and Sequelæ.—The complications, involving the cervical glands, the lungs, heart, kidneys, and nervous system, have already been mentioned. Nevertheless it is proper to say something more concerning them.

Sudden heart-failure toward the close of the attack, or after convalescence is established, sometimes occurs on the slightest exertion. The child sits up to take a drink, or to grasp a toy, or becomes angry, and drops over dead.

In other instances, instead of almost instantaneous cardiac failure with sudden death, a more gradual manifestation of grave heart disease is developed. A patient apparently on the high road to convalescence, except for the reddened throat and profound anæmia, is found to have developed a weak pulse, which flags, and he presents unduly feeble heart sounds. Endocardial murmurs may be present. Sometimes the pulse is abnormally slow. In other cases it is too fast, and, with these circulatory symptoms, some epigastric distress or even vomiting occurs. The pulse becomes weaker and weaker and arhythmia increases, the face is more and more pallid, and cardiac dyspnœa with lividity comes on. Auscultation reveals fetal heart sounds or there may be a "*delirium cordis.*" Death finally closes the scene at the end of twenty-four or forty-eight hours in the presence of gradually deepening asthenia and a mind which is clear almost to the very last. Acute cardiac dilatation may occur.

There are three causes for the types of heart-failure which arise as a result of diphtheria. When the heart fails in the course of an attack it is usually the result of cardiac thrombosis. When it occurs after an attack it is due usually to a toxic myocarditis or to the failure of the nervous supply of the heart through bulbar paralysis or paralysis of nerve fibres. Some statistics indicate that thrombosis is the most common cause of death after disappearance of the membrane. Of course all three of these factors may be present simultaneously. It is probable, however, that thrombosis is more frequently the cause of sudden death than is generally thought. Barbier in 71 autopsies on cases of sudden death in diphtheria found an antemortem cardiac thrombus in no less than 52 per cent. These thrombi were commonly found on the right side of the heart, usually in the right auricle.

Sometimes death is due to *paralysis of the phrenic nerve*, so that diaphragmatic paralysis ensues.

The septic condition of the throat, the labored respiration, the decreased vital resistance of the patient, and the feebleness of the pulmonary circulation in severe cases very greatly predispose the patient to *bronchopneumonia*, and this complication or sequel of diphtheria is the cause of death in a very large number of young children.

Local or *widespread paralysis* often follows an attack of diphtheria, and if it involves vital nerves causes death. On the other hand, these palsies are noteworthy because of the fact that they usually recover. It is by no means uncommon to see a child so paralyzed that it can neither move hand nor foot or even move its head, that lies perfectly limp in its mother's arms, entirely recover muscular power. These palsies are not usually immediate sequences of an attack of diphtheria, but are all the more alarming because they may manifest themselves from one to three weeks after an attack. Again, it often happens that a very mild attack of the disease is followed by this distressing sequence, although as a rule severe cases usually have this result. The palate is the part most commonly paralyzed, and this results in difficulty in swallowing, regurgitation of liquids through the nose, and in a peculiar tone to the voice. When the throat is examined the palate is seen to hang relaxed and motionless when the patient attempts to phonate, and it is also somewhat anæsthetic, so that the contact of food is not well recognized.

The time of onset of the paralysis varies somewhat with the parts involved. The form that occurs most frequently and which affects the muscles of the pharynx and eyes and extremities, or even that of the heart or the muscles of respiration, is a late palsy of the seventh to the twenty-first day of convalescence, whereas that form which affects the palate is more frequently met with at the end of the first week.

True facial paralysis very rarely occurs except as a result of otitis media arising secondarily from the diseased state of the pharynx.

Statistics as to the frequency with which paralysis accompanies or follows diphtheria vary. Hoppe-Seyler states it to be 27 per cent. Johannessen, for all Norway, 12.5 per cent.; the report of the Metropolitan Asylums Board of London for 1900, 18.5 per cent.

The collective investigation of the American Pediatric Society based on 3384 non-hospital cases treated with antitoxin showed that 328 cases of paralysis occurred, which gives a percentage of 9.6.

Hemorrhage from the ulcerative process in the nose may be sufficiently free to seriously exhaust the patient. When subcutaneous hemorrhages appear they are always a sign of very profound toxæmia.

When that practically constant sequel of diphtheria, *profound anæmia*, remains persistently present and is but little improved under treatment, the possibility of *renal disease* being a serious sequel is to be recalled.

Diagnosis.—There is no disease in which it is more important for the physician to make a correct diagnosis promptly than diphtheria, because if it be recognized in its earliest stage it can be cured by antitoxin in the majority of instances. On the other hand, there is no disease which is more difficult of prompt diagnosis in some cases. As diphtherial infection may be present without marked formation of membrane, all cases which manifest sore throat during an epidemic or, after exposure, should receive antitoxin as a preventive, and the throat should be swabbed and the secretion obtained examined bacteriologically for the bacillus.

In every large city at the present time the health authorities provide tubes and swabs for the transmission of cultures from the patient to a laboratory. Usually the swab is delivered in a sterile tube and the culture medium is placed in a second sterile tube. The directions issued by the New York Board of Health are as follows: "The patient should be placed in a good light, and, if a child, properly held. In cases where it is possible to get a good view of the throat, depress the tongue and rub the cotton swab gently but freely against any visible exudate. In other cases, including those in which the exudate is confined to the larynx, avoiding the tongue, pass the swab far back and rub it freely against the mucous membrane of the pharynx and tonsils. Without laying the swab down, withdraw the cotton plug from the culture-tube, insert the swab, and rub that portion of it which has touched the exudate gently but thoroughly all over the surface of the blood serum. Do not push the swab into the blood serum nor break the surface in any way. Then replace the swab in its own tube, plug both tubes, put them in the box, and return the culture outfit at once to the station from which it was obtained."

A loss of valuable time is prevented during the bacteriological test by using antitoxin, but many hours need not be lost if the bacteriologist is skilful. Dr. Park, of New York, who has done such excellent work along these lines, has this to say in regard to this matter: "The examination by a competent bacteriologist of the bacterial growth in a blood-serum tube which has been properly inoculated and kept for fourteen hours at the body temperature can be thoroughly relied upon in cases where there is visible membrane in the throat, if the culture is made during the period in which the membrane is forming, and no antiseptic, especially no mercurial solution, has lately been applied. In cases in which the disease is confined to the larynx or bronchi, surprisingly accurate results can be obtained from cultures, but in a certain proportion of cases no diphtheria bacilli will be found in the first culture, and yet will be abundantly present in later cultures. We believe, therefore, that absolute reliance for a diagnosis cannot be placed upon a single culture from the pharynx in purely laryngeal cases."

Diphtheria is to be separated from tonsillitis with exudation from the follicles of the tonsils and from the diphtheroid false membrane produced by the streptococcus and by an organism closely allied but not identical with the Klebs-Loeffler organism, which is found in scarlet fever, typhoid fever, and measles. Sometimes this can be done only by the bacteriological test.

In follicular tonsillitis the exudate may be scattered over the openings of several follicles, it is rarely as dark in hue as the true membrane, it can be wiped off with an applicator more readily than the membrane of diphtheria, and the tonsillar swelling is marked. The systemic symptoms are, however, of little value in differentiation, because tonsillitis is a disease characterized by very severe symptoms as compared to its gravity, for aching in the back and limbs, high fever, and great evidence of systemic depression are frequently seen during its course. Holt has pointed out the fact that the surfaces of the wound left after tonsillotomy may for a few days closely resemble tonsillar diphtheria, and I have seen the free application of a strong solution of silver nitrate to the pharynx produce an appearance which might readily be mistaken, if examined in a poor light, for diphtheria.

Reference has been made on several occasions to diphtheroid conditions of the throat. These states are probably in a large number of instances due to the streptococcus pyogenes. The false membrane, if none of the true bacilli of Klebs and Loeffler are present, is usually more soft and creamy in its consistency, it is not so tightly adherent to the underlying mucous membrane, and is often very foul. Occurring as a complication of grave infectious diseases such as scarlet fever and typhoid fever, it is dangerous, but otherwise the mortality is not high, being about 2.5 per cent., if we can take the New York city statistics as representative of all cases.

Prognosis.—At the present time it may be said that the prognosis of diphtheria depends entirely upon the promptness with which antitoxin is used. Without antitoxin the death rate varies greatly in different epidemics. In some it reaches the appalling rate of 50 per cent., while in others it is not more than 30 per cent. It is very much more fatal in babies than in older children. Symptoms of evil prognostic import are grinding of the teeth, gallop rhythm of the heart sounds, epigastric pain, and vomiting.

The cases manifesting laryngeal and nasal involvement are always grave as to prognosis. The physician should also be most guarded as to his prognosis as the disease passes its most active period, because everyone of experience knows that an attack of sudden heart-failure often occurs as the child, once more feeling strong, attempts to sit up. A rapid or gradual heart-failure may come on during convalescence. (See Sequelæ.)

Prophylaxis.—As the intimate association of a person, or garments, bearing the specific bacillus with another individual who is susceptible to the disease is essential for its spread, it is evident that by proper quarantine and isolation perfect prophylaxis is possible. All patients who have diphtheria should be isolated at once, and the attendant who nurses the child or adult who is affected should not associate with other persons until after a bath has been taken, the face and head well shampooed and the pharynx and nasal cavities well douched. After the patient is convalescent, it is to be recalled that the specific bacillus may remain in the nasopharyngeal mucus for a long period and so isolation is still essential. The child should not play with other children for at least two weeks, and during this period should have its nasopharynx sprayed daily with some bland antiseptic wash such as Dobell's solution, alkathymol, or normal saline solution. Whenever it is possible, particularly in public institutions, the nasopharyngeal secretion should be examined bacteriologically during convalescence to prove the presence or absence of the specific infecting germ before the child is discharged. As illustrative of this fact the results of the New York Board of Health investigation are of interest. Out of 605 cases examined it was found that the bacilli were not present in 304 on the third day after the membrane disappeared, in 176 they were present for seven days, in 64 for twelve days, and in 36 cases for fifteen days. Twenty-one days after the membrane was gone 12 showed bacilli, and 4 cases showed them for twenty-eight days. Another set of 4 cases yielded bacilli for thirty-five days, and 2 for sixty-three days.

It seems hardly necessary to add that the garments, bedding, and toys of all diphtheritic patients should be destroyed or thoroughly disinfected by steam or formaldehyde. The floors and walls of the room and the furniture should also be treated with formaldehyde, and it should be done as thoroughly as if the case had been one of smallpox. All discharges from the patient should be received in a vessel containing bichloride solution, or, if cloths are used, these should be burned.

A very important measure in all cases in which the disease arises in a family of children is the use of immunizing doses of antitoxic serum for the protection of the well from the disease. There is no doubt whatever that this is a most efficient, never-to-be-neglected measure. The dose is not less than 500 to 1000 units and the protection lasts about three or four weeks. Nurses as well as children should be protected by its use.

When treating or examining the throat the physician and nurse may protect themselves from the discharges by looking through a pane of glass held before the face of the patient.

Treatment.—The treatment of diphtheria at the present time is more scientifically accurate in its basis and in its results than that of any other malady save malarial fever, in which we know not only the cause but the remedy for its removal. *The keystone of the treatment is the liberal use of antidiphtheritic serum derived from the horse.*

It is not necessary in a work of this character to give massive accumulations of statistics to prove that this plan is based not only on scientific laboratory investigations, but upon bedside experience as well. A few instances may, however, be cited as illustrative of the facts. Thus it is interesting to note that a decided decrease in the mortality of diphtheria occurred immediately after the introduction of antitoxin.

The first statistics furnished by Roux are interesting because they permit a comparison to be made between the death rate of the two great children's hospitals of Paris for a certain length of time during which the cases in one were treated by antitoxin—while those in the other did not receive it. From February 1 to July 24, 1894, the mortality at the *Hôpital des Enfants Malades*, where antitoxin was used, was 26 per cent., while the mortality during this period at the Hôpital Trousseau, where serum was not employed, was 60 per cent. From October 1, 1894, serum therapy was practised at the Hôpital Trousseau, with the result that at the end of two months the death rate had fallen to 14.85 per cent. Similar results have been obtained wherever antitoxin has been used. In Geneva the death rate fell from 35.7 to 9 per cent., and the report of the American Pediatric Society, based on 5794 cases, gave a percentage of 12.3. The largest and most valuable statistics are those of Bayeux, who collected more than 200,000 cases from all parts of the world and found that the average mortality rate was 16 per cent. Making a most conservative estimate of a general mortality of 35 per cent. in preantitoxin days, Bayeux's figures prove a reduction in mortality of more than 50 per cent. since the introduction of antitoxin. In the first nine years during which antitoxin was used in the treatment of diphtheria in Chicago the mortality was 6088, while during the nine years preceding the use of antitoxin the mortality was 11,488, a decrease of 5400, or 47 per cent., although the population had increased nearly 600,000, or 52 per cent. If this increase in population is taken into consideration, the decrease in the mortality of the disease under this plan of treatment is 63 per cent. In New York City, out of 1702 cases injected on the first day, including moribund cases, only 85 died—a case mortality of 4.09 per cent. The following figures, which are Baginsky's, show the decrease in mortality according to age:

Before the introduction of antitoxin the mortality of diphtheria according to age was—

0 to 2 years	60.2	6 to 8 years	22.9.
2 to 4 years	51 2.	8 to 10 years	28.8.
4 to 6 years	38.0	12 to 14 years	18.5.

Since the introduction of antitoxin the death rate is—

0 to 2 years	25.88.	8 to 10 years	5.17.
2 to 4 years	17 12.	10 to 12 years	10.
4 to 6 years	17.24.	12 to 14 years	13.3.
6 to 8 years	11.39.		

The physician who fails to use antitoxin, when it is to be had, is guilty of a gross lack of professional knowledge or is atrociously careless of his patient's welfare.

There are several points to be borne in mind in regard to the use of antitoxin, namely (1) it must be employed early in the attack to get the best results, for it is manifest that after the disease has existed long enough to do permanent damage to the tissues no antidote can be satisfactory. No one would expect to give the antidote for arsenic two days after the poison was taken and get good results. Nevertheless, when antitoxin has not been used early it must be given freely in the hope of its aiding the patient sufficiently to help him withstand the infection. (2) The antitoxin should be given liberally. A few large doses in the onset of the attack not only are of great value, but are really economical so far as cost is concerned. (3) It must be given in particularly large doses in cases of nasal and laryngeal diphtheria, because these are forms in which rapid absorption of the toxin of the disease and respiratory obstruction takes place and the malady must be most actively opposed. In these cases it may be useful to give it intravenously. (4) Whenever a person is exposed to diphtheria he should receive a moderate dose of antitoxin to protect him from infection. The dose should be about 500 units, repeated in two weeks, if exposure continues. (5) When diphtheria is suspected to be present it is well to give antitoxin at once rather than run the risk of waiting for a sure diagnosis. In administering antitoxin the following rules should be followed:

1. The skin over the outer surface of the thigh or over the flank or lateral abdominal wall should be cleansed with soap and water and alcohol.
2. The serum should be injected, by means of a syringe or bulb, through a large hypodermic needle which is inserted through the skin where it has been cleansed.
3. The injection should be made slowly and quietly and the swelling which results should not be rubbed.
4. At least 1500 to 2000 units of antitoxic serum should be given at the first dose and repeated in four to eight hours, according to the severity of the case.

5. In nasal and laryngeal cases 3000 or more units for the first dose is usually necessary.

The result of this plan of treatment is often magical. The symptoms of general systemic disturbance, such as a rapid pulse and fever, become modified, the membrane ceases to grow and loses its tenacious hold of the subjacent tissues, becoming not only loose but softened, and speedily disappears.

The only disagreeable effects of using antitoxin in large doses is the subsequent development of some pain or soreness in the joints or the appearance of a roseolous rash; but even if these symptoms appear they are not serious and need give no alarm.

The local treatment of diphtheria consists in the application to the false membrane of peroxide of hydrogen by means of a spray or swab. This active disinfectant disintegrates the exudate and aids in its removal. The other local applications which have been used in the past are painful, injurious, or ineffective as compared to this agent.

The systemic treatment, aside from the use of antitoxin, consists in the employment of foods which are easily swallowed and which will maintain the vitality of the patient to the highest degree, such as concentrated broths fortified by barley-gruel or rice-gruel, which are digested in great degree by the administration of 2 to 4 grains of taka-diastase.

When the pulse flags, small doses of aromatic spirit of ammonia or Hoffmann's anodyne (10 to 30 drops in water may be used) or brandy which is old enough to have a "bouquet" may be given. Full doses of strychnine should also be used for the same purpose. Perfect rest of mind and body should be obtained if possible and great care taken during the illness and during convalescence that no sudden exertion, which may cause cardiac failure, is permitted.

When obstruction of the larynx takes place the patient's life may often be saved by intubation or tracheotomy. In these cases the patient should be kept in a room the air of which is moistened by steam.

The complications and sequelæ are treated in the following manner: The anæmia is to be controlled by the use of moderate doses of reduced iron. Large doses are unnecessary and tend to cause constipation and disorder digestion. A quarter grain three times a day is quite sufficient, given in a small chocolate-coated tablet or placed in a gum-drop. In some instances 3 to 5 minims of tincture of the chloride of iron is equally good; $\frac{1}{100}$ grain of arsenous acid in a sugar-coated granule may be given simultaneously or in alternate weeks. This treatment is also advisable for the relief of the local or general paralysis which is usually associated with marked anæmia. In other instances the syrup of the hypophosphites may be used, and phosphorus is often of value in the dose of $\frac{1}{100}$ grain three times a day. Another remedy of great value as a roborant is cod-liver oil.

In regard to the use of strychnine, which is so largely used as a circulatory and nervous stimulant in all conditions of depression, it should be remembered that it is never a stimulant which in any way increases the nutrition of the part involved. It simply acts as an irritant stimulant. If there is reason to believe that a "whip" is needed to spur atonic nerves to greater effort, it may be used, but if there is any evidence of nervous irritation it is better not to employ it. Aside from the treatment already named there is nothing which can be done to benefit the paralysis.

GONORRHŒAL INFECTION.

There are still some roués, and ignorant persons, who lie under the delusion, at one time prevalent, that an attack of gonorrhœa is of little more gravity "than a bad cold." A considerable number of both classes learn by experience sooner or later that this is a most mistaken conception of the disease. It must not be forgotten that within the last few years it has been proved again and again that the gonococcus may find entrance into the general system from the urethra and there cause the most disastrous consequences. Further than this, while systemic dissemination of the gonococcus usually is secondary to venereal infection, it is to be remembered that gonococcal inflammation of any susceptible mucosa, as the conjunctiva, may afford a point of entrance. Heimann has reported a case of gonococcæmia in which he thinks infection occurred through a wound, and Kimball believes the mouth or upper air-passages may constitute portals of entry. He has also reported a series of cases of *gonorrhœal pyæmia* due to vulvovaginitis in children, some cases being only three months old. If it gains access to the joints, it may not only produce a *temporary gonorrhœal arthritis*, but it may also cause a *chronic arthritis* which is usually multiple, and sometimes is so widespread that the patient is crippled hand and foot, finger and toe, for the balance of his life, the incapacity of the patient being even greater and more rapid in its onset than if he were suffering from rheumatoid arthritis.

Symptoms.—The *appearance of a joint* suffering from gonorrhœal inflammation does not differ materially from that of acute rheumatic fever. It is *swollen* and *exquisitely tender*. The *skin* about the joint is *hot*, but often it is not much reddened; on the contrary, in some cases it presents a peculiar leaden hue. The temperature of the body in general is usually normal, but there is often, at the time of onset, *some fever*. In some cases there is a notable *serous para-arthritis*.

Gonorrhœal arthritis, as a rule, attacks the large joints. It is most commonly multiple, but it may be single. I have seen not only all the large, but the small joints infected simultaneously. Even the sacroiliac, maxillary, and sternoclavicular joints may be involved. This is a noteworthy point when we remember that infection of the maxillary joint almost never occurs in ordinary rheumatism.

According to French statistics the knees are attacked more frequently than any other joint in the body—83 times out of 119 cases; the ankle 32 times, fingers and toes 23 times, the hips 16 times, the wrist 14, the shoulder 12, and the elbow 11.

It must be distinctly understood that gonorrhœal arthritis has no relation whatever to acute articular rheumatism. The tendency on the part of many physicians to call all swellings of joints rheumatism is to be deplored.

The mere presence of heat, swelling, and pain in a joint, with or without fever, does not necessarily indicate that rheumatism is the cause.

Suppuration of a joint as a result of gonorrhœal infection very rarely occurs, but *ankylosis*, due to thickening of the synovial membranes, ligaments, and periarticular tissues, and atrophic changes in the cartilages and in the ends of the bone are met with. In other words, gonorrhœal infection of a joint may result in fibrous ankylosis or in atrophy or in overgrowth of bony tissue, as in rheumatoid arthritis.

The *fascia* in different portions of the body may also be infected. This is particularly apt to occur in the plantar region and not infrequently stiffening and inflammation of the tendo Achillis is met with.

A second serious consequence of gonorrhœal infection is the development of a *gonorrhœal endocarditis*. As long ago as 1854 Brandes recorded two cases of gonorrhœa with arthritis and endocarditis, and in 1862 Traube reported another of gonorrhœal endocarditis without joint infection. None of these cases were proved to be due to the gonococcus because this organism was not known at that time, but in 1893 Leyden proved the presence of the gonococcus in the heart. Since then many more cases have been reported in which the gonococcus has been isolated from the endocardium or the circulating blood. Perhaps the most noteworthy paper was that of Thayer and Lazear in 1899.

There is no special time in the course of the attack of gonorrhœa at which the endocardial involvement takes place. Occasionally it has come in the stage of onset, but in most cases it occurs at about the fifth week. In others it is postponed for weeks, or even for months. In a case reported by Finley and McCrae a fatal endocarditis developed nine months after the onset of the urethral discharge, and when that discharge was no longer present, although a microscopic examination of the urethral mucosa revealed gonococci.

While it is true that these cases are comparatively rare when we consider the frequency of gonorrhœa, it is probable that they occur with more frequency than has been generally thought, and it is a noteworthy fact that in those cases in which the physician is skilful enough to examine the blood, or the endocardium, for the gonococcus, that it is found as a pathogenic micro-organism much more frequently than in those cases in which the physician does not possess such pathological training. With improvements in technique, general gonorrhœal infection will probably be recognized as being by no means as infrequent as it has been thought in the past.

Males are very much more frequently affected by systemic infection of this character than are women.

Systemic gonorrhœal infection follows not only the primary disease in the urethra or vagina, but has been met with in infants suffering from ophthalmia neonatorum due to the gonococcus. In some cases the infection is pure; in others it is mixed.

Diagnosis.—The statement of a patient suffering from an acute arthritis, acute ophthalmia, or, indeed, an acute endocarditis, that he, or she, is also suffering from gonorrhœa will do much toward making the diagnosis of the condition clear. But in the majority of instances the patient neglects to give this important information, and in a considerable number of cases denies gonorrhœal infection of the genitalia, thinking that it can have no bearing upon the inflammations elsewhere, and that therefore it is unnecessary to mention the fact that such a local infection exists. Not rarely patients will deny the existence of a local genital lesion, and it can only be discovered upon careful examination. It may be necessary in some cases to examine the secretions of the urethra or the vagina by staining and by the microscope.

It may be said that in every male suffering from acute arthritis between the ages of fifteen and sixty the possibility of gonorrhœal infection should be considered as having almost equal rank with the possibility of acute articular rheumatism, and the development of endocarditis should not be considered as indicative of one condition more than the other, although as a matter of fact endocarditis is, of course, infinitely more common in true articular rheumatism than it is in gonorrhœal infection. On the other hand the mere discovery by the physician of a presence of a purulent discharge from the urethra does not by any means prove that the patient has gonorrhœal arthritis. It is entirely possible for him to have acute articular rheumatism and gonorrhœa, and, again, it sometimes happens that gouty persons have a purulent discharge from the urethra which does not depend upon the gonococcus. Rarely, too, a purulent urethral discharge is found, in persons who are not gouty, which does not depend upon the gonococcus, but is due to another form of infection.

A therapeutic test of some value lies in the fact that full doses of the salicylates usually cause remarkable improvement in the arthritis of rheumatism, and affect in no way whatever the arthritis of gonorrhœa. Again, it is characteristic of true rheumatism to leave one joint as it affects another; whereas, in gonorrhœal rheumatism it is rare for the inflammation to diminish in the joint primarily affected when other joints become involved.

Prognosis.—The prognosis in gonorrhœal rheumatism is favorable in the majority of instances, provided the infection is not very severe, and is not persistent. The physician, however, must be most guarded in expressing an opinion as to ultimate complete recovery, for, as already stated, some of the severest cases of chronic multiple arthritis are met with as the result of this infection of the joints. The prognosis is also influenced to some extent by the history of the patient. If he has already suffered from previous attacks of gonorrhœal arthritis, the probability of complete recovery is not as good as in primary attacks.

Endocarditis due to gonorrhœal rheumatism is a very serious condition and often results in death.

Treatment.—The treatment of gonorrhœal arthritis consists, first, in the cure of the local area of primary infection as rapidly as possible. For this purpose the ordinary forms of treatment for gonorrhœa are to be followed. The arthritis is to be relieved by the use of a splint and by the application of a 50 per cent. ichthyol ointment to the joint. If the inflammation is exceedingly acute an ice-bag may be employed, and if the effusion is considerable aspiration may be needed to relieve pressure. In some instances the best results are obtained by opening the joint and permitting free drainage. Should the physician place the limb of the patient suffering from gonorrhœal arthritis upon a splint, it should not remain so fixed for any length of time, as ankylosis is particularly prone to ensue. The splint is used only for the relief of pain in the acute inflammatory stages.

As already stated, the salicylates are useless in gonorrhœal arthritis. Indeed, they are worse than useless in that they in no way influence the infection and they are apt to disorder the stomach. Rest in the acute stages and the treatment of the local infection is the most that can be done for the patient aside from local applications. Later, passive movements of the joints and the use of the iodides or of the syrup of the iodide of iron, if anæmia is also present, must be resorted to. Endocarditis is to be treated as is ordinary ulcerative endocarditis.

ERYSIPELAS.

Definition.—Erysipelas is an acute infectious disease due to the entrance into the skin in its deeper layers of the *Streptococcus pyogenes*, sometimes called the *Streptococcus erysipelatis*. The skin of the part affected becomes dusky, red, and swollen. A peculiarity of the area of redness is that it has a sharp line of demarcation separating it from the surrounding healthy tissue, which is usually of its natural color and appearance. The line of demarcation cannot only be seen but can be felt by the finger-tip, and if the affected area be punctured and the serum which then exudes stained with methylene blue the chains of streptococci can readily be found under the microscope. Erysipelas is sometimes called "St. Anthony's Fire."

Frequency.—Erysipelas is found in nearly all parts of the world and is not infrequently present in epidemic form in hospitals and other institutions in which large numbers of persons, with impaired health, are together in wards and dormitories. Under these conditions it spreads rapidly from patient to patient, particularly if wounds afford an entrance into the body. For this reason the outbreak of the disease in an institution should be followed by the immediate isolation of the patient and a thorough disinfection of the entire ward in which he has been lying. The disease occurs most frequently in the spring months, particularly in April, but is met with at all seasons of the year.

Etiology.—As already stated the cause of erysipelas is the entrance into the deeper layers of the skin of the streptococcus in a form which cannot be separated from that which sometimes produces purulent infection in other parts of the body. The anatomical and many of the clinical features of this disease may be produced by several closely allied bacteria, but the clinical manifestations of erysipelas are so constantly associated with the *Streptococcus erysipelatis* that the different infections may be ignored or grouped with this one. Two additional factors are nearly always active in the production of the disease, namely, a break in the skin or in a neighboring mucous membrane, so that the streptococcus gains access to the tissues, and, secondly, some cause, local or general, which diminishes vital resistance to such a degree that the tissues afford a favorable site for the growth of the micro-organism. Thus erysipelas may be due to the infection of a small pimple, by scratching it with the finger-nail, and it is not uncommonly met with in those who are suffering from renal disease, from diabetes, from alcoholism, or from some condition which distinctly decreases the ability of the body to protect itself from infection.

Sometimes the resisting power is decreased by local causes, as by exposure to great cold, but it is very doubtful if this cause alone, with the presence of the streptococcus, is capable of causing the disease unless the general systemic vital resistance is impaired.

The course to be followed in cases of early erysipelas is to examine into the state of the urine at once, and, even if this be found normal, to examine it repeatedly for evidences of renal disease or diabetes, since such causes render the patient very susceptible. Search for other causes of impaired health should also be made, because erysipelas is a malady which is particularly prone to attack those who are already ill, even if the primary illness is not apparent. Occasionally the physician meets with a case in which there is no underlying dyscrasia which predisposes to the disease. In these instances the patient may have repeated attacks, due apparently to general susceptibility to this infection, the streptococci remaining inactive in the tissues in certain cases for weeks at a time. Women during the puerperal period are especially susceptible to the infection.

Pathology and Morbid Anatomy.—Primarily the lesion of erysipelas consists of a hyperæmia; later, an exudate composed of cells and fluid appears in the layers of the true skin, associated with a rapid growth of the streptococcus in the lymph spaces in the margin of and often beyond the inflammatory zone. In severe cases the lesion spreads with great rapidity and may affect not only the deeper layers of the skin, but the underlying connective tissue as well. The destructive action of the bacterial toxin may lead to the formation of sloughs, gangrenous erysipelas, or the polymorphonuclear leukocytes may accumulate in such numbers as to constitute pus, forming the so-called phlegmonous erysipelas. In rare instances the streptococcus, after entering the body through some solution of continuity in the skin or mucous membrane, is carried by the blood or lymphatic system to distant parts, causing a development of the disease far from the site of the primary lesion.

The accompanying visceral lesions may be due to the absorbed toxin or to streptococcæmia. The former may cause degenerative changes, such as focal, or even diffuse, necrosis in the liver, spleen, kidneys, or myocardium. The entrance of the streptococcus into the blood may be manifested in an endocarditis, pericarditis, nephritis, pleuritis, meningitis, arthritis, osseous or pulmonary infections, or other evidences of colonization of the germ in the various organs or tissues.

The onset of erysipelas is associated with a leukocytosis of polymorphonuclear cells, except in malignant cases in devitalized persons.

Incubation.—The period between the introduction of the streptococcus and the development of the disease varies greatly in different cases. Usually the period of incubation lasts from three to seven days.

Symptoms.—In the great majority of cases erysipelas affects the skin of the face about the corners of the nose or near the ear. A *tingling* of the skin is felt which speedily becomes an *intense burning,* and is increased by rubbing or scratching the part. This reddened area spreads rapidly and is characterized at the end of twenty-four hours, or before, by the presence of a *sharp line of demarcation,* which marks the advancing line of inflammation, a margin which can often be felt as a slightly indurated and raised edge. Sometimes the inflammatory process projects well-defined areas of extension into the healthy skin. *Palpation* of the diseased skin also reveals the fact that it is hot and somewhat *brawny and tender.* The color of the part is not a bright red, but is dusky in hue. The *swelling* of the face when well developed is sufficient to render the patient unrecognizable, and the eye, or eyes, may be completely closed by the infiltration of the eyelids. The *ears* when involved become *swollen* to an extraordinary degree and the skin seems very tense and indurated. Not infrequently *blebs* or *blisters* form over the inflamed area.

After the early stage of onset it has been my experience that patients rarely complain very greatly of pain and burning.

The amount of *systemic disturbance* varies very much in different cases. In those who have previously been in moderate health the local lesion and the degree of systemic disturbance may be so slight as to be scarcely noticeable. The patient may complain of a *slight chilliness,* the *pulse* may be slightly *accelerated,* and the *temperature raised* one or two degrees. In other cases in which vital resistance is poor and the infecting germ virulent in form, the symptoms just described are very severe in degree, so that *rigors, high fever,* a *rapid pulse, delirium,* and *great prostration* may be present. In still other cases, not so common, when by reason of great diminution of vital powers the general health has been greatly undermined, as in advanced diabetes or Bright's disease, the disease attacks the patient so vigorously that he sinks beneath its onset without having enough stamina to resist the infection, and may pass into *semi-coma* or even *convulsions* followed by *collapse* due to the apparent exacerbation of the underlying malady by the secondary infection. In rare instances the part involved may become *gangrenous* and death follow from sepsis and exhaustion.

In cases of ordinary severity the *fever* lasts about five days and ends by crisis.

One attack of erysipelas does not protect but rather predisposes the patient to another.

Under the name *erysipelas migrans* a form of the disease is met with in which the disease spreads from part to part and, in the course of its wandering, may affect successively almost the entire surface of the body.

Complications and Sequelæ.—When erysipelas attacks individuals who are greatly impaired in health the results are often grave, not only because the onset of erysipelas is dangerous in itself, but because it is an indication in many cases of a grave disease, hitherto unrecognized, which may speedily cause the death of the patient by an exacerbation. Thus the development of erysipelas in cases of chronic Bright's disease not only means that the renal lesion has resulted in poor resistance, but in addition the task of eliminating the toxins of the new malady may so overwhelm the kidneys that they may cease to perform their function.

Again in cases of greatly impaired health the inflammatory process goes on to suppuration and the deeper tissues become filled with pus, forming the phlegmonous form of the disease. In other instances septic embolism occurs in the lungs, brain, kidneys, liver, and spleen.

A focus of erysipelatous inflammation also results sometimes in the production of *ulcerative endocarditis* or even *purulent pleuritis* or *pericarditis*, but these complications probably are of less common occurrence than has been thought. In 1674 cases of erysipelas collected by Anders from the records of five large hospitals and from private practice, endocarditis occurred only once, and pericarditis not at all. Pleurisy was present in seven cases. Roger, of Paris, did not have a single case either of endocarditis or pericarditis in 957 cases, and only one case of pleurisy.

In 2631 cases of erysipelas croupous pneumonia is said to have occurred in 17 cases, and the catarrhal form in 2. Of these cases 957 occurred in the practice of Roger, of Paris, and the remaining 1674 were collected by Anders, and represent chiefly the statistics of five large American hospitals.

Prognosis.—The prognosis in a case of erysipelas depends largely upon the general state of the patient. As already stated the presence of grave visceral disease, as of the liver or kidneys, renders it very dangerous, but in the great majority of cases, when it occurs in otherwise healthy persons, the outlook is very favorable. Anders' statistics give the mortality at 7 per cent. for hospitals and 4 per cent. for private practice. When it recurs frequently or develops in different parts of the body consecutively, it may cause death by exhaustion, but in nearly all these cases there is a chronic malady as a predisposing cause.

Treatment.—The treatment of erysipelas is *local* and *systemic*. If the bowels are not active they should be freely moved by a dose of 2 grains of calomel followed in twelve hours by a saline purge such as a Seidlitz powder or a half-ounce of Rochelle salts. As soon as the bowels have been evacuated thoroughly the patient should receive 10 minims of the tincture of the chloride of iron, well diluted with pure water, every three or four hours, or 30 minims four times a day. The excess of water protects the stomach from being disordered by the drug and also aids in flushing the kidneys, the activity of which prevents the accumulation of toxic material in the body. The diet should be as easily digested and as nutritious as possible, in order that the vital resistance of the patient may be maintained, and such foods as eggs, rare meats, broths, and milk should be freely given if the digestion of the patient is capable of dealing with them. If it is not, the food should be given in small quantities every two or three hours, and if necessary it should be predigested by a peptonizing tablet or powder

The local treatment is a very important factor in these cases. For many years I have used with excellent results an ointment of equal parts of ichthyol and lanolin, or lard, smeared over the inflamed area and the adjacent skin, and kept in contact with the skin by also smearing this salve on a mask of gauze or lint which is applied to the part so that the medicinal effect is continuous. By this means the pain and burning is almost entirely relieved and a very definite and distinct influence for good is exercised both in curing the inflammation and preventing its spread.

In cases in which the general systemic state is very much impoverished and the vitality of the patient is impaired, sufficient quantities of a good whiskey or brandy should be given to sustain the flagging powers. Moderate doses of quinine (about 3 grains t. i. d.) may also be useful at this time to support the system, but large doses are useless and produce headache and a disordered digestion without causing any benefit. If the fever is excessive it may be controlled by the use of an ice-cap and cold sponging with friction. The coal-tar antipyretics should never be used, as they decrease vital resistance.

SEPTICÆMIA AND PYÆMIA.

Definition and Etiology.—Septicæmia and pyæmia are terms which are dependent upon antiquated ideas of septic processes, and do not strictly represent the states they are now used to describe. Septicæmia originally meant that putrid material was in the blood and pyæmia that the blood contained pus. We now know that blood infection is due to the presence in it of bacteria (bacteriæmia) or to the entrance into this fluid of poisons made by micro-organisms not in the circulation (toxæmia). The older terms are placed at the head of this article because they are still commonly applied.

Pyæmia, as it is understood to-day, is that state in which bacteriæmia is present with associated septic foci, or, in other words, metastatic abscesses. As a rule these abscesses appear chiefly in the tissues which are not far removed from the seat of primary infection. But this is by no means always true, for a septic process in the foot may cause metastatic abscesses in the lungs, kidneys, or liver.

Cases are not rarely seen in which the patient is unable to give any history of even a small abscess or minute break in the skin through which germs may enter the circulation, and yet a diagnosis of septicæmia or pyæmia may be made even when no point of entry can be found.

The obscurity of most of these cases depends upon our inability to find the portal of entry, which may be the genito-urinary organs, the facial or cranial sinuses, the middle ear or mastoid, the mouth or pharynx, possibly the alimentary canal, the biliary passages or gall-bladder, an unrecognized appendicitis or other point of slumbering infection which may or may not be recognized during life or determined with certainty even at a postmortem examination.

Pathology and Morbid Anatomy.—The results of septicæmia are not seen in equal degree in all cases. In some they may be so slight as not to be readily recognized, except by careful bacteriological or microscopic examination. In other instances the secondary results are so patent that the most careless observer cannot fail to be impressed by their character. Thus, it not infrequently is found that septic infection is the cause of a severe inflammatory process in any one of the serous membranes, so that pericarditis, peritonitis, meningitis, or pleuritis may occur. Septic inflammation of these parts results either in a distinctly fibrinous or serofibrinous exudate or in one which is purulent. The synovial membranes and other connective tissues of the joints are frequently infected, so that septic arthritis develops.

Examination of the veins may reveal thrombi near or remote from the primary seat of infection, and these thrombi may be soft and even purulent. It is important that a clear distinction be made between simple or bland thrombi and septic thrombi. Emboli of the former type cause infarction when they plug terminal vessels and mechanically disturb the circulation, whereas septic emboli not only plug the vessel and so disturb blood supply, but as they contain bacteria they constitute new foci of infection.

Very intimately associated with the subject of septicæmia and pyæmia, so called, are the subjects of vital resistance and terminal infection. By vital resistance is meant that power, or property, possessed by the living body of protecting itself from the various micro-organisms which are continually gaining access to the system. This power lies largely in the ability of the blood to exercise its so-called bacteriolytic, or bacteria-destroying, power, and to the ability of the cells of the body to destroy invading micro-organisms by phagocytosis, and manufacture certain other antibodies by the action of which bacterial toxins may be antagonized, neutralized, or rendered inert. A number of valuable papers on the presence of bacteria in the blood have been published within recent years (see Rosenberger in the *American Journal of the Medical Sciences*, January to July, 1903, for facts and references). When, because of diminution of vital resistance the invading pathogenic micro-organisms obtain a foothold and multiply, we have developed an infection. When a patient suffers from some malady which saps his vitality and so causes the approach of death, even in a remote degree, these micro-organisms at once attack his debilitated body, and the patient now suffers from what is called a "terminal infection." Very commonly this terminal infection is the actual cause of death, so that it has been well said that death is "rarely due to the primary cause of the illness."

Symptoms.—The symptoms of septic infection vary greatly with the particular organs which may be the seat of the primary or secondary lesions. The manner of their onset varies likewise. In some instances the earliest manifestations consist in a *rigor* or *chill*, more or less severe, followed by *fever* which varies in its degree with the severity of the infection and the vitality of the patient. The chill and fever are followed usually by a period of normal, or nearly normal, temperature, and this is again followed by chill and fever. In this way the dominant symptoms of the case may closely resemble the quotidian malarial infection, a resemblance which is still further emphasized by the frequent occurrence of a distinct *sweat* as the fever falls. These sweats may be very profuse.

Not rarely the pus in the primary focus of infection changes its character, and becomes less healthy looking. It is thinner and more ichorous, that is, to use a word now rarely heard, it is no longer "laudable pus." The infection causes *general malaise*, *rapid loss of weight* and *loss of appetite* with gastric distress and perhaps *vomiting*. *Anæmia* is rapidly developed, the skin of the hands and face becomes not only pallid but develops a peculiar cadaveric hue, an appearance difficult to describe, but alluded to by those of experience as the "septic facies." Sometimes the skin may be slightly jaundiced.

If the septic process develops secondarily in any special organ, localized symptoms may at once appear, but it is noteworthy that they do not always ensue. *Severe pain in the chest* may betoken the presence of a *septic pleurisy* or *pneumonia*, or if the pain develops in the left side it is often due to septic *infarction of the spleen.* A physicial examination of these organs may reveal the typical signs of these affections.

As the case progresses *pulmonary abscess, empyema,* or *suppuration of the kidney* follows as the result of emboli in these organs. The *pulse* becomes more and more *rapid,* the general state more and more *feeble,* and the patient dies from *general asthenia* or from one of the acute complications just named.

There are other cases in which the onset of the systemic infection is not so pronounced, the chill, fever, and sweat being absent, but in their place a rapid extension of the local inflammatory process with the absorption into the general system of the poisonous products of the germ growth as well as the organisms themselves. In such cases the patient may speedily become not only feeble, but suffer from *stupor* and finally die *unconscious* within a few days of the beginning of the illness. These cases are usually those which, suffering from nephritis or diabetes, offer no vital resistance to infection, and die not only from this cause but by reason of rapidly increasing evidences of the primary disease as well.

Still a third class of cases may be called *subacute* or *chronic,* and last for weeks. Not rarely these cases tax the diagnostic acumen of the physician to the utmost. A child was brought to me in July with the statement that in the previous March she had acute articular rheumatism, but no cardiac complications. The fever of the disease in onset had lasted but a short time, and in its place only a slight evening rise of temperature took place. The acute swelling of the joints disappeared, but they remained tender, and the child was unable to walk. There was marked pallor, a septic hue of the skin, and a large boil on the buttock with smaller ones on other parts of the body. Occasional attacks of vomiting occurred. A diagnosis of chronic septic infection was made, and on the child's death six weeks after evidences of the correctness of this view were found in nearly every organ of the body, although in none of them were distinct purulent foci discovered. Circumstances prevented bacteriological examination of the blood either before or after death.

Diagnosis.—The presence of chill, fever, and sweat in any case should recall the fact that these symptoms may be due to sepsis as well as to other forms of infection. It must be recalled that a history of an infected wound is not needful to reach the conclusion that infection has occurred, for it may take place by a needle-prick, or through a small blister due to a badly fitting shoe, or through a break in the mucous membrane of the alimentary, respiratory, or genito-urinary tract. Typhoid fever often fails to cause death of itself, but a terminal septic infection following it may cause death. In other cases a fatal general infection follows gonorrhœa, and in tuberculosis of the lungs the septic symptoms are due to the pyogenic organisms which are associated with the tubercle bacillus, rather than to that organism itself. So, too, gallstones with septic infection of the gall-bladder may afford the opening for infection. Finally, whenever a patient presents acute arthritis the physician should think first of sepsis rather than rheumatism, and when he has a chill, fever, and sweat which does not promptly yield to quinine, he should also think of sepsis or tuberculosis.

Treatment.—The treatment of these states consists first in seeking and removing the cause and in the support of the vitality of the patient by every possible means, such as fresh air and sunshine, and good food which is easily digested and absorbed. Second, many cases are undoubtedly aided in combating infection by the use of tonic doses (5 grains t. i. d.) of quinine, with or without whiskey. In some instances well-diluted whiskey certainly seems to benefit the patient, and it is surprising how much can be taken without producing any signs of intoxication. Great care that doses large enough to be toxic are not given, for if they are, the toxæmia of alcohol aids the toxæmia of the infection. Full doses of tincture of the chloride of iron are valuable. The coal-tar products are never to be used, as they decrease vital resistance, increase sweating, and do not give any comfort or relief.

If abscesses form they should be opened and drained as early as possible.

When bacteriological tests reveal the presence of streptococci as the cause of the trouble, antistreptococcus serum should be used.

ACUTE RHEUMATIC FEVER.

Definition.—This disease, also known under the name of *acute articular rheumatism*, or *acute inflammatory rheumatism*, is an acute infectious, noncontagious, febrile malady characterized by acute inflammation of the synovial membranes and adjacent tissues about the joints of the extremities. It is to be distinctly separated from the various forms of septic arthritis.

Distribution.—Acute articular rheumatism is a disease which is found chiefly in the temperate zone and rarely occurs in the tropics or in the far North. At present we lack reliable statistics concerning its frequency because in many countries its occurrence is not reported, and in those in which records of the frequency of rheumatism are preserved, so many cases are reported which are not true acute articular rheumatism that the statistics are valueless. Osler states that he saw more cases in Montreal than in Philadelphia and Baltimore while connected with hospitals in those cities. I was firmly convinced from my experience in English hospitals that the disease was more prevalent in England than in the United States, but when I came to the study of the statistics of the relative frequency of acute rheumatism in these two countries, I found that out of 74,808 medical cases in hospitals in London, there were 3822 cases of acute rheumatism, a percentage of 5.1, and out of 73,839 medical cases in hospitals in different cities in the United States, there were 4153 cases of acute rheumatism, a percentage of 5.6. It would seem, therefore, that no marked difference in frequency exists in these parts of the world.

Etiology.—The influence of season upon the occurrence of the disease is marked. It is more common in the cool, damp months of the year than at other times. In London its greatest prevalence is in September and October, whereas in Montreal it is most frequent in March and April.

The influence of age upon the frequency of the disease is notable. It is met with in a very large proportion of the cases between twenty and thirty-five years of age and is very rare in children below five years. After forty-five years it is also rarely met with, comparatively speaking (Fig. 38). It must be remembered, however, that infection with the specific organism of this disease is probably more frequent in adolescents than is commonly supposed, and it may cause very mild arthritic symptoms and yet make a serious attack upon the heart.

FIG. 38

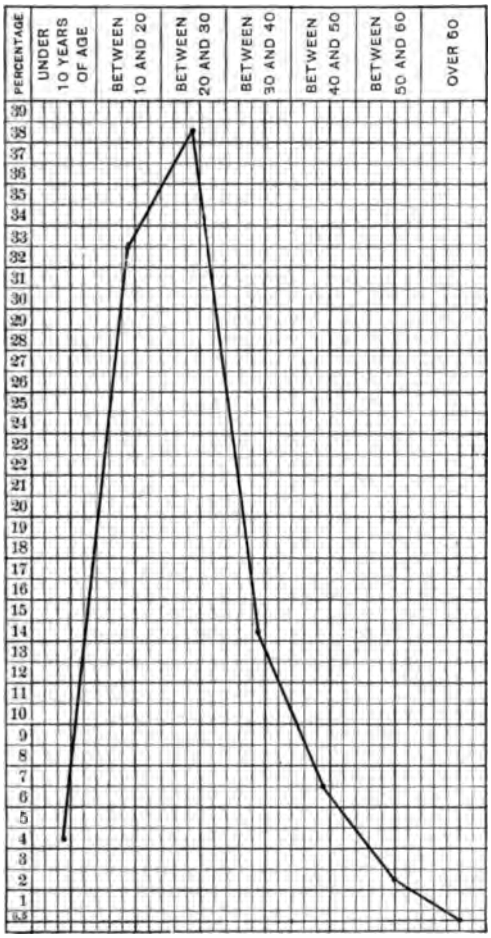


Chart showing age incidence of acute articular rheumatism, based on 4328 cases.

Males are more frequently affected than females, but this proportion is reversed when the patient is under twenty years of age, at which time females suffer more frequently.

The question of the influence of heredity is still undecided. It is probable that it plays a very unimportant part in the causation of the malady.

Of the immediate etiological factors we must include exposure to dampness and cold. These influences are not provocative of the disease unless the specific micro-organism is present, nor unless the exposure reduces the vital resistance of the joints so that the specific organism is enabled to multiply and induce its pathological effects.

Acute rheumatic fever is a disease which is endemic, but it has periods in which it is distinctly epidemic. In other words, it is much more frequent in some years than in others.

There can be no doubt that the infection usually gains access to the general system through the tonsils.

The old theory of acute rheumatism being due to uric acid is now exploded. The excess of uric acid present in the urine in this disease is the result, not the cause, of the affection.

BACTERIOLOGY.—A very large number of investigators have endeavored to isolate the specific micro-organism of this disease, but until recently no satisfactory proof that this had been accomplished was forthcoming. About fifteen years ago Achalme asserted that he had done this, and later Triboulet and Wassermann made similar claims. Up to the present time it would appear that Poynton and Paine have come nearer to success in this line of research than any of their predecessors, and Meyer has also carried out studies which seem to prove the correctness of their conclusions. Still more recently Walker and Beaton have further confirmed the correctness of the views of Poynton and Paine. These last investigators believe it is impossible to separate this special organism, for which they advise the name *Micrococcus rheumaticus,* from the ordinary streptococcus. Meyer succeeded in obtaining it in a form which produced all the lesions found in the course of the disease, and found the same organism in the sore throat, in the inflamed endocardium, in the pleura, and in the inflamed joints of persons attacked by this malady. More recently still Poynton has further enforced his views. The most that we can say at present is that acute articular rheumatism appears to be a streptococcic infection. Until, however, the supposed exciting cause can with certainty be differentiated from the streptococcus found in other lesions, the propriety of giving to it a special name must be regarded as doubtful.

It is a fact worthy of note that the introduction of many pathogenic micro-organisms into the body will result in endocarditis and arthritis, but these states are not true acute articular rheumatism.

Morbid Anatomy.—The changes produced by an attack of acute rheumatic fever are not pathognomonic. On the contrary, the condition of the synovial membranes is one of more or less intense hyperæmia with the effusion of fluid into the surrounding tissues and into the joint itself. The synovial liquid is turbid and contains leukocytes and some small flakes of fibrin.

The secondary changes produced by the disease are vegetative endocarditis, acute pleuritis, and pericarditis, but there is nothing about these lesions which is peculiar to this specific infection. (See Endocarditis.)

Symptoms.—The symptoms of acute articular rheumatism are usually *sudden in onset.* With or without premonitory signs of illness the patient awakes to find that one or more of his *larger joints is acutely inflamed* so that any movement causes great pain, and the part may be so sensitive to the touch as to prevent any thorough examination by palpation being made.

The *skin* over the affected part is usually *dusky red* in hue, and quite *puffy* in appearance because of the presence of subcutaneous exudation, and the inflamed area is much more *hot to the touch* than adjacent tissues. Sometimes, however, no local redness is seen, but in its place swelling and a peculiar sodden and livid hue of the skin. With the development of this *arthritis* a *distinct febrile* movement begins, and the fever may reach 102° or 103° on the first day in many cases. This level of temperature is not usually exceeded, but the variations in its course are very marked in that it is subject to great remissions, particularly if the *sweating is profuse.* The fever ultimately falls by lysis.

The *tongue is coated,* the bowels are usually confined, and the skin is hot and dry, or at times bathed in a profuse *sweat.* This sweat breaks out in paroxysms and is exceedingly acid, possessing a peculiar acid odor. It is probably an effect at elimination, but it does not develop in all cases. The *urine* is nearly always scanty and concentrated, highly acid, and on standing deposits urates in excess.

Acute articular rheumatism is characterized by the *speedy spread of the arthritis* to joints in other parts of the body. In some instances the involvement of a second joint is followed by diminution of the inflammation in the joint first affected, but in many instances the patient suffers from a progressively developing arthritis which soon involves almost all the larger joints. This fugitive character of the inflammation, wandering from joint to joint, is so very characteristic, that the presence of a monoarticular inflammation is a point against the disease being true rheumatism. The smaller joints, such as the fingers and toes, usually escape, but they are often apparently affected by reason of the fact that the swelling of the tissues extends from the large joint above so that it covers the smaller ones. In other instances, however, the joints escape severe infection, and the synovial coverings of the tendons suffer chiefly, so that parts near the joint may be swollen, and the swelling is purely periarticular. The vertebral and clavicular joints are very rarely affected.

There is no form of acute arthritis which seems to give the same amount of *severe pain* as does that of this disease, and the close of the attack usually leaves the general system of the patient considerably shattered because of the severity of his suffering, his *marked anæmia,* and the *exhaustion* caused by the sweats and the lack of sleep. The pain is also peculiarly trying because the state of the joints is such that movement is impossible and the patient gets bed-sore and bed-frantic.

In some cases a *subacute type of the disease* develops in which all the symptoms, in the joints at least, are very mild, but these cases are very prone to manifest *cardiac complications* later on. This is particularly true in children.

One attack usually predisposes to another. It certainly in no way protects the patient from subsequent attacks.

Duration.—The disease may run its course in a week or be continued over a very long period of time, lasting a month and causing great discouragement of both the physician and patient. In some instances, the arthritic state merges by degrees into one of chronic arthritis without fever, and the patient never fully recovers the free use of his joints, but this is fortunately very rarely the case. Even when the amount of inflammatory exudate seems very large gradual and complete absorption usually occurs.

Complications.—The complications of acute articular rheumatism are the means by which it produces fatal results, and they are most frequently found in the *heart muscle*, in the *cardiac valves*, or in *the pericardium*. The infection attacks the endocardium with great constancy, and even in those cases in which the arthritic changes are slight, it often plays the greatest havoc in the heart, so that we frequently see young persons with grave cardiac lesions who have presented such mild articular symptoms that the presence of rheumatism has been overlooked. Of 842 cases of acute articular rheumatism, all of which were first attacks, valvular heart lesions were present in 420, or 50 per cent. Of these lesions, 390 were mitral, 16 aortic, and 14 mitral and aortic.

The following table arranged from the statistics of Church at St. Bartholomew's Hospital illustrates not only the frequency of cardiac complications but also the age incidence as well:

Age.	Number of cases.	Number in which heart was affected.	Percentage of heart affection.
Under 10 years	25	20	80
10 to 20 years	244	170	69.16
20 to 30 years	241	124	51.1
30 to 40 years	115	35	30
40 to 50 years	41	9	21.39
Over 50 years	17	7	41.03
	683	365	48 78

This table includes pericarditis as well as valvular lesions.

It is generally recognized as a fact that heart lesions arise less frequently in the old than in the young, and therefore the percentage of 41.03 given for cases over fifty in this table is probably excessive.

These lesions are rarely lethal during their acute stage. They develop into subacute or chronic lesions, and days, weeks, or years after the patient has recovered from the acute illness become active agents in destroying life or incapacitating him for work. The reason for this lies in a continuation of the *endocarditis, in a subacute or chronic form*, for days or weeks after the acute symptoms have passed by, with the result that the valves become shrunken, and so are unable to perform their proper functions; or they become glued together, and in this way their action is interfered with. Acute articular rheumatism with cardiac complications may therefore cause death many months or years after the acute attack. (See Endocarditis.)

Pericarditis is by no means as frequent as endocarditis. It is usually of the fibrinous or serofibrinous type, and occasionally it is purulent, particularly in the case of children. In rarer instances a *myocarditis* develops.

It is of the greatest importance that the physician in charge of a case of acute articular rheumatism be continually on the lookout for cardiac complications. He can do something toward preventing these by following the directions given under Treatment, and by insisting upon absolute rest. The vast majority of cases of acute articular rheumatism manifest sometime during their course a more or less well-defined mitral murmur, and sometimes a pericardial friction sound. In many instances, instead of these lesions increasing in severity, they disappear with the subsidence of the joint symptoms.

Pulmonary complications are not exceedingly common. Stephen Mackenzie found that pneumonia, or pleurisy, occurred in about 10 per cent. of 3433 cases. Not infrequently slight pulmonary congestion of the bases occurs.

The *nervous complications* in acute articular rheumatism arise from three causes: the high fever, the profound toxæmia, and the nervous irritation and exhaustion produced by many hours of suffering. *Delirium* is not commonly met with. An active, *noisy delirium* sometimes develops as a result of the administration of large doses of the *salicylates*. Such mental disturbance is said to not infrequently complicate the development of rheumatic pericarditis. Sometimes, too, excessively high temperature is associated with delirium.

Meningitis occurs as a very rare complication.

The relationship between *chorea* and *rheumatism* is not clear. There can be no doubt that they bear some relationship one to another, but whether rheumatism is to be regarded as an etiological factor in chorea is undecided.

The skin lesions of acute articular rheumatism consist chiefly in the development of *urticaria* and *erythema*. *Purpuric rashes* sometimes appear, hence the somewhat old-fashioned term "*purpura rheumatica*;" but it is probable that these extravasations under the skin are due to an associated infection rather than to the rheumatism itself. Another very interesting lesion occurring as a sequence of acute articular rheumatism are *rheumatic nodules* which vary in size from a small pinhead to a large pea, and develop chiefly on the hands and wrists and about the elbows and knees, and sometimes upon the back over the spine. They often last for months, and are seen more frequently in children than in adults. Indeed, they are so characteristic in children that they may be regarded as a positive sign that rheumatism has at some time been present. They are not, however, pathognomonic of rheumatism in all cases, for they appear in gouty and rheumatic subjects who have never suffered from the acute form of the disease.

Diagnosis.—The diagnosis of acute articular rheumatism is by no means easy in all cases, but the presence of fever with progressive involvement of one joint after another is very indicative. The great difficulty lies in separating the various nearly related forms of septic arthritis from true rheumatism. If there is present a pre-existing septic focus from which septicæmia may arise, the probability is that the malady is not the specific disease we are discussing. Thus, a multiple arthritis, with or without fever, often follows or complicates gonorrhœa, and follows scarlet fever, typhoid fever, and dysentery, or any disease which, by providing a source of infection, exposes the joints to the invasion of micro-organisms.

The chief conditions, aside from gonorrhœal rheumatism and ordinary septic arthritis, that we must carefully exclude are acute osteomyelitis, which usually affects the femur, and which if it is not recognized early may destroy the patient's life; monoarticular inflammation, which is often due to syphilis, and acute gout, in which case the history of previous attacks of pain in the smaller joints will be present and the inflamed area will probably be in the ball of the thumb or in the joint of the great toe. In children an acute arthritis with little fever sometimes develops and soon suppurates. It is undoubtedly due to septic infection. Finally, let it be borne in mind that the mere presence of heat, pain, swelling and fixation, in a joint should not be called acute articular rheumatism until all other possibilities are excluded. Perhaps no more frequent error occurs than the calling of all forms of acute arthritis " acute rheumatism."

Prognosis.—Death very rarely ensues as a result of acute articular rheumatism without any complications. This is well shown by the following statistics. In 8431 cases of acute articular rheumatism collected from the official reports of several American and English hospitals there were 127 deaths, which gives a percentage of 1.5. From the years 1880 to 1903 1524 cases were treated in the Pennsylvania Hospital with only 14 deaths, a mortality of 0.9 per cent. While, therefore, acute rheumatism rarely causes death during its presence in the active stage it is nevertheless true that no acute disease causes death so frequently in after years because of secondary changes in the heart.

Treatment.—The treatment of acute articular rheumatism is at times eminently satisfactory and at others equally disappointing. In a certain proportion of cases, in which treatment fails to produce good results, the condition is perhaps maintained by the presence of associated infections which help to produce the arthritis. While no true specific exists for acute articular rheumatism, the salicylates act in some cases with a degree of celerity which is most gratifying, and therefore they are always to be considered as the most important remedies when the physician is called upon to treat a case of this disease. The chief objects of the physician under these circumstances are the alleviation, modification, and shortening of the disease, and, second, the protection of the heart from the secondary affections of its endocardium, its muscle, and its pericardium. For the relief of the pain and of the inflammatory processes in the joints the best remedy is the salicylate of strontium in the dose of 15 to 20 grains from three to six times a day. It should be given in capsules and followed by a copious draught of water or milk to prevent it from irritating the stomach. Sometimes a few swallows of the emulsion of sweet almonds may be taken to protect the stomach from irritation.

If full doses of the salicylates, sufficient to produce distinct physiological symptoms, such as fulness in the head and some deafness, do not produce signs of improvement in the course of five or six days, they will probably fail to cure, and had better be discontinued, as after this time they are apt to increase the discomfort of the patient, to disorder his stomach, and to increase the sweats. In their place the patient may receive 10 minims of the wine of colchicum root and 15 grains of iodide of potassium three times a day. While the salicylates are being given it is always advisable to give not less than 40 to 60 grains of sodium bicarbonate or bicarbonate of potassium in each twenty-four hours. The sodium bicarbonate seems to aid the stomach in withstanding the salicylates, and provides the body with a certain amount of alkali which is advantageous.

Copious draughts of water are always to be given in rheumatism for the purpose of flushing the kidneys.

For the prevention of endocarditis and pericarditis from four to six small fly-blisters may be placed over the præcordium, and their influence as preventive measures is thought to be aided by the free use of the sodium bicarbonate just named. If pericarditis develops and the action of the heart is very excessive, small doses of aconite may be cautiously given to act as a cardiac sedative. But this drug is not to be used if the patient is markedly depressed. Sometimes an ice-bag placed over the heart acts equally well.

The joints are best treated by anointing them with ichthyol and lanolin in equal parts, applying an excess of this ointment, and then wrapping them in cotton-batting. When the patient suffers pain because of the twitchings of his muscles, which in turn move his inflamed joints, some relief and comfort can be given by applying a splint to produce fixation of the joint.

The acute inflammatory process in the joint is usually severe enough to make the patient content to remain in bed. But it not infrequently happens that as the pain in the joint diminishes the patient is most anxious to get about and return to his usual pursuits. Nothing can be more dangerous than the pursuance of such a policy. A very large proportion of cases of valvular heart disease are due to the fact that the patient has suffered from rheumatism, and has returned to his occupation before the endocarditis produced by the rheumatic poison has entirely disappeared. For a time he may be able to perform his customary duties, but the increased labor thrown upon his heart by exercise causes a delay in the healing of the lesions in his endocardium, and as a result he suffers from mitral stenosis or mitral regurgitation, which sooner or later will make him a cardiac invalid. Even if these symptoms are not manifested for some time after the attack of rheumatism has been present, they may nevertheless become dangerously active when with advancing years cardiac compensation is lost. The physician should therefore impress upon every patient, with acute articular rheumatism, who insists upon rising as soon as he feels well, the fact that he is taking his life in his hands by so doing. Even after all articular symptoms are passed by, the patient who has had this disease should remain in his bed for at least two or three weeks, and this advice holds good even if during the attack no signs of an endocardial murmur have been manifest.

CHOLERA.

Definition.—The word cholera when strictly applied is used to designate a disease which is characterized by profuse serous purging, cramps, vomiting, and extreme prostration, and which is due to an infection of the bowels by the specific micro-organism of this disease, called the *Spirillum choleræ asiaticæ,* which, as it is often broken into short, curved rods, is frequently incorrectly termed the "comma bacillus." When it is desired to indicate that the true disease is present the term "Asiatic cholera" is used to distinguish the malady from other forms of serous diarrhœa of a severe type, such as cholera morbus or cholera infantum.

History.—Prior to 1817 cholera was confined to certain parts of India and never infected districts far removed from them. It is probable that the disease has occurred for many centuries, but it is a noteworthy fact that, unlike most epidemic diseases of pronounced characteristics and high mortality, no clear description of its presence was placed on record. Since 1817, when an epidemic of unusual severity broke out in India, it has been known to be constantly present in endemic form in some parts of that country, and it has from time to time been carried thence along well-travelled routes by pilgrims and travellers, or by their possessions, until many parts of the earth removed thousands of miles from the original focus have suffered from it. Seven distinct invasions of Europe have occurred since 1817, and the last one from 1891 to 1895. The disease was first introduced into America by emigrants who landed in Quebec and New York early in the decade of 1830 to 1840.

Distribution.—The geographical area of origin has already been described. The disease may occur in any part of the world to which the specific germ may be conveyed.

Etiology.—The cause of epidemic cholera is the spirillum which was first isolated by Koch. It is spiral shaped or assumes the form of segments of a spiral, or short curved rods and S forms.

The degree of curve varies greatly; sometimes the organism is almost straight, at other times it forms a partial circle. Bizarre forms also occur. It is active, motile, and flagellate. The bodies described by Hueppe as spores have not been so considered by other observers.

Cholera is distinctly a water-borne disease in the vast majority of epidemics. The specific organism gains access to the body through contaminated drinking water or soiled food. In the Hamburg cholera epidemic of 1892, about 18,000 persons were stricken, and of this number 8000 died. In the city of Altona, which is really a part of Hamburg, and which also derives its drinking water from the Elbe, there were only about 500 cases of cholera in a population of 150,000. Hamburg had no filtration plant at the time, while Altona had a sand filtration plant. It is only by water and food that cholera can be transmitted, except that if choleraic stools are desiccated, and the dust is blown on food or into the mouth, it is conceivable that the infection may occur. Aside from the rarity with which this accident takes place, the fact that the bacillus speedily dies, when dried, militates against it being active under these circumstances. A more possible and indeed probable method by which the infecting agent may reach the food is its carriage by flies, for in the body of the common house-fly the specific organism may exist for twelve days.

Hot weather favors the spread of the disease. As in all infectious maladies, all causes which decrease vital resistance, such as alcoholism, exposure, convalescence from other diseases, and even profound mental depression, distinctly increase the susceptibility of the patient.

Prevention.—It is evident from what has been said that there is no reason why cholera cannot be prevented, and it may be said of deaths from cholera, as it is said of deaths from typhoid fever, that every one is preventable if proper care is taken to destroy all the specific organisms the moment they escape from the body of a patient suffering from the malady. That they are not destroyed in cholera is all the more to be condemned by reason of the fact that they escape only in the stools, whereas in typhoid fever the specific bacillus escapes by the urine, the skin, and perhaps the saliva. The cholera spirillum is exceedingly susceptible to bactericides and particularly to acids, under favorable circumstances succumbing to such weak acids as vinegar.

All dejecta from cholera patients should be destroyed by heat or by the action of chlorinated lime, or formaldehyde, or of corrosive sublimate, contact with a solution of which should be complete and prolonged for at least one hour, for in the latter instance the mercury salt may combine with the albumin, or be decomposed by the gases in the stools.

During the presence of the disease no food should be taken in the raw state, and it should be cooked immediately before it is eaten, in order that there may be no time for it to become infected after it is cooked. With these precautions the danger to physicians and nurses is reduced to a minimum. When there is a possibility of negligence, a valuable prophylactic is the use of dilute sulphuric acid in the dose of 5 to 10 drops in water three times a day after food. This does good, by reason of the fact that dilute acids kill the comma bacillus, and again because this acid acts as an astringent remedy in diarrhœa. Care should be taken during an epidemic that bad food and exposure are avoided, as this may prepare the way for infection.

Through the researches of Haffkine in India, it would seem that it is possible to immunize human beings against cholera, but this plan of inoculation is of no value when the disease has once developed.

During the years 1894 and 1895 Haffkine inoculated 3951 individuals with his anticholera vaccine. Of this number, 33, or a little less than 1 per cent., contracted the disease, whereas, of 9335 individuals who were uninoculated and similarly exposed to the infection, 210, or 2.24 per cent., were stricken. These observations were made in India.

In July, 1902, an epidemic of cholera broke out in the prefecture of Nagasaki, Japan, and preventive inoculations were at once begun. Of 21,334 persons who were inoculated, 110 contracted the disease. In previous epidemics the number of persons affected ran well up into the thousands, but it is but fair to state that in this epidemic only 741 cases occurred in that prefecture among the uninoculated. As the number of uninoculated inhabitants is not stated, we cannot judge of the real value of the plan.

Haffkine's conclusions as to the result of anticholera inoculations are as follows:

1. The inoculation produces an effect within four days.
2. During these four days a difference in susceptibility shows itself in favor of the inoculated.
3. After the expiration of the four days and during a period of at least fourteen months, a high degree of resistance to attack is observed in the inoculated.
4. The proportion of deaths to cases is not influenced by the inoculation.

Pathology and Morbid Anatomy.—After death from cholera postmortem rigidity comes on very rapidly, and is persistent to such a degree that distortions of the limbs and body may be present. In typical cases the entire body appears shrunken and wasted and the dependent portions rapidly become livid. Not rarely a postmortem rise of temperature takes place.

When the body is incised the tissues are found to be devoid of their normal moisture, and the blood in the great vessels is thick and dark in hue. The stomach is empty, its mucous membrane is congested, and, in some instances, ecchymoses may be present.

The chief changes are to be found, however, in the lower part of the small bowel. Its mucous membrane is boggy or sodden, and covered by a glutinous material which is readily detached. Not rarely the mucous membrane is stripped off in patches or shed in flakes. These changes may extend as high as the duodenum, and in the lower ileum Peyer's patches and the solitary glands are found to be swollen and congested. There may be present a diphtheritic exudate, which is adherent in part, and in part is fleecy or flocculent in appearance. Deeper ulcerations and perforations are exceptional. Hemorrhages may also be found in the mucous membrane at this place.

Notwithstanding the active purgation, the large bowel in cholera is not as much altered as is the ileum, the only change, as a rule, being an acute catarrh of the mucous membrane.

It is important to the student to recall the fact that cholera is characterized by changes in the small bowel, whereas dysentery is chiefly characterized by changes in the colon.

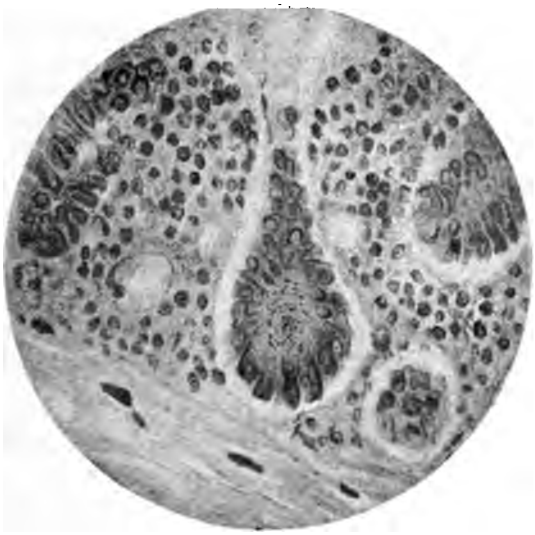
The intestines are contracted, their coats thickened, and the peritoneum possesses a peculiar rosy hue. The mesenteric glands are enlarged and infiltrated.

Granular changes in the large glandular viscera are present in a certain percentage of cases, and a complicating nephritis is occasionally seen. The kidneys may be enlarged and the vessels congested. Under the microscope the uriniferous tubules are seen to be filled with granular casts, but the tufts are not materially changed. The great loss of fluid by the serous discharges and the lessened absorption of liquids causes concentration of the blood and greatly interferes with the excretion of poisons by the kidneys.

The liver is not enlarged but rather shrunken, and its cells show, under the microscope, cloudy swelling, with patches of fatty degeneration. The spleen is usually small. The heart is flaccid and the lungs shrunken.

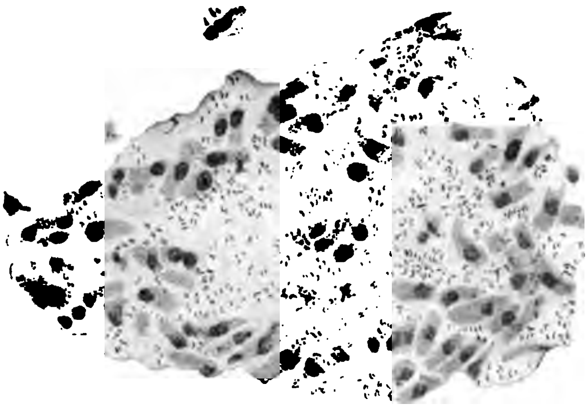
The cholera organism is found in immense numbers in the contents of the bowels and in the discharges of patients suffering from this disease, but, unlike the typhoid bacillus, it is not usually widely disseminated through the body (Figs. 39 and 40).

FIG. 39



Comma bacilli in the fundus of a gland of Lieberkuhn in the small bowel in a case of Asiatic cholera. (Kast and Rumpler.)

FIG. 40



Comma bacilli in the intestinal contents. (Kast and Rumpler.)

Symptoms.—The symptoms of cholera develop in from a few hours to ten days after infection has occurred. The average period of incubation is usually three to six days.

The earliest symptoms, aside from a feeling of depression, is the onset of *watery diarrhœa,* which may be associated with pain. The patient suffers from the *weakness* and *depression* characteristic of ordinary watery diarrhœa, and if the passages are very profuse there may be *great feebleness* and even *collapse.* In the majority of cases, however, the onset of the disease is more *abrupt* than that just described. With almost no indication of impending illness the patient is seized by *active vomiting* and *purging*, by *severe cramps* in the extremities and trunk, and passes into *collapse.* The stools, as soon as all the ordinary intestinal contents have been washed out, are rice-water in character; that is, on standing they separate into two layers, the upper clear and opalescent, the lower full of flakes of mucus and exfoliated necrotic mucosa.

The amount of serum lost by the purging is very large, and it is expelled with considerable force. Because of the large quantities of fluid lost by this means the urine becomes scanty and suppressed. This loss of fluid, combined with the changes in the kidneys, results in uræmia, which, of course, aids greatly in increasing the toxæmia of the disease. The vomiting is not only violent, but *persistent retching* may greatly exhaust the patient. The cramps in the muscles are due to the rapid abstraction of fluid from their tissues and perhaps, in part, to the toxæmia of the disease.

In about half the cases recovery begins to take place at this stage by a gradual modification of the symptoms, but if the patient is too ill to recover, the *second stage*, or that of *collapse* and *profound asthenia*, now develops. This stage may last from a few hours to two days. As it proceeds the patient becomes so feeble that the respirations become shallow. The fluid stools pass from the bowel involuntarily, escaping rather by relaxation of the sphincter than by the conscious act of the patient. Feeble attempts at emesis may still persist, and the cramps may be more severe than before.

As the exhaustion deepens the *pulse* becomes a *mere thread* at the wrist, and may even be *imperceptible* in the great vessels. The *heart sounds* become more and more *indistinct*, and occasionally soft murmurs are heard.

The face now bears the *Hippocratic expression*, the *nose* is *pinched* and *pointed*, the *eyes sunken* and surrounded by dark rings, the mouth is partly open, the teeth covered with sordes, the skin of the entire body is livid and often bedewed with a cold sweat. The *voice* is *whispering*, the thirst excessive, and the mind clouded. Toward the close of life *stupor* or *coma* mercifully relieves suffering. Finally, with a continued fall of bodily temperature, death takes place.

When the *stage of reaction* develops, before these grave symptoms threaten death, the pulse becomes a little stronger, the passages are less frequent and less copious, and the respirations grow deeper. Bodily heat is gradually restored, and the patient recovers, unless some of the complications mentioned farther on ensue.

Variations from the Ordinary Course.—The patient may have so mild an infection as to be but slightly ill and never forced to go to bed. In other instances the serous diarrhœa is excessive, but the urine is not suppressed, and the general debility does not become marked. These cases are sometimes called cases of "cholerine." They may speedily recover or rapidly proceed to the fully developed malady. In still another class the toxæmia of the disease exceeds all other symptoms. The diarrhœa may be absent, and the patient, overwhelmed by the poison, sinks into unconsciousness and death. This is called " cholera sicca."

The degree of *stupor* varies greatly. In some patients the mind, at the well-developed stage of the disease, is remarkably clear; in other instances it is, almost from the first, stupid from toxæmia.

In some instances high fever develops. This is a very unfavorable sign. In others an urticaria or erythema is seen.

Complications and Sequelæ.—Aside from the grave complication of urinary suppression followed by uræmia, the profound infection may result in *localized gangrene* of the toes and fingers. *Œdema of the lungs* often causes death, and *infectious arthritis* and *parotitis* may develop. Profound weakness and feebleness may persist for a long time in convalescence, and *secondary nephritis* may ultimately cause death.

Diagnosis.—The diagnosis of cholera is not difficult if the well-developed type of the disease is present, but in the early stages, or in the aberrant forms just described, the determination of the cause of the illness may not be easy. True cholera is to be separated from cholera nostras or cholera morbus, but in the presence of an epidemic of Asiatic cholera this may be impossible without bacteriological tests, for severe cholera morbus may not only be manifested by purging and vomiting, but by collapse as well, and even cramps may appear in the more severe types. Cholera must also be separated during an epidemic from the profuse watery purging sometimes met with in cases of Bright's disease, when the purging is due to an effort at elimination. Various poisons may also cause choleraic diarrhœa, notably antimony. Indeed, it is impossible to separate acute antimonial poisoning from cholera during an epidemic of the latter disease, because the symptoms are identical. Nothing but a chemical analysis, on the one hand, or a bacteriological test, on the other, can determine this question.

It is important to remember that while the presence of the spirillum of cholera is characteristic of cholera, that inability to discover it in the discharges is not positive proof that cholera is not present, because in rare instances it may be temporarily undemonstrable. A very valuable method of diagnosis is the test of agglutination of cholera bacilli by the blood of the patient in a manner similar to that of the Widal test in typhoid fever.

Prognosis.—The prognosis in cholera, whenever the symptoms are well developed, is always grave, for the mortality in most epidemics is about 50 per cent. In the old and very young the outlook is worse than in a well-developed person in the prime of life.

There are three facts aside from the severity of the disease which increase the gravity of the prognosis very materially, namely, alcoholism, renal disease, and disease of the liver. In addition, it must be remembered that any pre-existing disease which decreases vital resistance increases the gravity of the case.

In respect to the disease itself, it may be said that abruptness of onset, early hebetude, and rapid development of signs of collapse are the three facts that promise evil tendencies. If to these is added renal inactivity, pulmonary consolidation, or an abnormally low temperature, the case is to be regarded as almost hopeless. Contrariwise, there are several signs of good omen, namely, the presence of a good pulse and the maintenance of bodily heat, the return of a fecal color to the stools, and the absence of the great emaciation and wasting which severe cases usually manifest.

Treatment.—The three most important details in the treatment of cholera are the control of the diarrhœa, the maintenance of strength, and the conservation of body heat. All persons who have any tendency to diarrhœa during a cholera epidemic should at once be treated by astringent mixtures, which should be increased in their efficiency by the addition of a few drops of sulphuric acid. By this method of treatment the development of cholera can be probably prevented in a considerable number of persons. The use of an astringent and acid substance like sulphuric acid is far more advantageous than the employment of opium, because the acid is destructive to the comma bacillus, and it does not interfere with other functions of the body as does opium. If the diarrhœa is already active 10 to 15 drops of aromatic sulphuric acid, with double that quantity of spirit of camphor, should be administered, well diluted with water or with brandy, every three hours, and counterirritation in the form of a mustard plaster or sinapisms or capsicum drafts should be applied over the abdomen. If these cannot be obtained a turpentine stupe may be used with advantage.

It seems scarcely necessary to add that even in mild cases the patient should be kept in bed and the greatest possible amount of rest enforced. If vomiting is an active symptom it may be necessary to avoid all medication by the mouth and give stimulants hypodermically. Under these circumstances a grain of camphor dissolved in sterilized olive oil may be given by means of the hypodermic needle every eight hours. Such a method of treatment will usually do much toward the maintenance of active circulation, but should the circulation fail the physician must employ not only the camphor injections named, but give brandy and strychnine hypodermically, and more important still, for the purpose of compensating for the loss of much fluid by the bowel, hypodermoclysis should be resorted to. It is best to employ "concentrated sterile saline," one ounce of which when added to a quart of pure water makes normal salt solution. But if this cannot be obtained ordinary common salt in the proportion of a drachm to the pint may be injected by hypodermoclysis. This fluid should of course be first sterilized by boiling, and the injection should be made slowly, the fluid being at the temperature of 100°. It is quite remarkable how rapidly the thirsty tissues will absorb this fluid, which not only compensates for the loss by purging, but also aids in overcoming suppression of urine by supplying the bloodvessels with fluid. There can be no doubt that hypodermoclysis is a most valuable life-saving measure in the treatment of this disease.

The body heat should be maintained by the application of hot bottles about the patient, particularly at his extremities, and when the temperature seems to be falling and the skin has a tendency to be relaxed and bedewed with sweat a hypodermic injection of $\frac{1}{150}$ to $\frac{1}{100}$ of a grain of atropine is advantageous. This injection should be into the trunk rather than into one of the extremities, since if the circulation is poor it will be absorbed more rapidly from the body than from an arm or the leg. Thirst is to be allayed by giving fluids to the patient in small quantities frequently, but large draughts of water are disadvantageous. Sometimes barley-water or soda-water is retained better than plain water. For the relief of the cramps gentle rubbing of the extremities with chloroform liniment, or ammonia liniment, may be employed, but the severity of this symptom is usually modified by the use of the camphor and the subcutaneous saline injections.

To aid in the restoration of renal activity a hot compress may be placed under the loins.

During the last outbreak of cholera in Europe salol was employed by a large number of physicians with excellent results, its employment being based upon the fact that if it is added to an alkaline solution of pancreatic juice it rapidly destroys the cholera bacillus. As an illustration of its value it may be stated that Hehir treated 88 cases with corrosive sublimate with a mortality of 44.7 per cent., and 11 cases with salol with no deaths; while Gonzalez lost only 3 out of 53 patients when he employed salol. Similar statistics from other parts of the world might be quoted indicating its very great value.

Under the leadership of Cantani the following treatment by injection has also become popular, it being necessary to give it in large quantities not only for the purpose of irrigating the large bowel, but if possible getting it into the small bowel as well; for it is in the small bowel that the micro-organism of the disease exists in largest number. The fluid employed by Cantani consists of infusion of chamomile flowers, 2000 parts; tannic acid, 10 parts; gum arabic, 30 parts; and laudanum, 2 parts. The object in using tannic acid is not only to obtain its astringent influence, but also because in the strength of 1 per cent. it inhibits the growth of intestinal micro-organisms, and, Cantani thinks, also neutralizes the poisons formed by these bodies. Lustig treated 117 cases of cholera in this way with 34 deaths and 193 cases by other methods with 146 deaths. Care must be employed in giving these injections that the fluid is introduced into the bowel slowly, the buttocks of the patient being slightly elevated and his body turned slightly to the left. Any tendency to bearing down on the part of the patient is to be avoided by diverting his attention from the injection and by momentarily stopping its flow when he feels the desire to expel the fluid. The fountain syringe containing the fluid should not be hung or held more than eighteen inches above the anus. If the fluid flows in under this gentle pressure, very large quantities may be retained, and Cantani asserts that it will find its way through the ileocæcal valve and flush the smaller bowel. Such a passage through this valve in the healthy intestine is, I believe, impossible, but it is stated that in the relaxation of the muscular fibres produced by cholera its passage is by no means difficult.

When collapse is threatened or present the fluid injected should be as hot as 105°, and the fluid in the bag may be at 110° or 115°, as it loses much heat in flowing slowly through the tube. If, on the other hand, hyperpyrexia is present, cool water should be employed.

As a rapidly acting diffusible stimulant in conditions of marked collapse Hoffmann's anodyne in the dose of a drachm every hour may ge given hypodermically or by the mouth with shaved ice. A drachm of spirit of camphor may also be used with advantage for this purpose. Aromatic spirit of ammonia may also be given by the mouth, but is not so valuable.

YELLOW FEVER.

Definition.—Yellow fever is an acute infectious disease occurring chiefly in tropical or semi-tropical regions, and characterized by fever, yellow discoloration of the skin, black vomit in some cases, and a tendency to oozing hemorrhages from the mucous membranes. The early development of albuminuria is also a noteworthy symptom.

History, Etiology, and Prevention.—The earliest history of yellow fever records its occurrence among the followers of Columbus, and before that time it never attacked Europeans. It is, therefore, a disease indigenous to the Western Hemisphere. As early as 1648 the inhabitants of St. Kitts and in 1655 those of Jamaica were attacked by it.

Since then yellow fever has devastated North and South America many times. It has extended its ravages all the way from Quebec to Montevideo, and on the western coast of the Western Hemisphere has been almost equally widely distributed. In the latter part of the eighteenth century it destroyed 10 per cent. of the population of Philadelphia. On more than one occasion it has brought military expeditions to defeat by the frightful mortality which it has caused amongst the troops. During the French expedition to Hayti, in 1802, 22,000 out of 25,000 men died from it in one season, and the various attempts which were made by Spain to subjugate Cuba were practically frustrated by the mortality from yellow fever among the Spanish troops. Davidson states that out of a population of 9000 persons at Gibraltar in 1800 only 28 escaped infection. In 1878 the financial loss in the Mississippi Valley produced by a single epidemic amounted to over $15,000,000.

For one hundred and fifty years Havana was recognized as the focus in which yellow fever was practically constantly present, and from this focus many portions of the civilized world were again and again infected. It was not until the United States Army took possession of Havana and its medical officers instituted sanitary measures that any real attempt was made to discover the means of propagation of yellow fever or to limit its development in that city. When the brave, skilful, and scientific labors of these officers were completed one of the most brilliant medical discoveries in the history of the world was announced.

Under proper sanitary directions the death rate in Havana fell from 91 and a fraction, under Spanish rule in 1898, to 33 and a fraction in 1899 under American rule, to 24½ in 1900, and to 22 and a fraction in 1901, but there was not a simultaneous diminution in the frequency or mortality of yellow fever. Indeed, at that period there was an actual increase in the disease notwithstanding the fact that all other maladies were decreasing. It was under these circumstances that a commission was appointed by the Surgeon-General of the United States Army for the purpose of studying yellow fever. The chairman of the commission was the late Dr. Walter

Reed, a major in the United States Army, and associated with him were acting assistant surgeons James Carroll, Jesse W. Lazear, and Aristides Agramonte.

The medical profession should never cease to do honor to the members of this commission, which faced one of the most horrible and fatal diseases with the greatest bravery, and thereby have succeeded in saving the lives of hundreds of thousands of individuals. Dr. Lazear, who was one of the most enthusiastic members of the commission, allowed himself to be bitten by an infected mosquito. He was not infected by this bite, but several days after he was accidentally bitten, and lost his life from the consequent attack of yellow fever. Another member, Dr. Carroll, allowed himself to be bitten, was also attacked by the disease, and narrowly escaped death.

The fact that Ross and others had proved that the transmission of malarial fever was by the mosquito, and that Dr. Carlos Finlay, a physician of Havana, a graduate of the Jefferson Medical College, of Philadelphia, had asserted as long ago as 1881 that certain species of mosquito in Havana was guilty of transmitting yellow fever from person to person, led the Army Board to direct their attention to the investigation of this question, and they soon found that if a female mosquito of the species *Stegomyia fasciata* were allowed to bite a yellow fever patient during the first three days of the disease, and then, from twelve to twenty days later, permitted to bite a non-immune, the latter almost invariably developed yellow fever.

That the disease is never carried by fomites was also proved by these investigators, who had a number of young non-immunes sleep for twenty consecutive nights in a room which was hung with articles soiled by black vomit, bloody fecal discharges, and urine, from fatal and other cases of yellow fever. These persons also packed and unpacked these articles night and morning from boxes in which they were placed. Other non-immunes actually slept in garments and between sheets that had covered fatal cases of yellow fever, but in not a single instance was the disease contracted, although as soon as these non-immunes were exposed to mosquitoes several of them developed yellow fever.

The practical result of proving that the mosquito is the cause of the transmission of the infection has been the complete clearance of Havana of yellow fever. All cases of yellow fever were protected by mosquito netting so that mosquitoes could not carry infection from them to others. All pools and gutters containing water upon which mosquitoes could breed were removed, and the destruction of mosquitoes was carried on actively, with the result that it was possible in a year to diminish the number of deposits of mosquito larvæ in the city of Havana from 26,000 to 300. As a result, the death rate from malaria fell from 344 in 1900 to 151 in 1901, and up to July, 1902, it was only 47; while the diminution in the number of mosquitoes caused so great a decline in the prevalence of yellow fever that by September 28, 1901, new cases ceased to occur in Havana. Since that time, according to Dr. Gorgas, of the United States Army, from whose reports much of this information is taken, not a single case has originated in that city, until the latter part of 1905, when relaxed vigilance allowed the disease again to reappear.

The conclusions of the commission are so important that they are given verbatim:

1. The mosquito—*Stegomyia fasciata*—serves as the intermediate host for the parasite of yellow fever.

2. Yellow fever is transmitted to the non-immune individual by means of the bite of the mosquito that has previously fed on the blood of those sick with this disease.

3. An interval of about twelve days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection.

4. The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack.

5. Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of this disease.

6. An attack of yellow fever, produced by the bite of the mosquito confers immunity against a subsequent attack of the non-experimental form of this disease.

7. The period of incubation in thirteen cases of experimental yellow fever has varied from forty-one hours to five days and seventeen hours.

8. Yellow fever is not conveyed by fomites, and hence disinfection of clothing, bedding, or merchandise, supposedly contaminated by contact with those sick with this disease, is unnecessary.

9. A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitoes capable of conveying the parasite of this disease.

10. The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitoes, and the protection of the sick against the bites of these insects.

11. While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered.

Prophylaxis.—Attention has already been called to the fact that the infectious agent of yellow fever is conveyed by mosquitoes from patient to patient. It is therefore essential that all cases of yellow fever should be kept under mosquito netting so that they may not be bitten by mosquitoes, and it is also wise for those who are well to protect themselves at night from mosquitoes by similar means. For screening those who are ill, a gauze of not less than twenty meshes to the inch should be used, otherwise the mosquito may pass through it. An active crusade against all mosquitoes and the destruction of their breeding-places should also be instituted.

Pathology and Morbid Anatomy.—One of the most marked changes produced in the body by the infection of yellow fever is that which takes place in the blood. Many of the red cells are crenated and some of the white cells are granular. Free hæmoglobin, hæmin, and hæmatin are found in it owing to the destruction of the red corpuscles.

The heart is soft and flabby, and minute ecchymoses in its muscular tissue may be present. The pericardium may contain an excess of blood-stained fluid, and its membrane may be dotted with petechiæ.

The stomach shows changes with great constancy. It usually contains black fluid due to altered exuded blood; its mucous lining is congested in patches and is marked by ecchymosis or even softened. When placed under the microscope sections of the stomach show the bloodvessels engorged and their walls undergoing fatty degeneration. The intestinal canal also contains broken-down blood passed from the stomach, and its contents may be acid. Fatty degeneration of Peyer's patches and the glands of Lieberkühn is present.

The liver is often pallid or yellow in hue, and its cells also may undergo fatty change. Councilman states that associated with these signs of fatty degeneration areas of necrosis can be demonstrated in every case that comes to autopsy.

As in many acute and severe infections, the kidneys show signs of acute diffuse nephritis with fatty degeneration of the cells lining the tubules.

Small hemorrhagic spots are sometimes found in the meninges of the brain and cord, and fatty degeneration of the cells of the solar plexus has been described.

Symptoms.—A very noteworthy fact in connection with the symptomatology of yellow fever is that in a majority of cases its onset is *most abrupt.* There may be, for a few hours before the well-defined symptoms show themselves, a sense of *malaise* and *headache* or *vertigo.* The first symptom of prominence is the appearance of a *rigor*, or rigors, which may be moderate or severe, but Bemiss states that chills are rare. In addition the patient suffers from *severe lumbar* and *muscular pains*, *headache* and *eyeache*, and *marked pallor.* There is often *epigastric distress.* In children the disease may be ushered in by *convulsions.*

After the stage of onset the skin of the face becomes flushed and turgid; the mind may wander, but as the disease develops it is usually remarkably clear and alert, so that the patient watches those about him with the same degree of attention as is often seen in acute peritonitis. The expression is anxious. The *temperature* rapidly rises so that it reaches its acme of from 103° to 107° by the end of twenty-four or thirty-six hours.

If the case is a very mild one the febrile movement may cease as early as the end of the first day or on the morning of the second day, but usually the acme of the temperature is maintained for from two to three days, during which time there may be slight morning and evening variations. In cases which are moderately severe the fever usually begins to fall after this time and reaches a point near the normal in from twenty-four to seventy-two hours. That is, the fall is by lysis.

After the temperature has reached normal, that is when the *stage of remission* about to be described has become well marked, a *secondary fever* develops which, like that of the stage of onset, lasts from two to three days and falls by lysis. In cases which are likely to be fatal this fall may not occur.

The *respiration* and *pulse rate* are at first increased in frequency and the individual pulse beat is increased in volume, but these circulatory conditions speedily undergo a marked change with the approach of the period of remission. On the second or third day, even if the temperature remains as high as before, the pulse rate begins to fall, or falls even while the temperature is still rising, so that a pulse rate which early in the onset was as high as 110 may now be as low as 75.

After the fever begins to fall owing to the beginning of convalescence the pulse, as in many cases of ordinary catarrhal jaundice, may fall still farther till it reaches 45 a minute. It is the slowing of the pulse in the stage of onset, while the temperature is still high, that is particularly worthy of note.

The *tongue* is covered with a white fur save at its edges, which are red; the *bowels are constipated*, and there may be epigastric distress followed by the vomiting of acid mucus.

The *urine* is scanty and it may be distinctly albuminous as early as the third day. This *early albuminuria* is considered by yellow-fever experts to be a very important aid to diagnosis.

By the third day a very *marked remission* in the symptoms may occur. The pains and aches, the rapid pulse, the high temperature, and the flushing of the face all become modified. The gastric symptoms abate, but the conjunctivæ may begin to be jaundiced and the skin of the body may also begin to show a yellow hue. This is the critical period of the disease, for the patient is now "at the parting of the ways." One path leads to rapid recovery with marked amelioration of all the symptoms; the other leads, after a remission of from twelve to forty-eight hours, to a recurrence of all the dangerous symptoms in an aggravated form.

If the way is evil there is *præcordial* and *epigastric distress*, persistent *vomiting* of clear liquid with flakes of *brown reddish-looking* material, which speedily increases in amount until the well-known *black vomit* presents itself. The *urine is more scanty* and *more albuminous* than ever, and the general state of the patient is that of profound illness. The *temperature* in some cases *rises* as it did in the stage of onset, but it may, and this sign is of grave import, fall below normal. Even yet it is possible for recovery to occur by a gradual amelioration of all the symptoms, but usually the symptoms continually get worse. The *grave depression* increases, the yellow skin develops a greater degree of yellowness, and petechiæ are formed. The vomiting of black material is more severe and profuse, and hemorrhages may occur from other mucous membranes than that of the stomach. The scene closes with *hiccough*, *profound asthenia*, *subsultus tendinum*, the *Hippocratic face*, and in *exhaustion* and *coma*, due in part to the direct effect of the infection and in part to the uræmia produced by the intense renal lesions.

While these may be considered the symptoms of yellow fever in many cases, in others they are very different. In the so-called *apoplectiform type* the patient is seized with *vertigo*, *stupor*, *unconsciousness*, and *convulsive attacks*. He speedily becomes more and more deeply depressed, his circulation fails, the bowels and bladder are involuntarily emptied, and with the development of *multiple hemorrhagic extravasations* he dies.

In another severe type the symptoms are *algid*, the patient speedily passing into profound collapse with a subnormal temperature and profuse hemorrhages, death coming on in a few hours. In still another type the violent *vomiting*, *purging*, and *collapse* may cause the case to resemble one of *cholera*.

Diagnosis.—It is stated by all physicians of experience that in some cases it is almost impossible to make a diagnosis of yellow fever in its early stages, chiefly because it has few pathognomonic signs, and these are of value only when associated and not when they appear singly. Again, many cases of yellow fever pursue a very aberrant course, so that several days elapse before the diagnosis can be made.

Yellow fever must be separated from dengue, pernicious malarial fever, from malarial hæmoglobinuric fever, and from relapsing fever. The differentiation of yellow fever from dengue has given rise to much bitter controversy, and even at the present time physicians of wide experience with both maladies are by no means agreed about the separation of these diseases in their early stages.

Guitéras asserts that there are *three notable symptoms* of yellow fever which are of service in this connection. First, the *facial expression* of the yellow-fever patient is characteristic because in no other disease is it so flushed, the eyes so injected, nor the conjunctiva so icteroid after a few hours of illness. Second, the development of *albuminuria* as early as from the first to the third day, which may be transient and slight, or persistent and profuse. The third differential point is the *change in the pulse* already noted as occurring on the second or third day of the disease during the continuance of fever.

The jaundice of dengue rarely appears as early as the third day.

The history of the patient as to exposure, the presence of the æstivo-autumnal parasite in the blood, and the enlarged spleen of malarial infection point to pernicious malarial fever. A porter-colored urine, the blood infection, and the enlarged liver point to hæmoglobinuric fever, while the discovery of the spirillum of Obermeier in the blood will demonstrate the presence of relapsing fever. (See Relapsing Fever.) While all these facts may aid greatly in distinguishing yellow fever, it is not to be forgotten that the absence of some of them does not prove that the yellow fever is not present. Thus in some cases the albuminuria does not appear very early, in others the failure to discover the æstivo-autumnal parasite may be due to the lack of skill of the observer or to the well-known difficulty of its discovery even by the most practised observers. Again, it is possible for the malarial parasite to be present when yellow fever is present, the two diseases existing simultaneously.

Prognosis and Mortality.—In a disease which is so variable in its manifestations, prognosis must always be guarded. If the febrile, gastric, and renal symptoms are mild in the stage of onset, the outlook is more favorable than if they are severe. If the period of remission is not well marked and hemorrhagic tendencies are well developed, the prognosis is bad.

The mortality varies very greatly in different epidemics, as already shown in the discussion of the history of the disease. Sometimes it is as low as 15 per cent., again as high as 85 per cent. It is apt to be lower in private than in hospital practice. Some authors have made the interesting statement that the mortality is in inverse ratio to the morbidity. The average mortality may be stated at about 30 per cent. Thus, in 25,220 cases of yellow fever occurring in the West Indies, Central and South America, Mexico, and the United States, 8020 cases were fatal, a percentage of 31.8.

Treatment.—In the treatment of yellow fever it is essential that the patient shall have a plentiful supply of fresh air and sunshine, with absolute rest and proper sanitary surroundings. Bad hygienic surroundings always greatly increase the mortality of the disease.

As soon as the patient is suspected to be suffering from yellow lever, he should be put to bed and required to remain there until convalescence has been completed, for physical and mental unrest distinctly predispose the patient to a fatal issue. During the whole period of the disease the patient should not be allowed to sit up in bed, since sudden cardiac failure may occur. All the food and medication should be given to the patient when in the recumbent position, and the contents of the bowels and bladder emptied into a bed-pan. The patient should be lightly covered, and the use of heavy blankets or quilts should be discouraged.

Cleanliness of the mouth should be carefully maintained, since otherwise softening and ulceration of the gums not infrequently occur.

Active medication for the treatment of the disease itself is unwise. The physician should give only remedies when they are very distinctly indicated, as for the relief of a failing heart, with the hope of increasing the activity of the kidneys and for the prevention of profound asthenia. In some portions of the world where yellow fever frequently occurs, it is customary to employ hot mustard foot-baths and even hot packs during the early stages of the disease, but they are unwise after the malady is once well developed. For the relief of the fever cool sponging with alcohol and water, or even with ice-water, may be employed, an ice-bag being applied to the head. The employment of the coal-tar products is never advisable, and they are particularly contraindicated when the depression is marked. Many practitioners have employed emetics in the early stages of yellow fever, but these are certainly not required unless it is known that the patient's stomach is overloaded with food, when 10 to 20 grains of ipecac may be given.

Many years ago former Surgeon-General Sternberg advised the employment of bicarbonate of soda, corrosive sublimate, and water in the treatment of yellow fever, but, although this method of treatment has been widely employed, it has now largely gone out of use, although large quantities of bicarbonate of soda are given freely by many practitioners as a matter of routine.

As in most infectious diseases, the bowels, if constipated, should be moved by means of calomel, which in turn may be followed by one of the saline purges or by castor oil. Purgation may be resorted to every twenty-four or forty-eight hours, in order to keep the bowels thoroughly evacuated. To aid the purgatives and for the purpose of washing toxic materials from the large intestines, copious irrigations of the colon with normal salt solution are advisable. The patient should be urged to drink freely of water in order to flush the kidneys, and alkaline mineral waters, such as Vichy, Apollinaris, or Seltzer, or plain water, to which bicarbonate of soda has been added in small quantities, may be used with advantage to neutralize the acidity of the gastric contents and to act as diuretics.

For the arrest of excessive vomiting, cocaine has been highly recommended, but there is no reason to believe that it exercises any better anti-emetic properties in this disease than in other diseases in which vomiting is present. One or 2 minims of creosote or carbolic acid are equally valuable.

For the purpose of stimulating the heart and circulatory system, digitalis in the dose of 5 minims of the tincture, or strychnine in the dose of $\frac{1}{40}$ of a grain, or caffeine in the dose of 1 or 2 grains, may be given three or four times a day, and if collapse is threatened the strychnine may be given hypodermically with atropine, or Hoffmann's anodyne may be given by the mouth or by the hypodermic needle. Strong, black coffee may also be employed by the mouth or by the rectum, for the purpose of rallying the patient.

For persistent hiccough, sipping very hot water is often advantageous.

From the beginning to the end of the attack it is the duty of the physician to carefully watch the condition of the kidneys by making daily examinations of the urine, since uræmia is one of the greatest dangers which can beset the patient. After the kidneys have once become so inactive that the urine is exceedingly scanty it is often absolutely impossible to stimulate them to activity, whereas much can be done, if from the very first, renal activity is maintained. For this purpose calomel may be given as a diuretic in the dose of 2 or 3 grains every few hours for one or two days at a time, or one of the diuretic potassium salts, such as the citrate or acetate, in large amounts of water. Hypodermoclysis with normal salt solution may be employed. Renal congestion may be overcome by the application of mustard plasters and dry cups over the kidneys.

During the acute stage of the illness the condition of the stomach is such that the administration of food is almost impossible, but milk diluted one-half with Vichy water or with water containing bicarbonate of sodium may be given.

During convalescence the greatest possible care must be exercised that the patient does not take food in too large quantities. No solid food should be permitted before the end of a week, and, if the patient has been very ill, not for two weeks. In the mean time the diet should consist of partially peptonized milk, milk-toast, broths, and eggs.

As in all exhausting diseases, the physician must insist upon the patient remaining in bed until the heart muscle has entirely recovered from the profound depression of the disease. Bitter tonics, such as iron, quinine, and strychnine, may be given.

PLAGUE (BUBONIC PLAGUE).

Definition.—Plague is an acute, specific, infectious, and contagious disease caused by the *Bacillus pestis*. It occurs in widespread epidemics, is characterized by fever, inflammation of various glandular groups, and profound depression. The course of the disease is exceedingly rapid and the mortality extremely high.

History and Distribution.—In ancient times plague occurred in pandemics, spreading over the whole known world. Most of the old world epidemics about the beginning of the Christian era have been described as plague on wholly insufficient evidence. Hirsch dates the first recognizable epidemic in the second century B.C. Following this, historical descriptions do not satisfactorily identify the disease until the pandemic which persisted for nearly sixty years, during which time it ravaged the whole of Europe (A.D. 542). Following this epidemic, known in history as the plague of Justinian, the disease appeared from time to time, but only twice to so great a degree. The first of these two extensions was during the fourteenth century; the second, known as the Great Plague of London, began in 1664 and lasted until 1679. During the first year of this epidemic one-sixth of the total population of London perished. The advance of sanitary science since that time has gradually forced plague out of Europe and limited the area of its extension. During the last three decades it has lingered in Southeastern Europe.

We are now in the presence of what must be considered a world-wide extension of the disease, limited only by effective preventive measures. The present epidemic began in Hong Kong in 1894. In 1896 it reached the Presidency of Bombay, and, in the ten years following, it has spread through nine British provinces and fifty-one native States, the cases increasing in spite of all restrictive efforts. It is estimated that to the middle of the year 1903 two million persons have perished in the Deccan since the beginning of the outbreak. In the city of Bombay over one hundred thousand persons have died, and in the Presidency of Bombay alone during January, 1903, the deaths averaged eight thousand weekly. Later, the disease appeared in Japan, Madagascar, and South Africa. It obtained a temporary foothold in Glasgow, Lisbon, and Oporto. In 1900 it reached Sydney, Australia. On the American continent it appeared in Brazil and the Argentine Republic. In 1899 it established a foothold in San Francisco, in which city rigorous measures have limited it to a very great degree. During the year 1902 the disease appeared in Peru, Mexico, and Alaska.

Etiology.—Plague is caused by a specific micro-organism discovered by Kitasato and Yersin in 1894, during the epidemic in Hong Kong. This organism is a short, oval, non-motile, coccobacillus resembling the bacillus of chicken cholera. It occurs singly, joined in pairs, and occasionally in long chains. It is found in large numbers in the pus of plague buboes and in smaller numbers in the viscera and blood. It has been cultivated from all the accessible tissues of the body during life, and from all the excretions except the sweat. It has also been recovered from the floors and soil in the houses of patients sick with plague.

The bacillus stains with all the ordinary staining reagents and is decolorized by Gram's method. It takes up the stain much more strongly at its poles. Sometimes a capsule is observed, but there is no spore formation. It grows best at the body temperature and on all the ordinary media. In fluid culture media, overlaid with a film of bland cocoanut oil, the bacillus grows in the form of long stalactites hanging from this oily layer that are considered characteristic of this organism. Its viability is rather low. If kept moist and cool, it may keep alive and virulent for months, but if dried at the room or body temperature it dies in from three to four days. Exposure to direct sunlight destroys it in a few hours. The bacillus is pathogenic for nearly all domestic animals. Indeed, most of them are subject to plague and aid in its dissemination. Sheep, calves, pigs, ducks, and fowls readily contract the disease, and the bacilli may be recovered from their viscera and excretions. The disease also occurs in bats, and the common rat is peculiarly susceptible to it. The great mortality among rats, preceding and often signalizing an epidemic of plague, is an observation that was well known to the ancients.

The Chinese long ago recognized the association between the death of the rats in a house and the development of plague a few days later. Finally, the fleas that infest rats and the flies in infected houses also serve to carry the contagion.

The method of conveyance to man has been established with a fair degree of certainty. Inoculation experiments in man and animals have shown that when virulent bacilli are introduced into the tissues plague develops. Thus at Cairo in 1835 plague blood was used to inoculate two criminals, but though they developed the disease both recovered. The list, too, of physicians and laboratory workers, who have contracted the disease from accidental inoculation and dissection wounds, is a large one. Among them may be mentioned Whyte, who, in 1802, infected himself and perished, and Aoyama and his assistants who contracted plague from dissection wounds.

In 1898 three deaths occurred in Vienna as a result of laboratory inoculations, one in 1899 in Lisbon, and one in 1902 in Berlin, while thirteen cases resulting from accidental inoculations in postmortem examinations have been collected in India.

For a time it was believed that man was infected by inhalation of the germ, by swallowing it on infected food, and by direct inoculation. That infection takes place through air and food has not been substantiated. The disease enters through the skin, by direct infection through slight wounds or abrasions, or through the bite of suctorial insects. It has been shown that rats suffer more intensely perhaps than any other animal from plague. The rats are infested with fleas, which are also infected, and the disease is transmitted from rat to rat and finally from rat to man by the bite of these insects. There has been some conflict of opinion as to whether the fleas of plague-infected rats would bite man. To settle this question Tidswell examined the fleas from a number of rats and found five varieties, of which four were known to attack man.

The possibility of infection taking place directly into wounds and abrasions from infected soil must be admitted. Calvert reports an interesting case where the disease was acquired in sexual intercourse, and one case where the bite of an infected rat caused a fatal infection. Direct transmission from patient to patient, while always possible, occurs very rarely. This is borne out by the observations in Bombay, Hong Kong, and other places, that cases are extremely rare among the physicians, nurses, quarantine guards, and disinfection laborers, who are constantly in intimate contact with plague cases.

The disease principally attacks the poorer classes of the native population, those who live in the slums under poor hygienic surroundings. Lack of personal cleanliness and deficient light and ventilation in living rooms are predisposing causes.

Frequency.—Plague affects all ages and both sexes equally. Neither geographical location, character of soil, nor elevation have any influence on its spread. It prevails at all seasons of the year, although, generally speaking it is least active in the seasons of greatest heat or cold.

Epidemics begin slowly. The common history is that in the beginning a few isolated cases develop, the epidemic slumbering along in this way for a year or more before rapid extension takes place. It declines in the same way. Not only do the number of cases grow less, but their virulence notably diminishes. It creeps slowly from town to town, following the routes of travel. Its extension from one country to another over sea is due to the presence of infected rats on the ships plying between them. Thus the epidemic in Peru was shown to have spread from the rats on a ship carrying grain from India.

Symptoms.—Clinically plague may be divided into four varieties:

1. Bubonic plague, pestis bubonica, malignant adenitis.
2. Septicæmic plague, pestis siderans.
3. Pneumonic plague.
4. Larval plague, pestis minor, pestis ambulans.

BUBONIC PLAGUE.—This is by far the commonest type, averaging 80 per cent. of all cases. The incubation period varies from two to eight days, averaging four days.

The attack begins with *fever*, *lassitude*, *severe headache*, and *pain in the limbs*. Rigors may or may not be present, but vomiting is usual in this stage. There is *drowsiness*, *vertigo*, and *extreme anxiety*. After lasting from twelve to twenty-four hours, fever begins and the temperature rises rather quickly to 103° to 107°. There is now hurried pulse and respiration. The face is heavy, swollen, and flushed; the tongue is coated with a heavy black fur; the teeth are covered with sordes. *Vomiting* is often persistent and diarrhœa may develop. The patient is most profoundly depressed, the depression being out of all proportion to the duration of the disease, and a low muttering delirium is present. Death may occur in this stage, accompanied by *convulsions* and *collapse* or by *uræmic coma* with total suppression of urine.

In from twenty-four to seventy hours—that is, from the third to the fifth day of the disease—the characteristic *glandular swellings* develop. The glands involved are in the groin in 60 per cent. of the cases, the axilla in 35 per cent., and the neck and angle of the jaw in 5 per cent. The buboes are usually single and are much more common on the right side than on the left. Occasionally they are bilateral, rarely multiple. In size they vary from a pigeon's egg to the size of a fist. They are frequently painful and always exquisitely tender.

Coincident with the development of the buboes, small areas of *gangrene of the skin*, *carbuncles*, or generalized *pustular skin lesions* may develop.

The *buboes* increase in size for three or four days and then become stationary. In a small proportion of cases gradual resolution takes place. In the larger proportion softening and suppuration occur and the bubo is opened or ruptured and discharges a foul-smelling pus. At this stage free suppuration is usually a good omen. If the pus continues scanty and sanious the disease remains virulent.

In free suppuration the bacilli disappear from the pus in a very few days and convalescence is rapidly established. In the cases that terminate favorably a marked amelioration is observed with the development of the glandular swelling, and usually about the seventh day the temperature falls and the profound depression disappears.

SEPTICÆMIC PLAGUE.—In this form the symptoms are much more severe and the stage of bubo formation is lacking. That is, there is no one gland or group of glands conspicuously involved, but the *whole glandular system* is *engorged* and *swollen.* The essential difference seems to lie in that the infection is more severe either quantitatively or qualitatively. There is a marked bacteriæmia. Clinically, these cases differ from the former in the more profound depression, more moderate fever (100° to 102°), and the greater *tendency to hemorrhages.*

PNEUMONIC PLAGUE.—This form begins suddenly with rigors and all the symptoms of *acute pulmonary inflammation. Respiration is rapid* and *labored* and there is a *painful harassing cough.* So far the symptoms resemble an ordinary lobar pneumonia. The *sputum,* instead of being scanty, tenacious, and of the usual prune-juice color, is copious, watery, and spotted and streaked with bright blood. Physical examination shows areas of consolidation scattered throughout the lungs. An entire lobe is rarely involved. This form of the disease is the most fatal of all, patients rarely surviving after the third day. In these cases, too, although it is not clinically apparent, postmortem examinations show general involvement of the glandular system. Pneumonic plague is more common in children than in adults, and at the beginning of epidemics than at the end.

Hemorrhages occur in all the various clinical types of plague, more commonly perhaps in the septicæmic. They appear in the skin as *petechiæ* and *ecchymoses.* There may be *epistaxis, hæmaturia,* and *hemorrhage* from the *stomach* or *bowel.* Hæmoptysis is a very sinister symptom.

The urine is diminished and commonly contains large quantities of albumin with more or less kidney structure. *Albuminuria* is never absent in severe or fatal cases.

The *blood changes* are not characteristic. There is a marked leukocytosis, varying from 20,000 to 50,000, with moderate reduction of the hæmoglobin.

Relapses occur in a small percentage of cases and are always grave. Convalescence may be very much prolonged by indolent ulcers and burrowing sinuses at the seat of the buboes.

LARVAL PLAGUE, PESTIS MINOR.—Cases of this type occur in all epidemics and are very common toward their close. In larval plague the *typical buboes* develop with few prodromata. The constitutional reaction may be very mild, the fever is slight, and the patient is but little annoyed by the disease. Some epidemics are characterized by large proportions of such cases.

Prophylaxis.—Personal prophylaxis should be directed in the first place to avoiding too close contact with plague cases. Nurses and physicians should remain as short a time as possible in their immediate vicinity. Wounds, abrasions, and skin eruptions on the limbs should be carefully guarded, particularly against a germ-carrying finger-nail. Tight leggings or gaiters should be worn to prevent the bite of fleas. These measures combined with personal cleanliness, a good water supply, and abundant ventilation are efficient.

The general measures to be taken for the prevention of plague are, first, strict attention to sewage, water, ventilation, and cleanliness of dwellings; second, the extermination of rats and mice. This has been tried on a large scale by trapping and poisoning. Quarantine has never been an effective check to this disease.

The danger from contact is not very great. Exposed persons should be disinfected, given a prophylactic inoculation, have their clothing destroyed, and then be released. The quarters in which a plague patient has lain should be thoroughly scraped, disinfected, and repainted or whitewashed. Better still, when practical, they should be burned. The evacuations and bedding of the sick should be cremated.

In spite of the most stringent prophylactic measures, plague is very difficult to control. As a matter of fact, where it has once attained even a slight foothold it has not been successfully eradicated by any of these measures. Witness the cases in San Francisco, where after several years of effort the disease still persisted. It seems likely that the most we can expect with our present means is to hold the disease in check.

PROTECTIVE INOCULATION.—Haffkine introduced a prophylactic inoculation against plague. His method has been modified by Lustig and recently by Besredky. Briefly, these methods consist in injection of plague cultures killed by heat. Extensive experience has shown that these inoculations confer an immunity against plague, beginning in twenty-four hours and lasting from three to four months. Recent studies seem to show that, given during the period of incubation, they have the power to abort the disease in many cases. This system of protective inoculation was being tried on an extensive scale in the Punjab, when a very deplorable accident cut the experiment short. After more than 100,000 persons had been inoculated without untoward results, nineteen men received their injection from the same package, developed tetanus on the fifth day, and all died. This unfortunate affair practically stopped prophylactic work in India by greatly increasing the aversion the natives had always shown to it.

Pathological Anatomy. — The visceral lesions of plague are constant and uniform. Punctate hemorrhages appear not only on the skin, but throughout the whole gastrointestinal tract. They are found on the peritoneum, pleura, and pericardium, as well as in the capsules of the spleen, kidney, and liver. The cerebrospinal system is congested and there is an increase of its fluid. The liver and kidneys are hyperæmic and the spleen very much enlarged. In pneumonic cases the bronchi are injected and swollen and there are small areas of consolidation scattered throughout the lung. The pleural cavities frequently contain moderate quantities of seropus.

The glandular system shows constant involvement. In the bubonic form the glands appear on section as large, diffused masses, with extensive hemorrhages into their substance. This appearance is not confined to one group of glands, but extends along the lymphatic trunk and invades the glands in the immediate proximity to the main buboes.

Microscopically, intense hyperæmia with hyperplasia is found not only in the glandular but also in the periglandular structure. Before the glands break down the bacillus pestis is found alone; after suppuration is established other organisms are found with it.

In the septicæmic and pneumonic cases, or in those cases dying before marked bubo formation has taken place, the gross changes in the lymphatic system are not so apparent, but there is always enlargement of one or more groups of glands or slight tumefaction and congestion of the entire lymphatic system. The pathological process is identical in all the types, only that in the bubonic form the intensity of the affection is expended on one gland or group of glands, while in the other form the adenitis and lymphangitis are diffuse.

Diagnosis.—In the presence of plague in epidemic form the, rapid onset of the disease, the profound depression, the glandular swelling can hardly suggest anything else than this disease. The identification of the *bacillus pestis* in the blood, in fluid from the buboes, or in the sputum assures the diagnosis. Inoculations and culture experiments are important in the early stages of an epidemic with large numbers of atypical cases of plague. The best routine method of diagnosis is the microscopic examination of a drop or two of the fluid obtained from the buboes by means of a hypodermic syringe. The few drops of bloody lymph collected in this manner contain large numbers of bacilli. The diagnosis of the pneumonic form can only be made by demonstrating the micro-organism in the sputum.

Prognosis.—Varying in different epidemics, the average mortality runs from 70 per cent. to 95 per cent. The variations depend on the stage of the epidemic, the proportion of pestis minor cases, and the race and hygienic conditions of the patients. In the Hong Kong epidemic the average mortality was 93 per cent. among the Chinese, 77 per cent. among the Indians, 60 per cent. among the Japanese, and 18 per cent. among the Europeans. This gradation, as Manson has remarked, is "in general correspondence with the social and hygienic conditions with these different nationalities."

The influence of the type of the disease on mortality is shown in the following figures from an analysis of 13,145 cases. In the bubonic cases the mortality was 77.25 per cent., in the pneumonic cases 96.69 per cent., and in the septicæmic cases 89.62 per cent.

The number of the buboes and their location has no bearing on the mortality. Visceral hemorrhages are always unfavorable symptoms, while free suppuration of the buboes must be considered as a very favorable omen.

Pregnancy complicating plague is also very unfavorable. Abortion invariably occurs and death is almost certain.

Treatment.—Treatment of plague is wholly symptomatic. For the fever, headache, and delirium nothing is so effective as cold sponging. Cantlie recommends initial purging with calomel in large doses, followed by salines. This remedy frequently checks vomiting and permits nourishment to be taken.

For the pain and restlessness there is no remedy so effective as morphine, given hypodermically, in small doses. In the profound depression and collapse, diffusible stimulants are indicated; ammonia to the nose, mustard to the skin, and ether or camphor subcutaneously. Alcohol should be given freely, particularly in a septicæmic form.

Suppuration of the buboes should be hastened by poultices and hot fomentations. When fluctuation occurs they should be opened freely and dressed antiseptically.

Thomson reports excellent results in the epidemic in Hong Kong from the internal use of carbolic acid in large doses. He gave 144 grains daily in doses of 12 grains every two hours in a mixture of syrup of orange and chloroform-water. One patient took over 2500 grains of pure carbolic acid before his blood was free from plague bacilli. Beyond a few cases of carboluria no toxic symptoms developed. He considers this the most hopeful method at our disposal.

Yersin, Calmette, and Borrell have developed an antitoxic serum by the injection of ascending doses of cultures killed by heat into susceptible animals. Experimentally, plague in animals has been arrested by this means. Clinically the results with the antitoxic sera have been most contradictory. While they have not entirely fulfilled the hope that they first seemed to hold out, later experience in this direction is more encouraging. The antitoxin needs further study, and particularly needs standardization.

CLIMATIC BUBO.

Definition.—Climatic bubo, *tropical bubo, tropical adenitis* (non-venereal), is a subacute inflammation of the lymphatic glands of the groin, attended by a fever remitting in type and persisting from three to four weeks. The disease is widely distributed in tropical climates. It occurs on the coast of Africa and Asia, and is common enough in the Philippines, Japan, Malaya, the West Indies, and the Mediterranean.

The disease commonly affects individuals living together under the same hygienic conditions, as sailors and soldiers, and occurs in small epidemic outbreaks. There is some evidence to show that its origin is due to the entrance of bacterial infection, either through minute wounds in the legs and genitals or the bites of insects. It has been described as due to tropical heat and to paludism, as a sequel to dysentery, and even as a form of bubonic plague (pestis minor). Bacterial evidence disposes of the last theory, but in the presence of epidemic plague these cases demand careful study.

Symptoms.—They begin with moderate *swelling, redness,* and *tenderness* of the *inguinal* or *crural glands* of one or both sides. At the outset there is usually a *chill, fever* of a remitting type, *headache,* and *backache.* The *buboes* slowly increase until they attain the average size of a hen's egg, after which the fever gradually diminishes. After persisting from one to two months or longer they gradually disappear. In the large majority of cases the inflammation is limited to the gland structure proper. The periglandular tissues and skin are not involved and there is very little pain or tenderness. In from 3 per cent. to 5 per cent. of all cases the inflammation spreads to the periglandular tissues. The skin becomes adherent over the glands and they finally suppurate. In these cases the constitutional symptoms are intensified and the pain and tenderness are very great. The abscesses tend to burrow freely. After a period of free suppuration deep, sharp-edged, indolent, painful ulcers remain. The average duration of the suppurating cases is from two to three months.

Treatment.—The febrile condition is not severe enough to demand special treatment. Iodine and ichthyol may be applied to the skin over the gland, and, after acute symptoms subside, mercurial ointment and elastic pressure should be used. When suppuration takes place the gland must be laid open. Rife advises calomel as a dusting-powder to control the severe pain in the chronic ulcers.

DYSENTERY.

Definition.—Dysentery is a condition characterized by diarrhœa, abdominal pain, and the presence, as a rule, of considerable quantities of mucus in the stools. When the condition becomes chronic it is often interrupted by periods in which constipation supplants the diarrhœa. Dysentery is to be separated from the diarrhœa due to indigestion and to catarrh of the small bowel by the facts that tenesmus is usually marked, the lesions are, primarily at least, in the large bowel, and the stools are, in the early part of the attack, rather scanty and consist of mucus and blood.

It is, moreover, to be distinctly understood that dysentery is not a single disease, but that this term is applied to the conditions and symptoms which develop as the result of several distinct causes, although at present there is much confusion as to the causes of the various forms. Strictly speaking, amœbic dysentery should be classed among the diseases due to animal parasites, but it is best, from the clinical standpoint, to discuss it here.

At the present time at least four well-defined types of dysentery are recognized, namely, that which is known as *bacillary dysentery*, which is due to infection with the specific bacillus of Shiga, or a bacillus nearly related to it. Second, *amœbic dysentery, intestinal amœbiasis* (Musgrove and Clegg), which is due to the *Amœba dysenteriæ.* This form is found in all parts of the world, but is much more frequent in the tropics, from whence most of the cases seen in this country come. Strong, of the United States army, reports 561 cases of amœbic dysentery out of 1328 cases of dysentery in his service. Third, *catarrhal dysentery*, which is apparently not due to a definite infection, but to acute congestion of the mucous membrane of the colon; and finally, fourth, *diphtheritic dysentery*, which is *not* due to the Klebs-Loeffler bacillus, but is characterized by a yellowish exudate on the mucous folds of the bowel with areas of ulceration and necrosis. A form of catarrhal dysentery sometimes also develops as the result of renal disease.

History and Etiology.—Epidemics of dysentery have occurred since the earliest times, and Herodotus mentions one which attacked the army of Xerxes in the year 480 B.C. During the first part of the Christian era the disease raged in France, Germany, and England. It has existed in Europe in pandemic form on at least two occasions, namely, in the years 1538 and 1779.

In the year 1729 an epidemic, in which 5000 persons died, occurred in Holland, Friesland, Guilders, and Liege. More than 2000 of Napoleon's soldiers died from it during the expedition to Egypt, and 4000 cases occurred in the English army during the Crimean war. About one-fourth (288,000) of all cases of sickness among the soldiers of the War of the Rebellion were said to be cases of dysentery. In the year 1890 an epidemic broke out in the province of Tuhuoka, Japan. This province had a population of 1,231,387, of which 25,272 were attacked. Of these 25,272 cases 4742 proved fatal. Smaller but equally fatal epidemics of the disease have repeatedly occurred on crowded ships and in periods of famine. Further than this it has long been recognized that this epidemic form of dysentery was distinctly infectious, and it can even be spread from one continent to another by infected ships, as in the great outbreak in the United States from 1846 to 1856, when it was probably conveyed by emigrants from Ireland, where the disease was rampant. More recently an instance of ship conveyance of the disease has been reported from the New Hebrides. Davidson states that in the decade of 1841 to 1851 no less than 50,019 persons died in the Irish workhouses from dysentery.

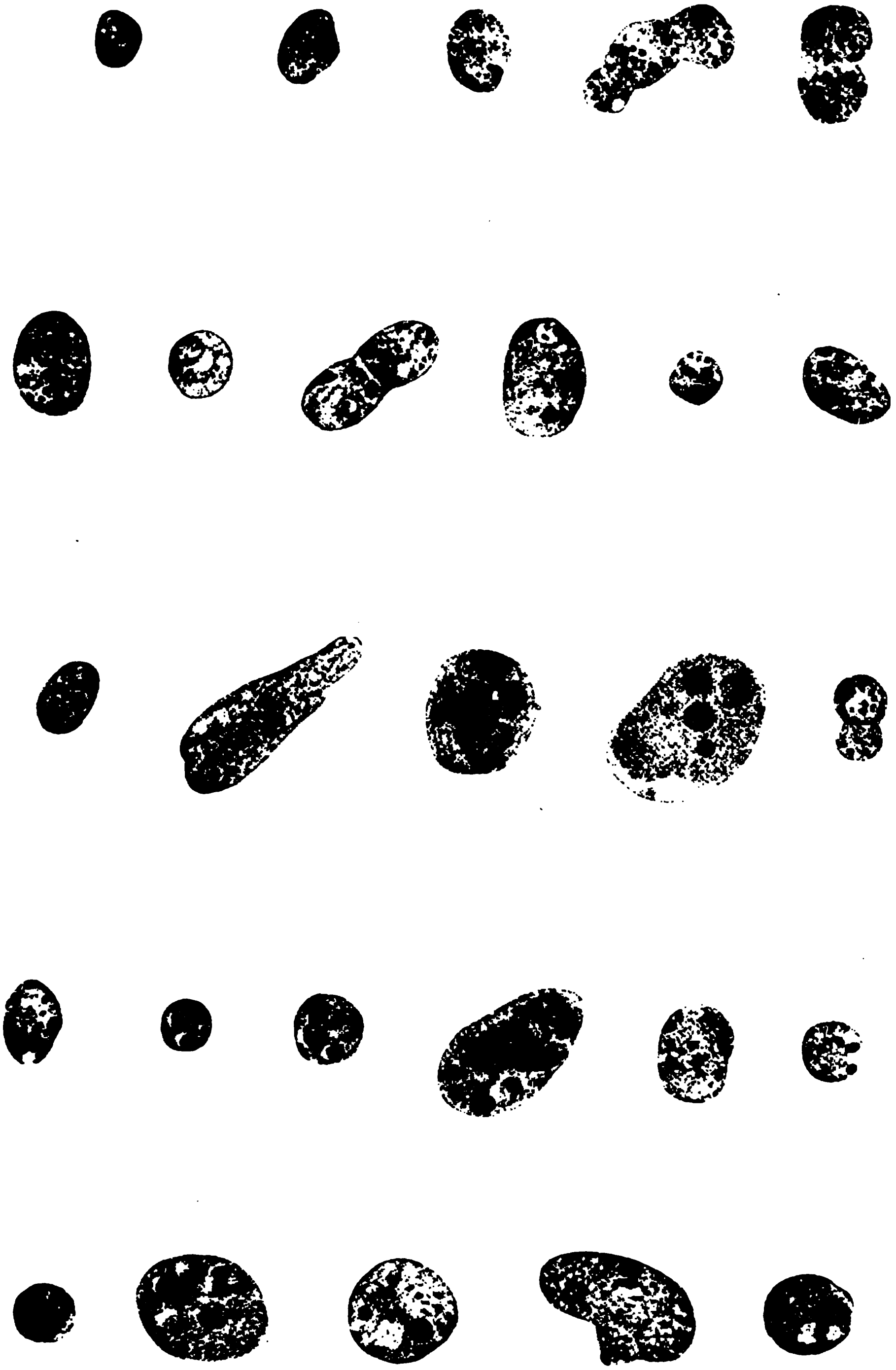
Marshy lands seem to have a pronounced predisposing influence. Water which has been contaminated by those who are ill with the disease is an important factor in its spread. Milk and solid food may also carry the infection.

Dysentery in its various forms is, in a large proportion of cases, the result of bad sanitation both as to surroundings and diet. It is much less frequent at present than in times past, and rarely ravages modern institutions or armies as it did fifty years ago.

Epidemic dysentery being exceedingly prevalent in Japan in 1897, Shiga, a Japanese investigator, became interested in its bacteriological study, and isolated from the stools of 36 patients suffering from this disease a slightly motile bacillus having rounded ends and decolorizing by Gram's method. When brought into contact with the blood serum of patients suffering with dysentery this bacillus usually agglutinates (as does the typhoid bacillus in the Widal test), although in a few mild cases the reaction fails to take place. Flexner, Strong, Kruse, Vedder and Duval, Vallard, Musgrave, Craig and Dopter, Spronck, Rosenthal, and other investigators have isolated in such cases organisms which they consider closely related to or identical with the one observed by Shiga, and which they believe to be the cause of acute epidemic, sporadic, and institutional dysentery.

Recently (1902) Duval and Bassett have obtained a similar organism from the stools of children suffering from dysentery or the summer diarrhœa of infants. Still more recently (1903) Wolfstein, Park, Dunham, and Carey have not only confirmed these findings, but have shown that at least two bacilli are present in cases of cholera infantum and dysentery. One of these corresponds to Shiga's bacillus, but they believe that in all probability several closely allied pathogenic bacilli will be found responsible in different epidemics. The bacilli are found in numbers proportionate to the severity

PLATE IV.



Amœbæ from Cases of Dysentery and Enteritis. (Roemer.)

of the illness, but often are not demonstrable in the stools until the latter are typical of the disease, and usually only after the lapse of five to seven days of illness. Chantemesse and Widal assert that a bacillus which they found in the stools of five dysentery patients, and which they also recovered from the mesenteric glands and intestinal wall of a patient who died of dysentery, is identical with the Shiga bacillus, and as their observations were made in 1888, ten years before Shiga published the results of his work, they claim priority of discovery. In France and Italy it is generally conceded that they were the first to find a specific organism in cases of dysentery.

Our recognition of the presence of *amœbæ* in cases of dysentery dates from 1859, when Lambl first discovered an amœba in the stools of this type of diarrhœa. Later the parasite was studied by Lösch (1875) and Kartulis, but it was not until Osler (in 1890), Councilman, and Lafleur (1891) reported upon its presence in several cases of dysentery, and in the past decade, that it received the attention that it deserves. Leukart has placed the *Amœba dysenteriæ* in the class of rhizopoda of the *Protozoa*. Schandinn calls it *Entamœba histologica* or *Entamœba dysenteriæ*.

The *Amœba dysenteriæ* is a spheroidal cell, four or eight times the size of the red blood cell. It consists of two parts, an internal part called the endosarc, or endoplasm, and an external part called the ectosarc, or ectoplasm. These two parts cannot always be clearly recognized when the organism is at rest, but they are easily identified when motion is present. The endosarc makes up the greater part of the body and its granules may be fine or coarse. In this portion several vacuoles are not rarely found and a distinct nucleus is discernible when the organism is stained. As in ordinary amœbæ the *Amœba dysenteriæ* often contains foreign bodies such as red blood cells, and even bacteria. The pseudopod, or arm, which is protruded from the amœba when it is engaged in amœboid movement, is of the hyaline ectosarc. (See Plate IV.)

In addition to this particular amœba other forms have been described by Quincke and Roos and other writers. One of these is much larger than that just described, called the *Amœba intestini vulgaris*, which is not capable of producing dysentery in man. The other is the *Amœba coli mitis*, which is pathogenic for man. It also is far larger than the amœba coli of Lösch—that is, the *Amœba dysenteriæ* of Councilman and Lafleur. The latter parasite is found in the stools of acute and chronic dysentery, in the floors of the intestinal ulcers, and in the secondary abscesses which it is prone to produce.

It is to be distinctly understood that two distinct types of infectious dysentery exist, one due to the bacillus of Shiga and one to the *Amœba dysenteriæ* Indeed, a third division due to the *Balantidium coli* may be made Savalier has collected 75 cases of this character and our own Strong has reported others.

Amœbic dysentery may occur at any age from infancy to senility, but it is most common between twenty and thirty years of age. It is much more common in men than in women. Thus, in 119 cases reported by Futcher 108 were males.

Prevention.—Dysentery in all its forms is to be prevented by the use of boiled water and cooked foods, by the establishment of proper drainage, and by the avoidance of cold and wet. Persons who are subject to catarrh of the colon and rectum should wear a flannel binder. When the disease develops, the stools of the patient should be thoroughly destroyed and the greatest care exercised that the food and drink of the healthy are not contaminated by his discharges.

Frequency.—Until very recently it was generally supposed that amœbic dysentery was the type of the disease most commonly met with in the United States, but now that Shiga's bacillus has been found in many cases of sporadic and epidemic diarrhœa in this country it must be regarded as the less frequent form of the two. Indeed, it would seem probable that many of the cases hitherto regarded as catarrhal are due to this bacillus. (See also Cholera Infantum.)

Pathology and Morbid Anatomy.—In *bacillary dysentery,* when death has occurred in the first week, the autopsy reveals the mucous membrane of the colon to be intensely corrugated and swollen, so that its natural rugosities are greatly emphasized, while over them is spread an easily detached layer of superficial epithelium, which has undergone necrotic changes. Numerous spots of ecchymosis, or hemorrhage, into the mucous membrane are often present, but ulcers are not found, although the necrotic process just named may be so severe that a superficial gangrene may be present. When the inflammation is very intense the whole thickness of the bowel wall may be indurated, and even the visceral peritoneum may be infected. In some instances an associated inflammation of the small bowel is present, somewhat similar changes being present in its coats.

Shiga described the morbid process of acute bacillary dysentery as a catarrhal inflammation proceeding to hemorrhagic, diphtheritic, or ulcerative inflammation. Kruse also observed diphtheritic membranes in eight cases which came to autopsy, and Flexner recognizes the tendency to their formation, although he did not find any in the cases which he examined postmortem in the Philippines.

Craig has reviewed the morbid anatomy of chronic cases of infectious or bacillary dysentery, recognizing follicular, diphtheritic, and gangrenous stages. In the first the coats of the colon usually are thickened, and the follicles, particularly of the cæcum, ulcerated. The mucosa is of a gray-slate color, and shows patches of acute congestion; the gut is narrowed, but there are areas of dilatation. The ulcers appear at the summit of the follicles as minute, ragged erosions. Later the necrotic areas extend, and their margins appear stamped out, but undermined. The ulcers measure $\frac{1}{6}$ to $\frac{1}{4}$ cm., but may attain diameters of 1.5 cm. and extend to the submucosa or muscular layer. Cicatrized and open ulcers may be found together. In the diphtheritic stage, which may be implanted on the follicular, the colon is grayish or greenish-blue, marked by red or dusky-brown areas and greatly thickened. The mucosa becomes necrotic, exfoliates in masses or irregular patches composed of granular detritus, leukocytes, and innumerable bacteria. Ulceration practically always accompanies the formation of the membrane. The gangrenous stage seems but an intensification of the diphtheritic. The serosa is more affected, and matted adhesions are the rule. The necrotic colon is easily torn, greenish-black, and marked by inky-black areas. The ileocæcal region is sacculated, and the sigmoid flexure and rectum dark olive-green in color. Tumefied, purulent elevations show through the serous coat. Internally the mucosa shows an indescribable admixture of necrotic or gangrenous lesions, with purulent suffusion of all the coats of the colon. In each of the foregoing forms parts of the mucosa escape, and these manifest more or less catarrhal inflammation. The protean manifestations of bacillary dysentery, both acute and chronic, are so influenced by the pathogenicity of the infecting organism, the activity of mixed or associated infection, susceptibility of the patient, duration of the process and other factors that an exact description is impossible.

The noteworthy difference between the lesions produced in children and adults by the *Bacillus dysenteriæ* is that in the former the solitary and agminated lymphatic tissues are much more commonly and more severely affected than in adults.

The lesions of *amœbic dysentery* are quite different from those of bacillary dysentery. In the first place the ulceration is confined almost entirely to the large intestine, although the lower part of the small intestine may be slightly affected. The submucous tissues become infiltrated and swollen in patches, which project above the level of the normal mucous membrane. These infiltrated areas undergo necrosis and slough away, leaving ulcers which may be superficial or deep, and which may extend as far as the peritoneal coat of the intestine, but perforation is rare. They are often very large and extend laterally as well as downward. The edge of the ulcer may be undermined and the floor honeycombed. Not rarely the extension laterally takes place under the mucosa or dissects the muscle coat so that there is only a small opening to a large area of necrotic tissue. Occasionally the submucosa is necrotic without evident superficial lesions. The *amœbæ* are found in the ulcers in the neighboring lymph spaces and sometimes in the bloodvessels of the part, but there is an extraordinary lack of pus when the severity of the necrotic process is considered.

When recovery takes place fibrous tissue covered by epithelium closes the spaces made by the ulcers, and as these scars contract strictures may develop. The colon becomes thickened and it may be adherent to the adjacent structures and uneven contractions form pockets in which the parasites may linger after apparent clinical recovery. The appendix may be involved.

The changes in the liver in amœbic dysentery consist of two alterations. The first are multiple areas of local necrosis, and secondly abscess, either single or multiple. The single abscesses are usually large and in the convexity of the right lobe, or else in the concavity of the liver where it lies nearest the large bowel. Roux has collected 639 cases of amœbic abscess of the liver. Of these, 435, or 70.8 per cent., were in the right lobe; 85, or 13.3 per cent., were in the left lobe, and 2, or 0.3 per cent., were in the lobus Spigelia. The multiple abscesses are usually small and widely scattered and often near the surface. It is noteworthy that these so-called abscesses do not contain true pus unless secondary infection with pus organisms has occurred. They are composed of a grumous material made up of a thick, coarse, irregular reticulum, in the meshes of which lie the semi-fluid contents. As the area increases in size the fluid becomes reddish, brownish, greenish-yellow, or chocolate color, and is mixed with pieces or shreds of broken-down hepatic tissue. Amœbæ may be found in the contents of these cavities.

Abscess of the liver due to the *Amœba dysenteriæ* nearly always develops in the first few weeks of the disease. Occasionally one of the larger abscesses ruptures into the right lung. (See Complications.) Boston has collected statistics of 2340 cases of amœbic dysentery. Of these, 486, or 20 per cent., suffered from hepatic abscess. (See Hepatic Abscess.) The percentage varies from 60 per cent. (Kartulis) to 21 per cent. (Councilman and Lafleur.)

A valuable contribution to the subject of the associated lesions of dysentery has been made by Craig, of the United States Army Medical Staff. Analyzing 120 cases of dysentery, of which 60 were of the bacillary and 60 of the amœbic type, he found that in nearly every instance the autopsy revealed an increase in the cerebrospinal fluid and œdema of the brain. In the amœbic cases an intense congestion of the cerebral vessels was also present, and in 50 per cent. minute capillary hemorrhages were present. In the bacillary cases, on the other hand, the brain seemed unduly anæmic. In the respiratory system bronchopneumonia is the most common lesion in the bacillary disease. Craig believes that fully 60 per cent. of the cases of dysentery seen in the San Francisco Military Hospital have coincident nephritis, and of the 120 cases already cited no less than 101 had this condition, usually of the parenchymatous type. More cases of nephritis occur in the amœbic than in the bacillary type.

In the *acute catarrhal form* there is a free production of mucus which coats the surface of the lower bowel, chiefly in the sigmoid flexure and rectum. This mucus is filled with exfoliated epithelium, some of which has undergone fatty degeneration. Not infrequently blood cells are present in the mucus. When the inflammatory process is severe, marked congestion and infiltration of the mucous membrane and submucosa may be present, and even a purulent and superficial ulceration may occur.

The *diphtheritic type* is characterized by congestion of the mucous membrane and the development upon its surface of a false membrane. The connective tissue under it, and between the glands, is infiltrated and filled with fibrin and pus. In cases in which the process is very active, the inflammation may reach not only the muscular coats, but even the peritoneal coat. The area covered by the false membrane varies greatly in different cases. In some only the rectal mucous membrane is affected, in others a continuous exudate covers the entire colon, and in still others it appears in scattered patches. If the process is severe healing takes place by sloughing of the necrotic tissues, which may reach to the deeper layers of the bowel, leaving ulcers which gradually undergo cicatrization, or the ulcers remain granulating surfaces for months and only heal under direct treatment.

Symptoms.—The symptoms of all the various forms of dysentery are closely similar. The *onset* is usually *sudden*, or it may develop in the course of a gradually increasing diarrhœa, which at first is thought to be an ordinary attack of looseness of the bowels. The patient suffers from *wretchedness*, which is thought to be the result of the intestinal disorder, and often has, in the earliest stages, a considerable degree of *griping pain*. The initial diarrhœa soon sweeps the bowels clean of their normal contents, and as soon as this is accomplished the stools become scanty and consist largely of mucus which, not rarely, *contains blood*. The griping pain increases in violence, and there is *marked tenesmus* which often causes the patient to break out in a profuse sweat. The rectal irritation causes a constant desire to go to stool, which is not satisfied by the small evacuation that occurs. At first the constant irritation of the anus may cause *spasm of the sphincters*, but later when the disease is severe the sphincter ani may become relaxed, and even *rectal prolapse* may ensue. The centres in the spinal cord controlling the bladder become reflexly irritated and *difficult urination* may add to the suffering of the patient.

It is manifest that such symptoms must speedily cause grave systemic disturbance by reason of the loss of nutritive material, the constant pain, the loss of sleep and straining, and so the *pulse* soon becomes *rapid* and *feeble*, and the patient rapidly *emaciates* not only because of the reasons just cited, but also because the local lesion in the bowels soon results in general systemic infection, either with the specific cause of the attack or with other micro-organisms which gain access to the general system through the diseased intestinal wall.

The *tongue* is very *foul* and the secretions of the mouth scanty.

If the disease persists the scanty mucous stools may be supplanted by more profuse serous discharges, which are often reddish in hue, and seem to contain small particles of flesh (probably bloody mucus and mucous membrane). This fluid is highly albuminous. The debility and emaciation of the patient speedily becomes profound, as the loss of fluid and albumin continues. Whether the *stools* are mucous or serous, they are *fetid* and have an odor which is quite characteristic.

In that form of dysentery called *bacillary* the fever at first may rise as high as 103°, but in the *amœbic form* the temperature is usually not greatly disturbed unless secondary abscess develops. It rarely rises above 102°, and may be subnormal after the stage of onset.

When the infection with Shiga's bacillus is very virulent, death from toxæmia and exhaustion may occur as early as the fourth day, but, on the other hand, the case may last for much longer periods before the fatal result ensues. There is sometimes met a subacute form, which lasts, in a modified type, for weeks or even months.

Cases of *amœbic dysentery* may be divided into *three types:* (*a*) A mild form in which the general health remains good, although the number of stools may vary from two to six in a day. (*b*) A moderately severe form in which the general health is greatly impaired and there is much loss of flesh, with an evening rise of temperature and frequent stools. (*c*) A very severe type is met with in which the prostration and loss of weight are extreme, the stools are bloody and very frequent, and the extremities cold. In all these cases the patient may without any apparent cause pass to better or worse with extraordinary speed.

Amœbic dysentery may cause death in a few days or last for weeks, and may cause death finally by the secondary abscesses in the liver. Free hemorrhages from the bowel may also occur in this form, and perforation from deep ulcers is recorded. A chronic form of amœbic dysentery also exists which lasts for months and has temporary periods of constipation. These periods of constipation, if abscess does not develop, give the patient an opportunity to be nourished, and so he may be able to retain strength and flesh.

In the *acute catarrhal* form of dysentery there may be *fever* at onset and scybalous masses will often be found mixed with the mucus which is expelled. After an illness of from four to seven days the quantity of blood in the stools is decreased, and they become less frequent. Marked *abdominal tenderness* over the course of the large bowel is usually present in all cases.

In the *acute diphtheritic form* the patient is usually extremely ill from the very first. The systemic depression is marked and *profound adynamia* is quickly developed. The *belly is distended* and *painful* upon pressure. *Bloody mucus* is not uncommonly absent from the stools. Just as in the other forms so in this, a subacute or chronic type is met with in which the abdominal signs are mild and the number of stools a day are as low as four or five.

Complications and Sequelæ.—*Perforation of the bowel* has already been named as a possible sequel of the *amœbic form* of the disease. In other instances a *localized peritonitis* develops, and as the result of infection of the tissues about the colon a perityphlitis or periproctitis comes on. *Rupture of an hepatic abscess* is a very frequent occurrence. The pus finds its way through the diaphragm into the right lung or right pleural space. Rarely it has burst into the duodenum and even into the vena cava, or backward and downward along the psoas muscle, or into the kidney. Even the pericardium and the bladder may be perforated. Strong has called attention to the occurrence of profuse intestinal hemorrhage.

In many epidemics of dysentery there is associated mild or severe malarial infection which renders the case difficult of treatment in that two infections have to be controlled simultaneously. *Septic arthritis*, *pericarditis*, and *endocarditis* sometimes occur as terminal infections. (See Pathology and Morbid Anatomy.)

Diagnosis.—The diagnosis that the patient has acute inflammation of the large bowel, and is therefore suffering from dysentery in one of its forms, is easily made if the symptoms just described are present. It is not, of course, so easy to determine which of the several forms of dysentery is present. The bacillary form is separated from the amœbic variety by the presence of marked fever, which is usually absent in the latter disease; by the discovery of the specific bacillus in the stools, which discovery, however, requires special training in searching for it; and by the agglutination test of the bacillus with the patient's blood serum, which, as in the case of the Widal test of the blood in typhoid fever, gives us such valuable information. This reaction is uncertain in the first week, often positive after the sixth day, but in some cases it does not occur for two weeks. For this reason it does not possess great diagnostic importance in the early stages of the disease. To be of value it must take place in a dilution of 1:200. The percentage of positive reactions according to Rosenberger is 80.2 per cent.

The amœbic variety can only be recognized during life by finding the amœba in the stools. This requires some practice and skill. The small pieces of blood-stained mucus are the parts in which the organism is to be sought for, first with a low-power and then with a high-power lens. (See Plate IV.) The light coming through the instrument should be stopped down by an appropriate diaphragm. Several negative examinations do not exclude amœbic dysentery, and particularly in chronic cases it is necessary to make repeated examinations. In such cases acute exacerbations may afford stools relatively rich in amœbæ even when intercurrent examinations have been negative several times. The stools should be as fresh as possible, unmixed with urine, and, if not warm, the slide examined should be warmed gently or placed on a warm stage so as to induce movements of the amœbæ. If an organism which possesses active amœboid movement is discovered and if it contains several red blood cells the diagnosis is practically assured.

When examining the stools it is essential to bear in mind that a multitude of intestinal bacteria are also present and that various parasites other than the specific amœba may be present. Thus, the *Trichomonas intestinalis* and the *Cercomonas intestinalis* are often found. Thayer has recorded a case in which the *Strongyloides intestinalis* was present as an additional parasite.

The diphtheritic form is to be suspected if from the first the patient seems profoundly adynamic. Typhoid fever is to be separated from dysentery by the fever, the rose rash, and the Widal test.

Prognosis.—The prognosis in dysentery depends to some extent upon the variety of infection which is present, and upon the hygienic surroundings and vitality of the patient, for even the mildest types may be fatal if the patient receives bad food and is exposed to excessive heat or cold or wet. When the *bacillus of Shiga* is the cause the prognosis in acute cases must always be most guarded, both as to the recovery and the duration of the illness. The mortality varies greatly in different epidemics in different parts of the world. Thus in Japan, Shiga found it varied from 22 to 55 per cent. In this country the mortality has been as low as 3 per cent. The general state of emaciation and depression must always be considered. If the stools contain gangrenous sloughs, the outlook is of course very grave; and if hiccough, great nervous depression, and low delirium develop, the outlook is probably fatal.

In the *amœbic type* the development of abscess in the liver of course adds very greatly to the gravity of the case; but even when amœbic abscess is present and ruptures into the lung, it is possible for recovery to take place, if proper surgical measures of relief are undertaken.

Even the most urgent cases may, when apparently near to death, recover, but convalescence is protracted.

According to Duncan prognosis can be based, to some extent at least, on the character of the stools. He believes a good result can be foreshadowed in those cases in which are passed mucus with minute fecal lumps, stained or not with blood, and in which the blood and mucus disappear, after which the ordinary fecal characters will soon manifest themselves.

The prognosis is of evil omen, according to Sir Joseph Fayrer: (*a*) in the cases in which pulpy stools without blood or mucus are passed;

(*b*) where fluid fecal matter is from time to time passed throughout the illness, the prognosis is unfavorable, inasmuch as these characters of the stools show the disease to be extensive, and affecting chiefly the upper part of the large as well as in some cases part of the small intestine; (*c*) where the stools in conjunction with the symptoms that are laid down as characterizing the true amœbic dysentery are present, the prognosis is again unfavorable, on account of the high mortality that is said to attend this form of the disease; (*d*) the prognosis is of the worst possible character where the stools consist of blackish-red or blackish fluid with a horribly putrescent odor, and of bits of gangrenous tissue. Duncan has never seen a patient passing this character of stool recover.

Treatment.—So far as diet is concerned it is self-evident that the food should consist of those substances which are readily digested and absorbed from the stomach and the duodenum, in order that as small a residue as possible may pass on downward into the large bowel. Milk, which is so universally resorted to in the treatment of all forms of diarrhœa, is not always as useful a form of nutriment as it is thought to be; for not uncommonly it will be found that when milk is taken it remains undigested, or forms curds which are indigestible because of the feeble secretion of digestive juice. These curds pass through the bowels and afford pabulum for micro-organisms which, in turn, are injurious to the mucous membrane. If it is given, it should certainly be diluted freely with lime-water, barley-water, or Vichy water, or else it should be peptonized in order that its digestion may be readily performed; and it is of vital importance in this connection that it should be given in small quantities, frequently, rather than in large quantities. Solid food is, of course, contraindicated, but semi-solid foods like milk-toast, the digestion of which is aided by pancreatin or taka-diastase, and a very soft-boiled egg, will often prove a better diet than one which is more liquid, but less nourishing, since the physician, in the presence of dysentery, is faced by two opposing factors; on the one hand a feeble digestion, and on the other hand the necessity of supporting vitality to the highest possible point by the administration of proper foodstuffs.

The treatment of the condition itself may be divided into three methods, and each one of these plans finds ardent advocates among those of the profession who have had sufficient experience to make us feel that their opinions are of value.

The *ipecac plan* may be considered among the first of these, and it cannot be doubted that physicians in tropical countries have found it of benefit in so large a number of cases that it is impossible to consider that they have been mistaken in their clinical observation. As Dr. Woodhull, late Assistant Surgeon-General of the U. S. Army, has said, the one remedy which, properly used, is as conspicuous in dysentery as quinine is in malaria is ipecacuanha. He gives the following directions for its use:

"The stomach must be empty and the patient recumbent. About twenty minutes before giving the ipecac it is well to paint the epigastrium, not the whole abdomen, with tincture of iodine, or to apply a mild sinapism sufficiently to induce gentle counterirritation. This precaution, however, may sometimes be omitted, or may be deferred until the medicine has been taken. Ten or 15 minims of laudanum may be given, always on an empty stomach, to be followed in ten or twelve minutes by from 15 to 30 or more grains of ipecac in pill form, or as a paste, with a very small quantity of water. No food or fluid should be taken for at least four hours, and recumbent rest should be strictly maintained. If the ipecac is administered in pill or capsule the laudanum may be mixed with it instead of given previously. One scruple of ipecac and 1 grain of opium can be made into four pills, or the laudanum can be put in the pills. When pills are used they should be freshly made. Or 20 grains of ipecac can be suspended in 2 fluidrachms of water with a few drops of an aromatic to disguise the taste. It is never advisable, on account of the popular idea associated with it, to disclose the name of the medicine, and the patient should be warned to resist any inclination to vomit. The size of the dose should be in proportion to the gravity of the case. Just as in severe colic very large doses of opium are tolerated, and in pernicious fever enormous quantities of quinine are indicated, so in dysentery surprisingly large doses of ipecac are well borne, although the magnitude of the dose should bear some relation to the severity of the disease. With a little experience, that relation will soon be determined. Sixty grains is not a maximum dose for an adult, but with ordinary acute dysentery from 15 to 25 grains at a time should suffice. If the first or any subsequent dose is rejected, which rarely happens if these rules are carefully followed, it is to be repeated after a short interval. The retching or vomiting of exhaustion or the restlessness of delirium is no bar, but rather an inducement to this treatment; and small children or delicate women can take it with impunity in proportionate amounts.

"The common course in acute dysentery is, first, the relief of pain, next the subsidence of fever, and then the cessation of the bloody discharges. The usual sign that recovery is at hand is a painless, copious, semi-fluid evacuation, much the color of the ipecacuanha powder, not black as has been stated. The medicine then may be reduced or entirely suspended. In acute cases these results will follow very quickly. In chronic dysentery complete recovery may be delayed or, indeed, may fail of absolute attainment, but great amelioration may be confidently anticipated. That the powder should be pure and comparatively fresh is always essential."

The second method of treatment is the *purgative plan*, which has come forward largely within the last few years, probably because of increasing experience on the part of American and English surgeons in the Philippines and in South Africa. Clinical evidence is rapidly accumulating which proves beyond all doubt that in a certain proportion of cases of acute dysentery the employment of sulphate of magnesium combined with aromatic sulphuric acid is a most advantageous method. The bowels are first thoroughly moved with Epsom salts or with Rochelle salts, and then aromatic sulphuric acid is given freely, so that it will exercise its well-known astringent or constipation influence. This plan is a more rational one than that which concerns the employment of ipecac, in that it is a well-known fact that the micro-organisms which are commonly found in the intestines in dysenteric cases are destroyed or rendered inert by an acid medium, and it has long been known by the profession that the administration of sulphuric acid is apt to produce an acid reaction of the stools, so that its beneficial influence in dysentery does not rest alone upon its astringent effect, but upon its power to destroy the infecting micro-organisms, just as its use in Asiatic cholera meets the double indication of restraining diarrhœa and destroying the comma bacillus.

The third plan of treatment consists in the administration of *intestinal antiseptics*, of which perhaps bismuth salicylate, benzonaphthol, and salol have been most commonly employed. Theoretically, it is easy to conceive that these substances may be advantageous, but practical experience has shown that they fail to exercise the degree of antiseptic influence with which they are credited, and they are not of sufficient importance to justify their employment to the exclusion of the ipecac or saline methods which have just been described. The employment of calomel and corrosive sublimate with good results in these cases rests upon the fact that they increase the activity of the liver, both in destroying toxic material and in secreting bile.

Without doubt *local treatment* by high intestinal irrigations is of very great value. Copious clysters which will reach far up into the descending and transverse colon are necessary. In a number of instances the writer found that injections of sulphocarbolate of zinc, in the proportion of 20 grains to the pint, have produced very satisfactory results, the zinc acting both as an astringent and antiseptic. Other practitioners have employed copious injections of weak solutions of nitrate of silver of the strength of 1 drachm to 4 pints. The tenesmus which is frequently associated with the dysenteric condition, or on the introduction of the soft rectal tube, can sometimes be avoided by the use of a 10 grain iodoform suppository used half an hour before the injection is to be given. This suppository, by its local anæsthetic effect, is of service, and I have thought that the absorption of the iodine from it was also advantageous.

The method of giving the intestinal lavage is of considerable importance. It should not be given with a Davidson or other pumping syringe, but always by means of a fountain syringe or surgical irrigator. The hydrostatic pressure employed should never be greater than two or three feet, and it is much better that the injection should be gently given, so that it takes fifteen or twenty minutes to find its way up into the intestine, than that it should be delivered forcibly enough to produce angry contractions of the bowel, which will cause great agony and so much irritation that the treatment makes the patient worse.

Where great irritability of the bowel exists it is probably better to employ two rubber catheters side by side, one being for the intake and the other for the outflow, since in this way great distention of the bowel is avoided. In instances in which cold-water injections seem inadvisable very hot water may be employed, but it is distinctly disadvantageous to employ tepid water, which has a relaxing and enervating effect, and does not possess the healthy stimulant effects of marked cold or high heat. Often it is best to employ normal saline solution, since by this means maceration of the intestinal mucous membrane is avoided.

Specific treatment for bacillary dysentery promises much for the future.

Attempts to produce a protective and curative serum have been made, and Shiga maintains that he reduced the mortality of dysentery one-third by the use of such a substance. Up to November 1, 1899, he had treated 156 cases with his serum, with a mortality of 8.5 per cent. During the same period 166 cases in Tokio and Honjo Hospitals under ordinary treatment gave a mortality of 37.9, and 398 at Komogone Hospital gave a mortality of 34.6. In private practice also mortality was high, 1119 cases giving a percentage of 28.5. Kitasato likewise obtained a low mortality rate by this plan of treatment. (See Cholera Infantum.)

Another form of specific treatment is that which is directed to combating *amœbic dysentery* by means of injecting *quinine bisulphate*, in the strength of 1:5000, sufficiently high in the rectum for it to exercise its fatal effect upon the amœba coli. Thymol 1:2500 may also be used in this way. (Thomas). Harris has highly recommended hydrogen peroxide given by injections as a parasiticide.

Stimulants well diluted with water or with nutritive broths should be given if needed, and strychnine and quinine employed in convalescence as tonics.

For the control of the diarrhœa when excessive, enemata of deodorized tincture of opium and starch-water are very useful.

EPIDEMIC GANGRENOUS PROCTITIS.

Definition.—Epidemic gangrenous proctitis is an acute contagious disease appearing as a rapidly spreading ulceration of the anus and rectum, with prolapse and gangrene of the ulcerated rectum and, in a large proportion of cases, death in coma or convulsions.

This disease, originally believed to be limited to narrow areas in Central and South America, is now known to occur much more widely throughout the tropical zones. It is generally distributed in tropical South America and in Central America. It has been observed in the Philippines, the Celebes, and New Guinea.

There is some question whether this affection should be regarded as a distinct disease entity or not. I (Kieffer) regard it as a localization of an intense dysenteric process unusually low in the colon, without hazarding any theory as to the cause of the localization. This idea is strengthened by the well-known liability in children to intussusception above the sigmoid in acute dysentery. It is quite fair to assume that the mechanism of the extrusion of the rectum in these cases is the same.

Etiology.—Nothing is known of the direct cause of this curious condition. Ackers, of Curacao, states that it is the common belief of the natives in Venezuela that the disease is caused by eating unripe maize, of which the children are particularly fond. This seems hardly probable, although the symptoms of pellagra, which are assumed to be due to fermented maize, would indicate the possibility that unripe maize may account for the symptoms. The disease, however, has been observed in countries in which maize is practically an unknown food product. I (Kieffer) have reported one case in which the *Bacillus pyocyaneus* was undoubtedly the active organism. A high degree of humidity seems an essential condition to the development of the disease. Ackers observed this disease in fowls and the smaller domestic animals, occasionally in calves. In Venezuela and New Guinea the disease is confined to children, particularly those of the poorer classes, and in the rest of the tropical world it also holds true that children are more frequently attacked than adults.

Pathology.—The rectum and anus in early stages are affected by deep ulcers overlaid with a diphtheroid pseudomembrane. Two forms can usually be distinguished: the low form with limitation of the lesions to the rectum between the sphincters and the high form in which the disease extends well up to the sigmoid.

Symptoms.—The disease begins with local symptoms referred to the anus and rectum. There is burning and intolerable itching followed by severe dysenteric symptoms. After twelve to twenty-four hours there is more or less constant and severe tenesmus.

The evacuations, at first feculent, become mucoid and finally consist entirely of mucus and blood. The distress grows urgent, effort and pain become continuous, and there is a constant flow or bubbling out of slimy mucus stained with blood, or almost pure frothy blood. The evacuations are very fetid. As the disease progresses there is profound collapse. Nervous symptoms appear and the patients become either delirious or comatose. In children there may be convulsions. Emaciation is rapid and death usually occurs in convulsions or coma. If the patient survives this stage the rectum is extruded and undergoes rapid necrosis and sloughing.

In the early stages the diagnosis between epidemic gangrenous rectitis and dysenteric lesions in the descending colon can only be made by examination of the rectum. In advanced cases the condition is self-evident. The mortality is very high, but even comatose patients need not be despaired of. Recovery occasionally occurs after prolapse and sloughing of the rectum.

Treatment.—The Venezuelan treats this disease by introducing lemon-juice or diluted *aguardiente* into the rectum. When extrusion of the rectum occurs he keeps it dry by dusting with wood-ashes. Indications are for active antisepsis of the rectum with diluted creolin or hydrogen peroxide. Opium will be necessary for the control of pain, and is best applied directly to the diseased area. If prolapse occurs no effort should be made at first to replace it. The rectum should be dusted with an antiseptic powder or freshly made charcoal. If gangrene occurs the rectum must be extirpated.

HILL DIARRHŒA.

Definition.—An acute morning diarrhœa with white stools and attended by marked flatulency. It is a disease of the acclimatized and not of the new-comer in the tropics.

Etiology.—The etiological factors in the production of hill diarrhœa are, first, prolonged residence in hot countries with the establishment of acclimatization, then an unaccustomed altitude, five to six thousand feet, with a high degree of humidity. Under these conditions the dweller in the low, hot plains who goes to the hills is very prone to fall a victim to this disease. It is consequently observed when business or relaxation takes the colonists into mountainous portions of the tropics. It is common in the hill sanatoria of tropical countries, particularly in India, where it was named Hill diarrhœa, Simla diarrhœa, etc. Hill diarrhœa bears a very close resemblance to sprue. The cardinal symptoms differ only in degree, but the tendency of hill diarrhœa is so constantly to recovery, and that of sprue so constantly downward, that they must be considered separately.

Pathology.—Very little is known of the pathology of this peculiar condition. It seems to be clear that there is a temporary suspension of function of the liver and pancreas. This is probably the expression of exhaustion resulting from the extra strain on already overworked digestive organs seeking to adapt themselves to a further change of climatic conditions. Scheube thinks this condition depends on an atonic state of the colon which he believes to be a common sequel to long residence in hot countries, and that the diarrhœa is due to chilling of the abdomen in the unaccustomed cold and dampness of the early morning hours of the mountainous region of the tropics.

The tendency in the vast majority of cases is to prompt readjustment and restoration within one or two weeks. A small proportion of cases persist and may end in typical sprue. Crombie reports cases in which cure has taken place only when the patients returned to the plains. In these cases every visit to the hills was followed by this diarrhœa.

Symptoms.—Shortly after arrival in the mountains the patient is troubled with *dyspeptic symptoms* and a *morning diarrhœa.* On the succeeding days the diarrhœa becomes more troublesome until it reaches eight to ten movements daily. It comes on in the early morning, at or near dawn, with a sudden call to stool. It continues during the forenoon and *ceases abruptly about mid-day.* There is very little pain and that only as a vague, indeterminate discomfort over the colon, and no tormina or tenesmus. The *movements* are *large* and *frothy,* they are devoid of coloring matter, and look like stirred mortar or whitewash. They have an unpleasant, mawkish odor. Dyspeptic symptoms are pronounced. There is *distress after eating,* particularly in the morning, and there may be marked *tympanitic distention* of the abdomen.

Treatment.—Treatment is directed to the restoration of intestinal digestion and the maintenance of a relative degree of rest to the gastrointestinal tract by putting the patient on liquid or milk diet. Small doses of calomel to stimulate the hepatic function are of value. Similarly pilocarpine should be tried with the idea of increasing the flow of the pancreatic secretion. Judicious use of the digestive ferments should be made. These patients should be advised to keep to their beds during the morning hours. In persistent cases it may become necessary to send the patients down to, or near, the sea level.

SPRUE (PSILOSIS).

Definition.—Sprue (*Atrophic Enteritis of the Tropics*) is not a distinct morbid entity; it is but a terminal condition of many devitalizing and depressing factors. Sprue may be defined as a chronic catarrhal inflammation of the entire alimentary tract tending ultimately to most extensive atrophy of the gastrointestinal glandular structures, characterized clinically by three cardinal symptoms—sore mouth, flatulency, and diarrhœa.

Sprue is a disease of the entire tropical world. The Malayan Archipelago, particularly the Philippine Islands, South China, Amoy, Ceylon, Java, and the Malayan Peninsula are the regions of its greatest development.

It is not seen in subtropical or temperate zones, unless in imported cases, with the exception of Japan and northern China. It has been known by many names, all of them referring to one or another of the peculiar clinical manifestations of the disease. Thus, it has been called "diarrhœa alba," "aphthæ tropicæ," "Ceylon sour mouth," and "white flux." The name *sprue* was given to it by the Dutch, who found it a veritable plague in their Javanese possession. Since the United States has acquired colonies in the sprue region, imported cases have become fairly numerous throughout this country, and their consideration is of great interest to the general practitioner.

Etiology.—Extensive studies of the disease have failed to show any specific etiological factor. There is some argument whether sprue should be considered a specific disease or whether it should be considered as a terminal state following other lesions. Sambon maintains the former. Extensive studies by army surgeons in Manila show that the latter view is the correct one. The disease commonly develops in the tropics, but may lie dormant for years after the return of the colonist to his home in the temperate zone.

The conditions that predispose to the development of sprue are the following: The large majority of cases follow chronic amœbic dysentery and its sequelæ, chronic ulceration of the colon and abscess of the liver, with prolonged suppuration. Following dysentery the intestinal parasites, and chief of these the uncinaria, are common antecedents of sprue. Next is the general deterioration in chronic malarial poisoning, and lastly syphilis is a cause. To these must be added the physical deterioration incident to prolonged residence in hot climates, even when no acute illness is suffered, and the following depressing conditions: childbirth and miscarriage, chronic disease of the kidneys, suppurating lesions anywhere, excessive fatigue, long marches, and prolonged campaigns. Some writers report the development of sprue as following the prolonged administration of iodides and mercury. Numerous organisms have been described as the cause of sprue and several parasites, particularly the strongyloides. Amœbæ are almost constantly found and various bacilli of the typho-colon group, but these must all be considered remnants of the preceding pathological condition rather than direct causes of the disease itself.

Pathology.—Postmortem examination shows complete loss of subcutaneous and mesenteric fat. The tissues and cavities are extremely dry; the small intestine presents extreme thinning of its walls with atrophy of the mucosa and, in some cases, entire destruction of the glandular structures. The serous coat is normal. Swelling and ulceration of Peyer's patches are seen and in many cases the colon presents the usual appearances of recent or present dysentery. Parenchymatous changes occur in the pancreas, liver and kidneys, and occasional areas of fatty degeneration are present.

Symptoms.—Sprue is essentially a very chronic disease. The average duration is from one to two years, although cases lasting ten years or more are not by any means unusual. The patient is *emaciated* and *anæmic.* The *complexion* is *muddy* and *sallow.* There is *great lassitude* and weakness, and mental irritability with pronounced disinclination for physical and mental labor. The disease passes through numerous stages of amelioration and numerous recrudescences, but its general tendency is always downward. The principal symptoms of sprue are *sore mouth, diarrhœa,* and *flatulence.*

The *mouth lesions* of sprue are constant and striking. On examination the tongue is found unusually clean. The organ is small, pointed, and somewhat yellowish. Along the dorsum of the tongue, along its edges, and on the under side, particularly along the frænum, there are numerous, fine, minute ulcers, with a thin, aphthous pellicle. These aphthous spots may also be present on the uvula and palate. Very commonly the tongue is covered with very superficial erosions from one-eighth to one-fourth of an inch in diameter, frequently coalescing and resulting in a serpiginous appearance. Where these marked lesions are temporarily absent, the tongue still has an unusually clean, dry, glazed appearance, and looks very much as though it had been recently varnished. The condition in older patients may be very much accentuated and extensive fissures may develop. The patient complains of soreness that may be limited only to the tongue, or may involve the palate or uvula, or the entire mouth. He particularly complains of burning or a stinging pain on taking salt or highly seasoned food. Occasionally the pain is also present on deglutition, and the progress of the food bolus on its way to the stomach is indicated by a burning and *stinging pain in the gullet,* showing that the œsophagus is in the same condition as the mouth. *Nausea* and *vomiting* are sometimes present in advanced cases, the vomiting coming on without reference to the time of taking food. *Eructations* and *waterbrash* are present; the *appetite* is very variable, sometimes being entirely absent, at other times ravenous.

Flatulence is quite marked. The patient is swollen until the abdomen is tense and drum-like. In this condition the appearance of the very much emaciated figure, with extremely thin arms and legs, and large, inflated abdomen, is very characteristic. The flatulence is always aggravated by taking food and is accompanied by a constant sense of oppression and a gnawing and burning pain in the stomach.

Diarrhœa is the most distinctive and constant symptom. There may be only one or two movements daily or there may be as many as ten or twelve. They are usually passed without pain; they vary very much in their character, but are nearly always liquid or semi-liquid. They are frothy, white, and have a fetid, mouse-like odor. Manson describes them as looking like recently stirred whitewash. They are usually remarkably large.

Their reaction is commonly acid. Microscopically they are found to contain bowel structure, a few red blood cells, and intestinal parasites, of which amœbæ are the most frequent.

As the disease advances *emaciation* and *asthenia* become extreme. The skin is dry and scurfy, the patient is unable to assimilate or retain food, and, in many instances, he involuntarily abstains from it on account of the severe pain in the mouth and throat. In all marked cases there is *secondary anæmia,* the red blood corpuscles being reduced as low as 1,000,000, with some degree of poikilocytosis and no leukocytosis.

Diagnosis.—Diagnosis presents no difficulties, when the existence of such a disease as sprue is known and its cardinal symptoms are remembered. Incomplete cases in which one or the other symptom may predominate or be absent may give rise to some difficulties.

Sprue in particular must not be confounded with chronic dysentery, although it is difficult, in cases where sprue develops from dysentery, to definitely mark the period where the one disease begins and the other ends.

Prognosis.—If treatment is instituted before the atrophy of the bowel is too far advanced, cure is the rule. If, however, so much of the secreting surface of the bowel is destroyed as to make assimilation impossible, death is, of course, inevitable.

Treatment.—Treatment consists in putting the patient on absolute milk diet. He should preferably be kept in bed and milk administered in small feedings. The quantity is increased as rapidly as possible, the mouth symptoms and the appearance of unchanged milk in the bowel movements being the index as to the quantity to be taken. If there be any increase in the amount of soreness of the mouth, the milk must be reduced for a time and later gradually increased, until the patient is taking from four to six quarts daily. This regimen must be persisted in from four to six weeks after the mouth symptoms disappear and the bowel movements become solid. Then soft diet should be resumed very carefully. Where milk cannot be taken, pure meat diet may be used or an exclusive diet of meat-juice. Success has also attended the exclusive use of a fruit diet. Recent observations have shown the value of fruit, particularly of berries, in the treatment of sprue as well as chronic dysentery. Manson reports the case of a man over fifty years of age, at which time of life the prognosis in cases of sprue is exceedingly grave. The patient was first treated, with little benefit, with milk diet and other special diets; finally the patient was put on a diet of strawberries. The stools at once improved and the patient was soon restored to health. This was not the only case in which recovery followed the use of strawberries. Manson considers this a decided advance in the treatment of sprue. Medicinally salol and Dover's powder may be given for control of the diarrhœa. For their tonic and reconstructive properties iron and arsenic should be used. These drugs have been shown to be of particular advantage in this disease when used by hypodermic injection.

NASHA FEVER.

Nasha, nasa, or nakra fever, first described by Fernandez and Mitra in 1894, is a specific fever confined to certain sections of India. It is preceded by marked congestion of the nose and circumscribed swelling of the nasal septum. The disease begins with malaise, headache, and severe pain in the muscles of the neck, back, and shoulders. There may be a small, diffuse, rosy eruption. The fever is moderate and usually lasts from three to five days, gradually subsiding with the disappearance of the nasal symptoms. In rare instances sudden diminution of the local symptoms has been followed by severe cerebral symptoms, coma, and death.

The etiology is unknown. It occurs principally in adults, children and the aged being relatively immune, and it comes on usually in the summer months. Exposure, unhygienic surroundings, and poor food seem to be the predisposing factors. One attack does not confer immunity.

There are some reasons for believing this to be an atypical form of malaria. It occurs in highly malarious localities, where, according to Bose, swelling of the nasal mucosa is a common symptom with fever. As against this theory, the malarial parasite has not been found in all cases and quinine is said not to be effective.

Treatment is symptomatic. Puncture of the swollen septum is said to give great relief to the local and general symptoms.

MALTA FEVER.

Definition.—Malta fever, or, as it is sometimes called, "undulant fever," is a disease which is comparatively common in the island of Malta. The malady occurs not only in Malta, but along the shores of the Mediterranean Sea, and so is sometimes called " Mediterranean Fever." When it occurs at Gibraltar it is called " Gibraltar Fever " or " Rock Fever," and when in Italy, " Neapolitan Fever." The malady is due to an infection by the *Micrococcus melitensis*. Its chief clinical characteristic is wave-like or undulant curves of febrile movement. There are also recurring exacerbations of fever with profuse sweats, pains in the limbs, swelling of the joints, and enlargement of the spleen.

History and Geographical Distribution.—Although the first accurate account of this disease was published in 1861 by J. A. Marston, who described it under the name of Mediterranean, remittent, or gastric remittent fever, it probably has been endemic in the islands and along the shores of the Mediterranean Sea for centuries. Hippocrates described cases of continued remittent fever which in their entire symptom-complex correspond with certain manifestations of undulant fever, and references to a protracted form of fever prevailing in Mediterranean countries were made by writers of the eighteenth century and by Sir William Burnett and Dr. Hennon early in the nineteenth century. While the disease is most common at Malta, Gibraltar, in the Balearic Islands, in Cyprus, in Crete, and along the southern coast of Italy, evidence is constantly being produced to show that it has a wide distribution throughout tropical and subtropical regions. Cases of undoubted authenticity have been reported from China, India, Porto Rico, and the Philippine Islands, and a fever occurring in Venezuela closely resembles it clinically, although no cases in which the specific organism has been obtained in cases in that country are on record. Recent investigations have revealed its presence in the United States, but all of the patients seem to have contracted the disease in some one of its endemic foci. The first case was reported in 1898 by Musser and Sailer, in Philadelphia, eight cases have been treated in the Army and Navy Hospital at Hot Springs, Arkansas, and another has recently been reported from Texas by Major C. F. Mason, of the U. S. Army.

Etiology.—The pathogenic organism of Malta fever is the *Micrococcus melitensis*, which was discovered in 1887 by David Bruce, who isolated it in pure culture from the spleens of nine patients who died, and found it in two instances in blood drawn from the spleen during life. This microbe is a minute, round or oval coccus, staining readily with the aniline dyes, but not by Gram's method. It grows very slowly in bouillon, agar-agar, and gelatin, and agglutinates when placed in blood serum drawn from individuals affected with the disease. A joint commission, appointed by the British Government, has proved it possesses great vitality, for the organism was found alive after sixty-nine days in dried, sterilized manured soil, after eighty days on dried fabrics, after seventy-two days in damp soil, and after thirty-seven days in sterilized water. When injected into monkeys it produces a malady similar to Malta fever, and its specific action in the human subject has been demonstrated by several cases of accidental inoculation. Some investigators believe that this organism gains entrance to the body through the alimentary organs, others think that it enters through the respiratory tract, while still others are of the opinion that mosquitoes are the agents of its dissemination. Monkeys have been infected at the will of an experimenter, when forced to breathe an atmosphere laden with dust containing the specific organism. Horrocks has shown that in regions in which the disease is prevalent, milch goats are often infected and that they may excrete the micro-organism in their milk, so infecting human beings who partake of it. Malta fever is a disease of summer, being most prevalent in June and July. Persons of all ages are subject to it, although the period of its greatest incidence is said by Maltese physicians to be between the sixth and the thirtieth years. Sex appears to be without influence in its causation. One attack appears to confer immunity, at least for a number of years.

Pathology.—The gross morbid changes observed after death vary somewhat according to the stage of the disease in which death occurs. In those cases which die during the first four weeks of the attack, the spleen is invariably congested and enlarged and often is so soft that it resembles a large mass of clotted blood. The meninges, the liver, the stomach and intestines, and the kidneys are also frequently congested, and the lungs are always congested at their bases, while in some cases lobular consolidation takes place. The heart occasionally shows granular or fatty degeneration and in a few instances pericardial effusion occurs. In cases which die late in the disease there is evidence of a prolonged toxic action upon the tissues. The liver and spleen are larger than normal and of firm consistency, due to the formation of fibrous tissue, and the heart is usually pale, its walls are thin and its cavities dilated. The spleen is the only organ which shows characteristic microscopic changes, namely, an increase in lymphoid tissue and the presence of large numbers of the specific micro-organism. Sections of the liver and kidneys show granular or fatty degeneration. As to blood changes there is a reduction in the number of red cells, alterations in their size and shape, and a deficiency of hæmoglobin. The white cells are often relatively increased, the basophiles being in excess.

The incubation period is from three to twenty days, most commonly fifteen.

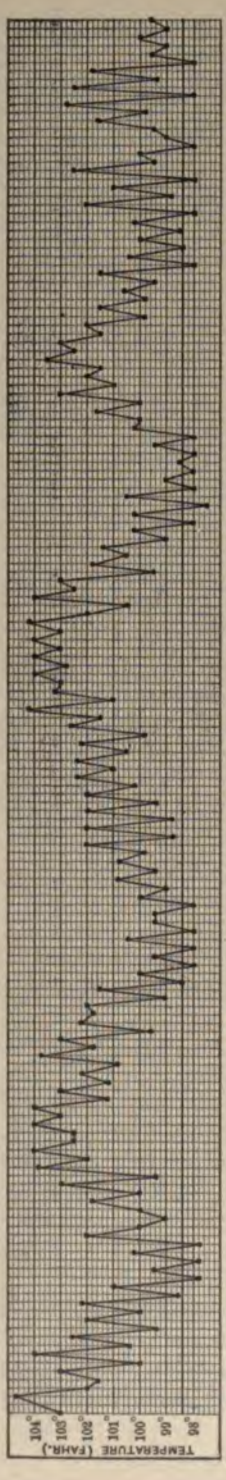
Symptoms.—The onset of the typical or undulant form of the disease is gradual, and is attended by *lassitude, anorexia, nausea, headache, insomnia,* and *slight evening elevation of temperature.* As the morbific process advances the digestive and nervous symptoms become intensified, and the temperature rises slowly day by day, remitting somewhat each morning, until it reaches a level varying from 103° to 105°. Here it is maintained for a varying period of time, and then falls slowly with profuse sweating. The other symptoms abate simultaneously. Soon, however, the *temperature rises again* and a condition similar to the one just described supervenes, constituting a *relapse, of which several occur.* The most noteworthy symptom of Malta fever is therefore the *persistent recurrence of febrile movements* which are wave-like in character, and which last from seven to twenty-one days. They are followed by a period of apyrexia or of very moderate fever, which lasts for a few days, when the febrile movement returns as before. In this manner the *disease may persist for months,* not being self-limited, as is the course of typhoid fever. The active fever, the profuse sweats, and the pain continuing for so long a period produce great exhaustion and emaciation. Sometimes cardiac or pulmonary complications arise which determine a fatal issue of the malady.

In the majority of cases of this type, however, convalescence ensues, but it is slow and often attended by neuralgic and rheumatoid symptoms, and occasionally by orchitis.

Variations from the typical form of the disease are not at all uncommon Some cases are characterized by rapid onset and the early development of severe constitutional symptoms, which usually end in death, while other cases run an extremely mild course with little constitutional disturbance other than general malaise and slow but progressive anæmia and emaciation. In this form the temperature is often intermittent, rising several degrees each afternoon and falling to normal or nearly normal the next morning.

Diagnosis. — Accurate diagnosis of Malta fever depends upon the agglutinative serum test between the blood and the micrococcus, which should be made whenever possible. This reaction is almost always obtainable by the sixth day after the development of pyrexia and often as early as the fourth. F. J. A. Dalton, of the British navy, finds that trustworthy re-

FIG. 41



Temperature chart of the undulatory form of Malta fever. (Hughes.)

sults are obtained by the use of a dilution of 1 : 50, with a time limit of half an hour.

Clinically, diagnosis presents many difficulties, for the different manifestations of the disease make it particularly liable to be confounded with a variety of affections, such as typhoid fever, tuberculosis, chronic rheumatism, malaria, and malarial cachexia. Suspicion as to the nature of typical cases should be aroused by the presence of an undulating temperature curve and the characteristic frequent relapses. Additional aids to the differential diagnosis may be named as follows:

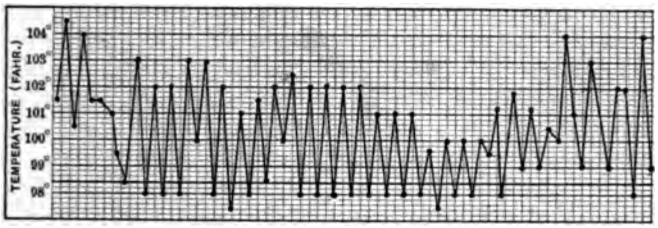
From malarial fever we can separate Malta fever by several factors which make differentiation possible. In the first place the absence of the plasmodium of malaria in the blood, and the presence of the *Micrococcus melitensis* in the spleen on puncture is of course a definite means of separation. Again, the fever does not yield to quinine as does that of malaria, and the pyrexia is too persistent for the intermittent type of that disease, although at times the waves of fever may be abrupt enough to resemble it. Again, the marked arthritic symptoms and the neuralgic pains are not met with in malarial infection.

The possibility of tuberculosis must be also considered. Careful physical examination of the thorax and abdomen will usually reveal signs of tuberculosis if it be present, and if need be the tuberculin test can be applied. Typhoid fever presents a temperature range after the first few days which does not resemble that of Malta fever, and the absence of rose spots in Malta fever is also an important differential point. The presence of the Widal test will also aid in the differentiation.

Duration and Prognosis.—The average duration is from seventy to ninety days, although some cases last as long as six months. Prognosis as regards life is favorable, the mortality being about 2 per cent. From the evidence thus far accumulated it seems that the serum test may afford prognostic as well as diagnostic information, for it has been observed that those cases in which the agglutinating power of the blood serum is high during the early stages of the fever run a favorable course, while those in which the agglutinins remain low during the entire course of the fever are subject to many relapses and a protracted convalescence. A continuous rise with improving clinical symptoms indicates approaching convalescence (Bassett-Smith).

Complications.—The principal complications are hyperpyrexia, cardiac failure, and pulmonary congestion. Pleural and pericardial effusions sometimes occur, and persistent vomiting has been observed by Hughes, who regards it as a very dangerous complication.

FIG. 42



Temperature chart in the intermittent form of Malta fever. (Hughes.)

Treatment.—Malta fever has to be treated solely on the expectant method, for there are no specific remedies. The diet should be nutritious and supporting. Dalton, of the English navy, deprecates the practice of keeping Malta fever patients on liquid food. All his patients who do not have an evening temperature higher than 103° are put on solid food, such as eggs, bread, and rice, and in addition receive two or three pints of milk a day. If this diet is well borne, it is supplemented in the course of a few days by fish or meat. In severe cases with high temperature, foul tongue, and diarrhœa, nothing but liquid diet is given. Dalton also believes that patients whose temperature keeps below 102° are benefited by being allowed to sit up part of the time, it being necessary, of course, to have them avoid exertion and not remain up too long. The bowels should be carefully regulated. Cold spongings with friction should be used to reduce fever and the kidneys be kept active by mild diuretics. When the patient is strong enough to travel, he is greatly benefited by change of climate. During the early stages of convalescence he should receive inunctions of oils and cocoa-butter, get plenty of fresh air and sunshine, and receive aids to digestion, with iron to overcome his anæmia, which is always marked.

BERIBERI.

Definition.—Beriberi is sometimes called *Endemic Multiple Neuritis*, and is a specific infectious disease occurring in nearly all tropical and subtropical countries. The disease is endemic, is associated with marked evidences of peripheral neuritis, with sensory and motor palsies, and profound alteration of the motor mechanism of the heart.

History.—The recognition of beriberi as a distinct morbid entity is almost as old as recorded medicine. The first mention of it was made by Strabo, who describes the development of this disease in the Roman armies operating in Arabia (24 B.C.). It is also described in the medical writings of the Chinese of the second and seventh centuries. It has occupied an important place in the histories of all colonizing powers. Dutch and, later, British observers recognized its specific nature in the beginning of the nineteenth century. Our later knowledge of the disease, particularly the recognition of the specific pathological lesions, is due to the labors of the German teachers, Scheube and Baelz, in Japanese universities.

Distribution.—Speaking generally, the centres of greatest intensity of beriberi are the Malayan countries. It devastates the coolie mining camps and the plantations of the Malayan peninsula. It has been a veritable scourge to the armies of England in India, and of Holland in Sumatra. It is always present in Japan, and in portions of the Philippine Islands it is very common and was the scourge of Aguinaldo's armies. It has occurred in widespread epidemics throughout the whole tropical and subtropical zones. It is found in the principal parts of the world and has appeared as an asylum disease in many temperate regions. In the United States beriberi has been encountered with considerable frequency in the principal ports. It has been observed in asylums for the insane in Alabama and Arkansas. Birge and Putnam have encountered it among the fishermen on the Grand Banks. It seems very likely that beriberi occurs quite frequently in Louisiana. Within recent years it has been observed among Chinese fishermen from the Alaskan coast; by Currie and by Bailey in a settlement of Japanese at Cumberlands, British Columbia; in New Bedford whalers, and in several Dutch ships entering our ports.

Etiology.—In studying the etiology of beriberi, the most striking fact is the part that the general hygienic surroundings play in the development of the disease. The chief factor is overcrowding. Where natives are crowded together, as in jails, ships, and barracks, particularly under poor hygienic surroundings, with an insufficient or poorly selected dietary, in the presence of considerable heat and moisture, the conditions are very favorable to the development of beriberi.

The climatic conditions favoring the spread of beriberi are increased heat and increased humidity. Consequently, in tropical countries the greater number of cases are observed during the seasonal rains. In the Malayan peninsula Wright has observed the greatest number of cases during the prevalence of the southeast monsoon, during which season the relative humidity is at its highest. The disease is also observed with great frequency when these conditions are artificially maintained, as in the crowded, excessively heated and poorly ventilated forecastles of ships carrying Lascar or Coolie crews. Beriberi, as a rule, is a disease of lower levels and ceases abruptly at 3000 feet above the sea, although even in this important point exceptions have been noted.

With regard to the specific etiology of beriberi much confusion has existed. Two principal views are held: The first, and the one to which all the later evidence points as the correct one, is that the disease is due to a specific germ infection, and, second, that the disease is a nutritional disorder consequent on either deteriorated or deficient food, or a nitrogen starvation.

Gelpke, Miura, and Grimm have advanced the theory that in various ways fish were the infecting agents or carriers of the infection of beriberi, the various theories ranging from the latency of infected or spoiled fish to the specific statement that beriberi is due to eating raw fish, particularly a number of varieties of the scomboidæ (Miura) or the infection of dried fish by a species of trichina (Gelpke).

The recent exceedingly valuable studies by Wright have definitely excluded the fish theory, since his observations were made among a number of prisoners from whose dietary fish was totally excluded. Ross has recently suggested the idea that beriberi may be due to arsenical poisoning. In pursuing these studies he found arsenic in appreciable quantities in the hair of a number of cases from Penang. Manson has called attention to the very common adulteration of Chinese tobacco with arsenic in order to give a particularly desired garlic flavor. These cases must, however, be considered accidental or coincidences in which laborers in arsenical ores, canned provisions, adulterated tobacco, etc., have added to the symptoms of beriberi a chronic arsenical poisoning closely resembling beriberi. Competent observers have failed to find arsenic in the great majority of cases.

The greatest conflict has always been waged over the theories connecting beriberi with rice and rice-eating. The disputants are divided into two classes, one of which considers the disease as a toxæmia due to a fungus poisoning of the rice, placing it in the category of diseases like chronic maize intoxication, or pellagra. The second class assumes that with rice as the bulk of an inadequate dietary the disease develops as the expression of nitrogen starvation. It is indeed curious to see how universal is the belief, in countries and among peoples where beriberi is endemic, that the disease is due to bad rice. In the Philippines the sailor or convict suffering from beriberi not only ascribes his condition to rice, but will confidently point out the particular rice which has brought him to his evil state. There is very much that is significant in the facts adduced in support of this theory. The disease is known to affect almost entirely the rice-eating peoples, and it is carried by these people into new regions. Perhaps the most suggestive of all facts in the etiology of beriberi is the extraordinary decrease in the cases occurring in the Japanese navy and army coincident with an improved dietary, in which a large part of the rice component of the nation was substituted by bread and meat. But it must also be remembered that at the same time the most pronounced advances were made in Japan in general hygienic and sanitary reforms, and a large part of the good results must be ascribed to these.

As against the theory that rice diet involving nitrogen deficiency is the cause of the disease we have a host of valuable observations. In the Dutch armies dietary changes produced no mitigation of the disease. Wright in his observations in Kwala Lumpor Jail, in the Federated Malay States, kept the prisoners on a dietary in which there was a decided preponderance of nitrogenous elements with the same incidence rate of the disease. Finally, in this country Ashmead in New York and Daland in Philadelphia have observed the disease in sugar ships that had no rice aboard. Nevertheless it must be conceded that nutritional disturbances which are very commonly present in rice-eating peoples have a distinct predisposing influence to the development of the disease. With regard to the theory that smutted or infected rice is the cause of the disease, exhaustive examination of raw rice has revealed nothing but *Aspergillus niger* and *Bacillus subtilis*. In order to exclude this factor the rice has been boiled and prepared under two or three atmospheres of steam pressure, thus ensuring the destruction of any germ or toxin with entirely negative results as to the prevention or diminution of the disease.

Other observers have connected the disease with various intestinal parasites, uncinaria and strongyloides intestinalis, or with hæmic plasmodia (Glogner), but these have all been shown to be accidental complications, not causes, and due to the prevalence in given sections of either intestinal parasites or the malarial plasmodium along with the beriberi.

There remains, then, the theory that the disease is due to a specific germ infection. The studies of Wright have given a very strong evidence in this direction. The statement of Wright's views is best given in his own words: "The theory of the causation of beriberi that fits the facts is that beriberi is due to a specific organism which gains entrance to the body *via* the mouth, that it develops and produces a toxin chiefly in the pyloric end of the stomach and duodenum, and that the toxin, being absorbed, acts atrophically in the peripheral terminations of the afferent and efferent neurons. Further, that the specific organism escapes in the feces and lodges in confined places through accident or careless personal habits of those affected by this disorder, and that in the presence of congenial meteorological, climatic, and artificial conditions of close association from overcrowding, the organism becomes virulent and, gaining entrance to the healthy body in food, etc., contaminated by it, gives rise to an attack of the disease."

This theory of the disease is also borne out by the peculiar epidemic extensions of beriberi and by the well-known infection of places, separate dwellings, particular ships, and certain wards or barracks in jails or camps. Wright has clinched his arguments by infecting monkeys, and also by producing typical beriberi in them by placing them in the cells with beriberi cases. I (Kieffer) have seen a typical case of beriberi in a monkey which was fed by a Filipino convalescent from beriberi, and have produced unmistakable beriberi in other monkeys in the same way. In support of the idea that the contamination is carried by the feces, the universal lack of any hygienic precautions in defecation or disposal of the fecal matters of the sick in tropical countries must be remembered. Also, that cleanliness of the anal region is very much neglected. When we finally remember that these peoples almost universally eat with their fingers, that the fingers are almost certainly contaminated with fecal matter, and that the custom is to eat out of a common bowl, these theories gain very much more weight. To complete the observations on the etiology of beriberi it must also be remembered how often beriberi has spread to the proportions of an epidemic in tropical countries following an epidemic of cholera.

Frequency.—Beriberi is observed much more frequently in males than in females; not because there is any greater susceptibility in the male, but because women form a comparatively small number in Coolie camps, jails, prisons, and ships. Given an equal degree of exposure, there is no reason to believe one sex more resistant than the other. With respect to age, beriberi may be said to be a disease of early adolescence and adult life. Although it is true that cases have been observed in infants in the endemic areas, yet the occurrence of beriberi in children under ten years is very rare. The great majority of cases occur between the twentieth and fortieth year, although it must also be remembered that a large proportion of those exposed to the disease are young adults.

Beriberi is chiefly a disease of the yellow races, occurring principally in emigrants. At the present day beriberi is very rarely observed in Europeans and North Americans. The white races enjoy a nearly complete immunity, due in part to the better hygienic conditions under which they live.

The Prophylaxis of Beriberi.—When the disease breaks out in ships, jails, asylums, or barracks, these had, whenever possible, better be temporarily abandoned. They should then be thoroughly disinfected and whitewashed or repainted. Old woodwork should be scraped and painted or torn out. In ships the bilges should be cleaned. If dampness exists from deficient drainage it should be remedied. Ample ventilation must be provided. The dietary should be revised, and wherever possible beans or meal substituted for rice, and a sufficient amount of fresh animal food allowed.

Pathology and Morbid Anatomy.—When death occurs rigor mortis is slight and of short duration. If the case be recent and acute, or if death be due to cardiac paralysis, the cadaver bears all the evidence of intense dyspnœa and cyanosis. The eyes are staring, the conjunctiva suffused with blood, the cervical veins tumid and full, the lips covered with bloody froth. In the atrophic cases there is very considerable wasting. In the dropsical cases the effusion is commonly associated with huge thoracic, pericardial, and abdominal dropsies and œdema of the lungs. In acute cases marked congestion of the pyloric end of the stomach and of the duodenum with punctiform hemorrhages is found. The duodenal glands are swollen and congested. Wright considers this to be the specific lesion of the disease, and states that it is always found when the case ends fatally within three weeks of the beginning of the disease.

The liver and kidneys are enlarged, hyperæmic, and show cloudy swelling. The spleen is enlarged, but otherwise shows no characteristic changes. The heart is enlarged, the principal changes being found in the right side. The ventricle is both hypertrophied and dilated. Microscopically there is usually marked fatty degeneration of the myocardium. The intrinsic nerve cells of the heart show marked atrophic changes. The terminations of the vagus are also atrophied. The trunk of the vagus is not involved in early cases, but in later cases the trunk of this nerve as well as the splanchnics and phrenics are profoundly degenerated. The peripheral nerves show striking and constant changes. These begin with degeneration of the terminal branches of the nerves. Not only the muscular but also the cutaneous twigs, and in advanced cases the main nerve trunks, may be involved. The nerve changes are present in proportion to the extent and intensity of the paralysis during life.

The voluntary muscles show similar changes in the distribution of the degenerated nerves. The muscle fibres undergo fatty degeneration, the striations disappear, the nuclei are enlarged, and the interstitial connective tissue may be increased. The central nervous system shows in the brain and cord congestion of the meninges and brain substance, with increase in the cerebrospinal fluid. Degenerative changes can be found in the posterior ganglia, and sometimes atrophy of the posterior columns is present.

Incubation.—The incubation period of beriberi has not been definitely determined. Pekelharing and Winkler have placed it as being a long one, possibly extending over months. In Wright's study of newly arrived emigrants into infected regions, 78 cases developed within thirty days after arrival and no less than 12 of them within ten days; so that he has no hesitation in saying that the incubation of beriberi is between ten and twenty days. These observations need confirmation.

Symptoms.—Clinically, several types of the disease are recognized. There are *atrophic* cases (paraplegic or dry beriberi), *dropsical* cases (wet beriberi), *acute pernicious* cases, and *mild* or *rudimentary* cases. These classes represent not only variations of type, but variations of intensity.

The disease is usually ushered in by marked prodromata. There is loss of appetite, with severe pain in the epigastrium and oppression in the chest. With these symptoms there is a slight febrile rise. In a few cases rigors, lassitude, mental disturbances, and head pains are observed.

ATROPHIC CASES.—After a few days or weeks the patient notices a slowly increasing *weakness of the legs,* with pain and tension in the calf muscles. There is next a loss of sensation in the soles of the feet. The patient does not "feel" the floor, or feels as though a soft insole had been placed between the foot and the shoe or sandal. This increases until the patient becomes bedridden. As a rule, the palsies are confined to the legs and trunk, sometimes invading the arms, and very rarely affecting the head and neck. The paralyzed limbs rapidly atrophy and areas of anæsthesia and hyperæsthesia develop. Examination shows the superficial reflexes preserved; the deep reflexes lost. The calf muscles and extensors of the legs are extensively wasted. The palsy is nearly always flaccid, may be quite profound, and is more marked in the extensors than in the flexors, resulting in drop-wrist and paralytic equinovarus. The electric muscle reactions are markedly altered. From the beginning there is marked diminution, going on to total, loss of reaction to both galvanic and faradic stimulation.

The sensory symptoms closely parallel the distribution of the motor symptoms, and are even more constant. Spots of *anæsthesia* and *paræsthesia* are formed on the feet, calves, legs, trunk, and arms. Recovery from the condition is extremely tedious. Gradually the areas of hyperæsthesia disappear, sensation and motion return, and in the course of ten to twelve months the atrophied muscles regain their contour.

DROPSICAL CASES resemble the atrophic cases plus marked cardiac phenomena with dropsy. Sometimes the dropsical cases develop from the atrophic. The dropsy begins in the feet and legs and spreads until the whole body is affected. The face and lips are puffy and heavy. The arms, legs, and trunk are pudgy. With this there are marked evidences of cardiac distress. There are *cyanosis* of the lips and fingers, dyspnœa, and a marked sense of oppression in the præcordium. Usually the patient is quite helpless, or, if he can walk, the slight exertion is attended by breathlessness and palpitation, these symptoms being very much increased by effusion into the serous cavities. The urine is greatly diminished, but contains no albumin. After persisting for weeks or months the dropsy may rapidly disappear, with an enormous increase in the urine. As a rule, these cases do not present the extreme grades of paralysis and atrophy that are found in the first type. Yet the absorption of the dropsical fluid will reveal marked wasting that may have been completely masked by the semi-solid appearance of the effusion.

In both types of cases the *cardiac changes* are marked. The pulse rate is usually increased and slight exertion serves to further increase the rapidity to 120 to 140 beats per minute. The heart is enlarged, particularly on its right side. The carotids pulsate violently and in the severe cases pulsation is seen in the jugular veins. Systolic and diastolic murmurs are heard, the murmurs being propagated very widely, sometimes even into the bronchial and femoral arteries. Reduplication of the sounds is frequently heard, and there is equal spacing between the sounds. Despite the forcible cardiac beat and the violent pulsation in the vessels of the neck, the peripheral pulse is remarkably small and weak. All these cardiac phenomena are exceedingly fugitive. Even the most pronounced murmurs and evidences of dilatation come and go with rapidity.

ACUTE PERNICIOUS BERIBERI.—This form attacks, as a rule, the more vigorous adults. It may appear as an acute type from the very beginning, or it may represent a sudden, fatal episode in mild or convalescent cases. Beginning in the ordinary way, the disease advances with great rapidity, so that the man becomes bedridden in a few days. The symptoms of *cardiac involvement* begin early and are marked. When this type develops from the milder forms the change is very sudden. *Palpitation* and *dyspnœa* become more and more severe. The patient gasps and struggles for breath. He complains of extreme pain in the præcordium. He breathes with tremendous, laboring, gasps. The vessels of the neck pulsate violently. The eyes are suffused and staring. A blood-flecked foam collects on the lips; unless speedily relieved the patient dies a most dreadful death. In these cases the urine is notably diminished or even suppressed. Nausea and vomiting are common toward the end.

MILD AND RUDIMENTARY BERIBERI of all degrees are observed. These cases usually complain of pain and tension in the legs, with weakness and numbness. The anæsthetic areas may be very small and sharply marked. The patient usually develops some degree of *cardiac irritability* and *palpitation*. There may or may not be *œdema of the legs*. These cases are important because here and there a very mild case may suddenly develop an acute pernicious cardiac attack. As a rule, however, they clear up completely, rarely lasting over the cool season.

Special mention should be made of the skin symptoms. *Petechiæ* and *herpes* of the lips are very common in beriberi, as is also a diffuse or blotchy redness in the arms and legs. After the very beginning of the disease there is no fever. A marked rise of temperature means a reinfection or the development of some complication.

The *blood* shows no characteristic nor, indeed, any marked changes in beriberi. As a rule, there is a very moderate diminution of the red cells. In severe and long-continued cases the anæmia may become more marked, with some considerable loss of hæmoglobin, the color index being minus. In the average cases there is no change in the white corpuscles. In severe cases a moderate leukocytosis is found. The bacteriological findings are negative.

The *urine* shows very little change. The urea is diminished, and, as a rule, the specific gravity is also decreased. Sugar and albumin are not found. Indican is present in large amounts (Baelz). The urine is diminished in quantity in the pernicious cases, even to *complete suppression*. In the cases of wet beriberi the urine is greatly diminished. The secretion is re-established when the exudations begin to be absorbed. Under these circumstances an unusually large amount of urine may be passed, and this re-establishment of the renal function is a very favorable prognostic symptom.

Diagnosis.—The occurrence of beriberi in its usual form in the endemic area, or in persons hailing from infected regions, offers no diagnostic difficulty. When the cases occur in groups, the symptoms of peripheral neuritis point to nothing else. In isolated cases, however, the diagnosis is not by any means easy, and the distinction between arsenical or alcoholic neuritis and beriberi may be difficult. The presence of œdema is significant of beriberi. The earlier and more decided alterations in the deep reflexes and the palpitating and irritable heart also point to beriberi.

Prognosis.—The percentage mortality of beriberi depends on the pernicious cases. The latter are almost always fatal and furnish certainly 90 per cent. of the total mortality. In the remaining cases the mortality varies widely in different epidemics. Ten per cent. would be a very fair average mortality, although it may run as high as 40 per cent. There is no disease in which prognosis is so uncertain and hazardous. Again and again I (Kieffer) have seen cases I considered practically well, certainly out of all danger, die with rapidity in the appalling cardiac crisis of this disease. The prognosis is favorable, without regard to the extent of the paralytic lesions, in proportion to the integrity of the innervation of the heart. Increase of the urine is a favorable sign. So, too, are return of appetite and sexual desire. On the other hand, increasing irregularity of the heart; equal spacing of the cardiac intervals, the short and long pause becoming equal or nearly so; increasing cyanosis, paralysis of the diaphragm, and diminishing urine are very unfavorable signs. The presence of bronchitis, pneumonia, dysentery, alcoholism, and icterus are unfavorable. Vomiting is as sinister a symptom in beriberi as is black vomit in yellow fever.

Treatment.—Baelz has recommended the use of salicylic acid or the salicylate of sodium in 15 grain doses four times a day. Their value is problematical. As much may be said for the plan of freely purging these patients. When constipation exists in beriberi a mild saline is indicated, otherwise it is not useful. In acute cases the dyspnœa, cardiac distress, and girdle pain are best controlled by morphine hypodermically. This remedy may allay the vomiting.

For cardiac cases, particularly those with dropsy, digitalis is the best remedy. It must be given freely in large doses. The cardiac attacks are best controlled by nitroglycerin or inhalations of the nitrite of amyl. These remedies should always be readily accessible in beriberi wards. If the symptoms of cardiac failure become severe, the patient should be bled. It will frequently be found impossible to bleed at the elbow, under which circumstances the patient should be bled from the jugulars. About 400 c.c. should be drawn. The relief from this measure is prompt but evanescent, but, as Manson says, "the patient is for the time being tided over an acute danger and given another chance."

The patient should be put on a liberal diet scale in which nitrogenous foods and fats form a conspicuous part. He should be kept in a dry, sunny room, and whenever possible should be out of bed in the open air. The best remedy we have is removal of the sufferer from the endemic area. The extreme value of this measure is common knowledge to all tropical practitioners. If the patient cannot be removed from the town or province, removal to another house does a great deal of good, particularly if one on high and dry ground is chosen.

The treatment of the residual palsies is the same as that of any other form of severe polyneuritis.

ANTHRAX.

Definition.—Anthrax is an infectious disease due to the presence of the *Bacillus anthracis*. It is much more common in Europe and in South America than in the United States and England, and affects animals far more frequently than man. While it is possible for one man to convey it to another by contact, the infection in the great majority of instances takes place directly from one of the lower animals. Among animals it is met with most frequently among herbivora, next among omnivora, and least frequently among carnivora. Anthrax is sometimes called malignant pustule, splenic fever, charbon, and carbuncle. The first synonym is unfortunate, for in many cases no pustule is found; the second synonym is incorrect, as in man the spleen is not particularly affected, and the last is equally erroneous, as it is an entirely different state from ordinary carbuncle due to the staphylococcus.

History.—Anthrax as it occurs in man has been recognized for over 2000 years, and as long ago as the time of the Romans it was treated by the cautery. During the seventeenth and eighteenth centuries it was very prevalent. Barthélemy proved in 1823 that animals could be inoculated with it. In 1850 Heusinger published an accurate and exhaustive account of the disease. The bacillus was observed in the blood by Pollender in 1849 and its relation to the disease was more fully worked out by Davaine in 1863. Since then this discovery has been confirmed by many observers, the chief of whom are Pasteur and Koch.

Etiology.—As already stated, anthrax is due to the entrance into the body of the anthrax bacillus. It usually occurs as a result of handling some part of an animal which has suffered from this malady. Of Legge's 211 cases 72 were in workers in wool and 65 were handlers of hides. The infection takes place through some break in the skin, as a rule, and in the great majority of cases the site of inoculation is the hand or forearm, but it may appear on the face and chest. In Legge's series infection occurred in the neck in 84 cases, in the face and head in 77, forearm 16, and in but one case was the finger thought to be the point of infection. Sometimes more than one point of infection is present; but it occasionally happens that no external lesion is to be found, although general systemic infection is manifest. In such instances the bacillus gains access to the body by being inhaled in dust, or by being swallowed in milk or other food. Rarely infection of an external wound takes place by the transference of the bacillus by flies from an infected animal to the break in the skin of man or animal.

Prevention.—The disease can be prevented in man by forbidding workmen to handle raw hides or infected animals if they have any superficial wounds, by the use of respirators designed to prevent the inhalation of dust laden with the bacillus of the disease, and by the disinfection of wool, hair, rags, and other articles of commerce which may convey the infection

All animals suffering from anthrax should be killed and then destroyed by burning. Mere burial is insufficient, for it is claimed that earth-worms are capable of carrying the bacilli to the surface and so causing the reinfection of healthy animals. When incineration is impossible burial in quicklime may be resorted to. It is needless to add that the utmost care must be exercised by physicians and nurses in dressing cases of this disease when it occurs in man.

Frequency.—In the United States anthrax is not a very common disease even among sheep and cattle, and is rarely met with in man. Inquiry among employers of men who handle raw hides in Philadelphia develops the fact that it is very seldom met with, and when it occurs is nearly always the result of handling imported hides.

Pathology and Morbid Anatomy.—The changes in the skin produced by primary external anthrax will be described later under the head of Symptoms. When systemic infection occurs as the result of either external or internal primary inoculations, very marked lesions of the viscera become apparent. The bronchial glands are generally swollen, and their increase in size may be quite remarkable. The pericardium may be dotted by petechial spots, and its cavity may contain a considerable quantity of gelatinous material. The muscles, including the heart, are dark colored, soft, and flabby. The blood is fluid and dark in hue—sometimes almost black. Clots may be found in the pericardial space. Clear, straw-colored fluid may be present in the pleural cavity, and if the lungs are affected they are found engorged with dark-colored blood, the right lung being more affected than the left as a rule; the posterior portion is most congested and œdematous, particularly at the bases. Sometimes pulmonary infarctions are present.

In the abdominal cavity numerous extravasations of the blood may be found in the mesentery. Petechial hemorrhages may be present in the stomach and intestines. The spleen is usually enlarged and contains a large amount of grumous blood. Microscopic examination of the tissues of the body, when general infection is present, discloses the bacillus usually in large numbers. They are particularly numerous in the small blood-vessels and lymph glands which are near the site of the primary lesion.

Symptoms.—Anthrax occurs in two forms, the *external* and *internal.* The *external* form manifests itself by the development at the point of infection, about three to six days after contact with the source of the disease, of a *small, itching papule,* which is soon surrounded by an inflamed area. Usually this lesion is so insignificant that no attention is paid to it save the scratching or rubbing of it. There may be a history of an abrasion, scratch, or pimple through which infection has occurred. The papule speedily becomes red and angry-looking, and at its summit a *vesicle* develops which is filled with bloody serum. Around this centre of infection, on the reddened and œdematous zone or base, additional papules and vesicles appear and the inflammatory process spreads rapidly in all directions. The vesicles may become dry and crusty, and as they do so the tissues underneath undergo softening, the central part becoming black and necrotic. Curiously enough, this rapid process rarely causes much pain, but the neighboring *lymphatic vessels* become reddened and the *nodes enlarged.* By the end of forty-eight hours after the papule first appears the anthrax bacilli may be found in the blood, and in such a case the symptoms of systemic disturbance rapidly become very marked.

The local lesion rapidly spreads up the arm if the hand be the part first attacked, and the part becomes *intensely swollen* and livid, the skin being dotted by blebs, but it is a noteworthy fact that the rapid spread of the surrounding inflammation is due largely to secondary infection by other organisms. In cases of pure anthrax infection the central papule is often surrounded by an area of induration, but no red areola even after the slough has formed. There is general wretchedness and rapidly increasing debility, followed by *rigors, high fever, sweats,* and *diarrhœa,* but after the early stages the fever falls and the temperature may be normal. Delirium rapidly ensues, and dyspnœa and cyanosis, with profound evidences of septic infection, close the scene in death. In some cases, however, the mind remains perfectly clear.

When recovery takes place the local area is walled off by protective efforts on the part of the body, so that severe constitutional symptoms do not appear. The diseased tissues at the focus of infection undergo necrosis, are thrown off, and healing is finally accomplished.

A second form of external anthrax infection is that which, because of its course, is called *malignant anthrax œdema.* This usually develops on the face or head and differs from the type just described in that no papule or similar local lesion is present, but in its stead an intense œdema of the tissues is produced. So active may be the local process that the parts may speedily slough or become gangrenous. Death usually comes rapidly to such cases.

The *internal* form of anthrax manifests itself in the lungs or alimentary tract. In the first instance the anthrax bacilli enter the respiratory passages by inhalation in the dust arising from the handling of dried hides or wool. Hence it is called "wool-sorters' disease." The symptoms in these cases vary to an extraordinary degree in their severity. In some instances the patient feels wretched and miserable, and soon has a *chill* which is followed by *fever* and very marked *thoracic distress*. There may be pain in the side and labored, difficult breathing. Cough may or may not be present. The face becomes livid, marked cyanosis develops, and the patient dies in a few hours or days in asthenia and collapse. Pneumonia is rare, but areas of impaired resonance on percussion and bronchial breathing may be found as the result of enlargement of, and pressure by, the bronchial and mediastinal glands. In other cases the symptoms are so mild and indefinite as to possess no diagnostic value. A workman may feel only weak and feeble, his hands may be cold, and his breathing oppressed, yet he may die within twenty-four hours in collapse. Bell records several cases in which death came within twenty-four hours of what seemed to be perfect health.

When the intestinal tract is infected, there are present *diarrhœa, vomiting, great weakness*, and *failure of the circulation* followed by *collapse* and death in from two to five days.

Diagnosis.—In the external form of the disease the occupation of the patient and the presence of an itching papule should at once arouse the suspicion of anthrax infection, which will be strengthened by the rapid formation of the vesicle already described. The diagnosis can be confirmed by a microscopic examination of the fluid for the bacillus, or by inoculating a mouse with one drop of the fluid from a vesicle. This will cause the death of the mouse in about forty-eight hours, and in its organs the bacillus will be found in immense numbers, and from these cultures may be made.

This condition is separated from carbuncle by the lack of pus and by the absence of its sloughing core. From erysipelas of the phlegmonous type, or from diffuse cellulitis, it is separated in the later stages by the absence of pain and fever. From malignant œdema it is distinguished by the absence of crepitation due to gas in the tissues. Agglutination tests based on the same principle as the Widal test in typhoid fever have not been generally adopted.

Prognosis.—The prognosis of the external form depends upon the degree of general systemic infection, and therefore the size of the local lesion has not any great importance in determining the outlook It not rarely happens that a small papule may be followed by the death of the patient in a few days, whereas a larger lesion may be recovered from. Thus, Bell states that a patient with so severe a lesion on the face as to have large bullæ and a free discharge of straw-colored fluid, with swelling of the entire head and the submaxillary glands, may recover. In other words, everything depends upon the degree of systemic infection. A rapid-running pulse is always an evil omen.

Death may come as early as the first day of illness, but the majority of deaths occur on the fourth to seventh day. So far as mortality is concerned statistics vary very greatly, probably because of variations in the virulence of the infection. Thus, Woolmer states that out of 50 cases he lost only 2, and Muskett treated 50 cases with one death; whereas in England, even when the workmen have been taught to present themselves for treatment at once, the mortality has been 21 per cent., and in some collections of statistics it has reached 50 per cent. In Eppinger's epidemic among rag-pickers 78 out of 88 cases were fatal.

In the internal form the prognosis is bad and death often comes, as already stated, as early as twenty-four hours after the primary symptoms. Cases in which recovery has taken place are, however, on record.

Treatment.—The treatment of the external form consists in the destruction by actual cautery of the primary focus of the disease at the earliest possible moment. If this is not done it should be excised. Not only should the infected tissues be removed, but the surrounding tissues for at least an inch as well. As soon as this is done the part is to be swabbed with pure carbolic acid and then dressed, so that drainage into the dressings may occur. The patient's vitality should be maintained by good food and stimulants, and anthrax antitoxin should be given.

Within the last few years an anti-anthrax serum has been employed with success. Legge states the following facts as to its use: (1) In very large doses it is innocuous; (2) it can be well borne even when introduced into the veins; (3) no case taken in an early stage, or of moderate severity, is fatal if treated with serum; (4) with the serum some cases are saved when the condition is most critical and prognosis almost hopeless; (5) when injected into the veins the serum quickly arrests the extension of the œdematous process so as to reduce notably the danger from suffocation which exists in many of the cases where the pustule is situated on the face or neck; (6) the serum, if used early enough, reduces to a minimum the destruction of tissue; (7) in some situations of the pustule, as the eyelid, serum must be used in preference to any other treatment; (8) persons attacked, when treated with the serum, appear to become convalescent in the course of a few hours; to these I may add (9) that in internal anthrax if it is administered intravenously it is the only treatment which can hold out any hope.

In almost all cases injection of the serum is followed by a rise in temperature often to over 105°, and with this there is an improvement in the general condition of the patient. The prognosis where there is this rise Sclavo regards as favorable. In the same way the necrotic process itself is to be regarded as a sign that the organism is making effort to resist the anthrax infection.

HYDROPHOBIA.

Definition.—Hydrophobia is an acute infectious disease of animals communicable to man, the specific cause of which has not as yet been isolated. It is characterized by great restlessness and delirium, by an apparent dread of water in some instances, and by delirium and paralysis in its later stages. It is often called "*rabies*" or "*lyssa*."

History.—Hydrophobia was well described as long ago as nearly 500 B.C. by Democritus, but not until about 200 years B.C. was it described in man. Since then it has been discussed by many ancient and modern writers, of whom Grüner, in 1813, found that the saliva was the vehicle of infection.

Trousseau wrote its best description in modern times in 1850. In 1882

Pasteur first clearly discussed the cause of the disease and devised a plan of rational treatment.

Distribution.—No less an authority than Virchow believed that hydrophobia was not to be met with in Greenland, Denmark, Africa, and parts of Asia and South America, and with others claimed that it is peculiar to temperate zones; but in 1860 an epidemic occurred in Greenland among animals when the temperature was 25° below zero. Boulanger is probably correct in stating that no part of the world is free from it. The idea that it is more prevalent in summer than in winter is erroneous. Suzor has reported twice as many cases in animals in March, April, and May as in the summer months. Two-fifths of all cases in human beings are under fifteen years of age. The disease affects dogs, skunks, foxes, and wolves more commonly than other animals, but all animals are apparently susceptible to it. Cows may develop it from dog-bites or from bites of other infected cows, and in 1888 I saw a number of deer from the royal herd in Richmond Park, near London, which suffered from this disease and which were studied at the Brown Institution.

Etiology.—As already stated, the cause of this disease has not been determined with certainty, although recently Negri claims to have established that it is due to a protozoal organism. It is known that its cause has a special affinity for the nervous system, and is found in the saliva, but not in the urine or the blood. The disease can be passed from animal to animal, from an animal to man, and from man to an animal, and it can be passed on from one to another without rapidly losing its virulence. The transfer is always made through some solution of continuity, usually a bite, but it has occurred through a pimple, and it is said to have been transferred by the milk of a nursing mother.

In the dog it has been proved that the saliva may be virulent as long as three days before any symptoms of the disease appears.

Prevention.—The only efficient measures of prevention are the universal muzzling of dogs, particularly when a mad dog is known to have been in the neighborhood, and the killing of all animals found suffering from the disease. In London the muzzling of dogs decreased the disease from 176 cases in dogs in 1889 to 3 in 1892, but on relaxation in enforcing the law the number of cases in dogs and man rose again to about the original number. (For preventive inoculation see Treatment.)

Frequency.—Hydrophobia is not a common malady in animals, and is rare, comparatively speaking, in man. Sporadic cases are met with in animals in every large city during the year. (For statistics see Salmon's article in the *Year Book of the U. S. Department of Agriculture,* 1901.) Woodhead states that only about 16 per cent. of those bitten by rabid animals become victims of the disease.

Pathology and Morbid Anatomy.—It has been generally stated that there is nothing pathognomonic in the morbid anatomy of rabies, but recent studies controvert this view. Examined microscopically the medulla and spinal cord show small hemorrhages and large numbers of small round cells in the perivascular lymph spaces and around the motor ganglia cells, and progressive degenerative changes in the spinal nerve cells appear, consisting in chromatolysis and overgrowth of the nucleolus. These changes are, however, by no means pathognomonic, as they may be found in other diseases.

Van Gehuchten and Nelis have, however, discovered changes in the peripheral, cerebral, and sympathetic ganglia, in the intervertebral ganglia, and in the plexiform ganglia of the pneumogastric nerve, which they consider to be diagnostic of rabies. These changes consist in the destruction of the nerve cells by newly formed cells from the capsule. Ravenel and McCarthy have studied (1901) 28 cases of rabies occurring in dogs, rabbits, and other animals, and found these changes in all but one case. The plexiform ganglia were affected in all in which positive results as to the presence of the rabies were obtained. In 21 cases the bulb was examined for the rabic tubercle of Babés, and positive results were obtained in 19, although in 2 no distinct tubercle formation was observed, chromatolysis only being present. The rabic tubercle of Babés is an accumulation of embryonal cells around the nerve cells. The cells of the bulbar nuclei undergo degeneration and manifest various stages of chromatolysis.

As a result of their studies Ravenel and McCarthy conclude that these changes, taken in connection with the clinical symptoms, afford a rapid and trustworthy means of diagnosis of hydrophobia, but that their absence does not imply that the disease is not present. They also believe that the rabic tubercle of Babés is present often enough to afford diagnostic evidence in cases where the central nervous system only is obtainable without any ganglia.

Symptoms in Animals.—The symptoms of rabies in animals vary greatly. In the dog we find that he is at first stupid and heavy and often cross and restless. When he stands up he may sway slightly and stagger when he runs. At this time he is easily frightened and his reflexes are acutely increased. He usually refuses food and drink, but will often gulp down all sorts of substances not food, such as rags, manure, and pieces of wood. Even at this time he may be obedient, and may not bite his master, although he will snap at a stranger. The bark is muffled and peculiar, and may be a series of yelps or howls, the lower jaw never completely closing as in health. Thirst may be manifest, but though the animal may lap the water, spasm of the throat prevents swallowing. The idea that a mad dog has a peculiar dread of water is erroneous. Any repulsion he may have to it is due to the spasm of the gullet.

He next becomes delirious and maniacal, galloping or swiftly trotting, with a slouching demeanor, as if shrinking from some enemy. The jaws are usually open, and the saliva may flow freely from the mouth. Some amblyopia may develop so that he is prone to run into objects which should be easily avoided. This may, however, be due to stupidity or muscular inco-ordination. Sometimes he seems to see imaginary objects and snaps at them. Rapid emaciation is a noteworthy symptom. Finally, the animal becomes more feeble and paralyzed. The paralysis is gradual in onset. The hind legs are at first moved with difficulty and finally the animal sinks on his haunches, there being a simultaneous loss of power in the forelegs, upon which, however, he can occasionally raise himself. During this period convulsions may ensue. Death occurs on the fifth or sixth day as a rule, but life may be prolonged until the eighth day, but never longer than the tenth day.

In some cases, probably in those which are very severe, the paralysis may develop almost at once.

Animals sometimes manifest symptoms of what is known as "dumb rabies," which is to be distinguished from the maniacal form. In this condition the lower jaw is dropped and the animal is unable to close the mouth. The tongue hangs out and the saliva dribbles. As the jaw is paralyzed the dog is unable to bite, and does not attempt to do so. This form usually causes death in about two to four days. It is important that these manifestations of dumb rabies be remembered. Dr. Gill, a veterinary surgeon of New York, asserts that this is a very common form of the disease, which frequently misleads persons into thinking that the dog has a bone in its throat. They are still further deceived by the fact that the animal has no hydrophobia and not infrequently actually plunges his head into water, or will even swim a river. He adds as additional advice: "Beware of a dog when it becomes dull and hides away, appears restless, is always on the move and prowling, whose countenance is sombre and sullen, and which walks with his head down like a bear. Beware of one which barks at nothing when all is still. Beware of the dog that barks incessantly and tears up things. Look out for the dog which has become too fond of you and is continually licking your hand and face; and beware, above all, of the dog which has difficulty in swallowing, which appears to have a bone in its throat, and of one which has wandered away from home and returns covered with dirt, exhausted and miserable."

These symptoms in the dog have been described in detail because a correct diagnosis of the malady in the dog is of vital importance in determining whether a patient is to be a victim of rabies and if he should be given Pasteur's treatment.

Symptoms in Man.—When a human being is infected by rabies the stage of incubation may last from fourteen days to eighteen months, although if the animal produces a punctured wound and is in the active stage of the disease the shorter period is more common. It is usually shorter in young children than in adults. At the end of the period of incubation the part infected begins to itch and tingle and then to burn. The skin in its neighborhood may develop vesicles, and the old wound may open.

The primary systemic symptoms in man are *apprehension*, *restlessness*, and finally *marked anxiety*. This is followed by thirst, but when the water is brought near the patient he seems to have great fear of it—hydrophobia. This fear is chiefly due to the *pharyngeal spasm*, which is produced at the sight of water, which, if the patient tries to swallow, becomes exaggerated. This spasm is the most pathognomonic symptom of rabies in man.

Often the site of the inoculation is red and inflamed and there may be local irritation or pain. This stage lasts about five days and is followed by the stage of excitement, with labored respirations and spasm of the laryngeal and pharyngeal muscles. The reflexes are greatly exaggerated and delirium or mania may come on. Occasionally the jaws may be snapped together, although snapping is said to be characteristic of false rabies. Very commonly the curious symptom of spitting develops, the patient ejecting small quantities of spittal upon surrounding objects.

Occasionally paralytic rabies occurs in man, but it is very rare.

Diagnosis.—It is only in the early stage of the disease in either the animal or man that any difficulty can exist as to its diagnosis. As the saliva of a dog for several days before it seems very ill is virulent, all sick dogs, ill of unknown causes, should be regarded with suspicion or caution. In man the history of having been bitten will usually be obtainable.

Occasionally a hysterical person, after reading or hearing a description of rabies, develops symptoms which resemble it. The fact that the patient is of this type, and that threats, or inhalations of amyl nitrite, speedily cure the ailment will permit a differentiation. This state is called pseudohydrophobia or lyssaphobia. It is important to remember that symptoms of pseudohydrophobia or hysteria simulating the true disease often develop as early as twenty-four or forty-eight hours after exposure; whereas true hydrophobia rarely develops in less than fourteen days. Again, the hysterical patient often presents the symptoms of the second convulsive stage, without having shown any primary symptoms. He is apt to show a disposition to bite, which is very rare in true human hydrophobia, and if he barks, growls, or snaps it is an imitation and not the true disease. Finally, should the patient survive for a period of active symptoms longer than ten days the case is probably hysteria.

True hydrophobia is to be differentiated from tetanus by the presence of marked lock-jaw in the latter disease, and by the fact that in tetanus there is no dribbling of saliva and no expression of terror. The convulsions in true hydrophobia are rarely as tonic as in tetanus. The paralytic form of rabies may resemble Landry's paralysis.

Prognosis.—It may be possible for true hydrophobia to permit of recovery in man, but, if it is, no case of recovery has ever been reported in which there was undeniable evidence that the diagnosis was correct.

Treatment.—The treatment of hydrophobia is entirely in the line of preventive medicine, for, once the disease is developed, curative measures are not possible. As soon as the bite is received the wound, if a punctured one, should be washed and then sucked and the spittle expectorated. If the part injured be an extremity a tourniquet should be used until this is done. The punctured wound should be converted into an incised wound, and the opening should not be closed, but given free drainage and kept open, well protected from other infection, for several weeks. Where possible, without great mutilation, the part should be excised as is now recommended for the prevention of tetanus. The value of caustics depends on the one employed. Nitric acid is the best. If the wound is incised, well washed with normal saline or with bichloride of mercury, real tissue destroyers may be set aside.

The specific and rational method of treatment is that proposed and instituted by Pasteur by means of attenuated virus. This investigator found that if the virus of hydrophobia is propagated, through the inoculation of a series of rabbits, it increases in virulence, and the spinal cords of the rabbits of the last series of inoculations contain the poison in a very active state.

If these spinal cords are preserved under certain conditions this degree of virulence progressively diminishes. If an emulsion be made from the attenuated cords and a dog inoculated with it in small amount the animal survives, and if successively inoculated with virus of increasing virulence gradually becomes immune as larger doses are given, until he is able to stand inoculation with the most virulent matter obtained from the cords of rabbits. This same process is now employed for the treatment of a man who has been bitten, the endeavor being made to produce an artificial immunity before the stage of incubation following the bite is completed.

Two methods of producing immunity to hydrophobia are now employed. One is known as the "simple" and the other as the "intensive." In both methods spinal cords of infected animals are employed in gradually increasing strength until finally what is known as a three-day cord—that is, one which has been kept three days—is administered subcutaneously. In the "simple" method nineteen injections are given in fourteen days.

In those cases which have severe lacerated wounds of the face, in which not only infection has taken place, but the tissues have been devitalized by traumatism, the "intensive" method is used. This consists in the administration of twenty-eight injections in twenty-one days. During the first three days as many injections are given in the "intensive" method as are given in five days by the simple method.

Out of 17,395 cases treated at the Pasteur Institute in Paris in this manner between 1886 and 1895 there were 139 deaths from rabies. If the average mortality after inoculation is in the neighborhood of 20 per cent., as already stated, it is evident that instead of there being 139 deaths there would have been approximately 3476 deaths, which shows the great value of the plan. As the treatment is absolutely harmless if properly employed, the value of Pasteur's method cannot be doubted. It is, of course, useless after the malady is well developed.

After the disease is established care must be taken that the patient does not wound his attendants. His sufferings should be relieved by morphine or chloral in sufficient doses to spare him from much misery. Nutrient enemata may be used to help support nutrition if food cannot be swallowed.

Efforts have been made from time to time to provide an antirabic serum. Babés and Lepp, in 1889, Tizzoni and Schwarz, and later Tizzoni and Centanni have all endeavored to reach this much-desired product. They have used virus which has been attenuated by digestion with gastric juice, and their serum is of such a strength that in the proportion of $\frac{1}{25000}$ per kilogram weight of rabbit it will protect against one infection unit of virus if administered subcutaneously twenty-four hours before the subdural injection of virus is given to an animal. An infection unit of the virus is the greatest dilution of any virus which will surely kill, without prolongation of the incubation period, when 0.012 c.c. per kilogram of animal is administered under the dura. These investigators believe that the antirabic virus should be injected subdurally, as it is far more efficacious in this way than when it is given by the hypodermic needle, and they claim that the subdural dose is 12,000 times as efficacious as the subcutaneous dose.

Unfortunately, these investigations only give promise of good for human beings. Sufficient statistics as to their value have not as yet been forthcoming.

TETANUS.

Definition.—Tetanus is an acute infectious disease due to the entrance and development in the body of a specific organism, the bacillus of tetanus. It is characterized by the development of rigidity of the muscles so that the limbs are fixed and the jaw locked.

History.—Tetanus has been known for many centuries as a disease that occasionally follows small wounds, but it was not until 1884 that Carle and Rattone discovered that when an animal showed symptoms of tetanus it was possible to produce similar symptoms in healthy animals by injecting virus obtained from the first. In 1885 Nicolaier obtained from the pus of infected animals, bacteria which, when inoculated into healthy animals, caused tetanus, but he was unable to isolate the organism absolutely, although he described it as a small, slender bacillus. In 1886 Rosenbach confirmed Nicolaier's discovery, but he also did not get a pure culture of the bacillus of the disease. In 1889 Kitasato, Tizzoni, and Cattani succeeded in its complete isolation. Faber also proved that he could obtain from a culture of this bacillus a toxin which, when injected into animals, caused symptoms identical with those met with in human beings suffering from this malady.

Distribution.—Tetanus is met with everywhere in tropical and temperate zones. Its bacillus is particularly prevalent in garden soil and about stables and dungheaps. In the United States it is most prevalent in Louisiana, New York, Pennsylvania, Texas, and Ohio in the order named. Wells has shown that the curve of deaths in this disease starts in May, reaches its highest point in July, and then declines to October.

Etiology and Frequency.—The specific organism, the *Bacillus tetani*, is 4μ to 5μ in length and about 0.4μ wide; during sporulation one end enlarges giving the organism a drumstick appearance. This bacillus is an anaërobic, slightly motile, flagellated rod, possessing unusually resistant spores and the faculty of producing a highly poisonous toxin. It is frequently demonstrated in discharges from wounds in cases of tetanus, and has been found on the object producing the wound and in freshly made wounds.

The chief causative factor in tetanus is the presence of a wound through which the specific germ may enter the body. This wound may be so insignificant as to be overlooked. In other cases the infection takes place through a break in the mucous membrane of the mouth. Accidents of this type probably account for the cases of so-called idiopathic tetanus. Small, punctured wounds are much more apt to result in the development of the disease than large ones with free drainage, for the accumulated necrotic tissues of punctured wounds afford approximately ideal conditions for the development of the anaërobic bacillus. Within the last few years several outbreaks of tetanus have followed the use of contaminated vaccine.

Tetanus is not a very common disease, but nearly every large hospital service has presented to it occasionally an isolated instance. It has been epidemic in many hospitals and camps; it has also been epidemic among newborn infants, infection taking place through the umbilicus and causing a frightful mortality, particularly in the West Indies, where at times more than 60 per cent. of all children born died within eight days after birth from its ravages. In this country it is seen in hostlers, gardeners, agricultural laborers, men employed about stables, and in children who run about with bare feet. By far the most common incidence of the disease occurs in children who suffer from wounds produced by toy pistols and fire-crackers. As many as 466 cases of this disease were due to these causes in the celebration of the Fourth of July in 1903 in the United States; but it is interesting to note that owing to the warning issued by medical men against the use of these explosives the number of deaths due to this cause was only 105 in 1904.

Prevention.—Tetanus is to be prevented by the excision or conversion of all punctured wounds into incised wounds with free drainage, by the use of tetanus antitoxin as soon as the wound is received, and, if the disease develops in a hospital or camp, by the careful isolation of those who are ill with it.

The measures taken to destroy the bacillus and its spores outside the body, as in dressings and clothing, must be very radical, because the spores are extraordinarily resistant to those measures usually employed to destroy pathogenic germs. Thus, the spores can survive two hours' exposure to corrosive sublimate 1:1000, and even survive exposure to boiling water if the exposure is brief. So, too, fifteen hours' treatment with 1:20 of carbolic acid is necessary to destroy their vitality. Drying does not kill the bacillus. Miguel has produced the disease from infected soil kept for eighteen years.

Pathology and Morbid Anatomy.—A most important fact to be remembered in regard to tetanus is that the specific organism primarily does not spread through the body, but develops at the site of infection, and from this focus the toxin which produces the symptoms of the malady is disseminated. It has been proved by Meyer and Ransom that the poison passes to the central nervous system through or along the nerve trunks. Another fact of importance is that the toxin combines with the cells of the nervous system with remarkable celerity, and having done so forms so firm a combination that it cannot be dislodged, and in consequence the subsequent use of antitoxin often fails.

The tetanic convulsions are not due to any influence of the poison on the nerves or muscles, but upon the spinal cord and brain.

In cases of death from tetanus there are no characteristic changes in the tissues of the nervous system.

Symptoms.—The symptoms of tetanus are so characteristic that they can hardly be mistaken for any other disease save hysteria and strychnine poisoning. The dominant symptom is the state of *rigidity of the voluntary muscles*, which, when the disease is well developed, are practically constantly contracted, although at intervals they relax and contract spasmodically, causing the well-developed convulsions of the disease. It is a curious fact that the earliest symptoms often emanate from the muscles nearest the focus of infection, but very commonly they originate in the muscles of the jaw and neck, producing the symptom called "lock-jaw," that is, a state in which, by reason of the spasm in the masseter muscles, the lower maxilla is firmly pressed against the upper jaw.

The contraction of the facial muscles in the spasm gives the face a peculiar expression of painful mirth, or *risus sardonicus*, and it is a noteworthy fact that this expression may be the first warning of an oncoming attack of the disease, for as the patient attempts to show his tongue to the physician who is inquiring as to his general health, the physician is startled to see the facial muscles produce this strange expression.

The muscles of the back and abdominal wall are rigid to the touch, and pain and oppression due to *spasm of the diaphragm* may be present when the disease is well developed. The muscles of the hand are the least affected of all the voluntary muscles, as a rule.

If the more powerful muscles contract forcibly the patient's *body is arched*, resting on his heels and the occiput; this is called opisthotonos. If the muscles of the anterior part of the body are the more powerfully contracted he may be arched forward—emprosthotonos.

Pain in the affected muscle is not severe as a rule, but is rather the aching due to prolonged strain and weariness. Sometimes, however, it is severe. There may be alarming *spasm of the glottis* or fixation of the respiratory muscles endangering life, and, indeed, in severe cases, this is the cause of death, particularly when, by reason of exhaustion, the patient is unable to withstand asphyxia for any length of time.

The *mind usually remains clear* till the time of death. The *temperature* is moderate if the convulsions are moderate, and high if they are severe, ranging from 100° to 106°. The *pulse* varies in speed, becoming rapid during a seizure. Finally it becomes feeble from exhaustion.

Diagnosis.—Tetanus rarely is as sudden in onset as is strychnine poisoning, and it very rarely causes death so rapidly. It affects the muscles of the face primarily, which strychnine very rarely does. There is usually a history of punctured wound in one case or of the ingestion of poison in the other. In strychnine poisoning the convulsions are followed by periods of complete relaxation, whereas in tetanus constant spasm with exacerbations are present.

In hysteria the ecstatic facies of the patient, the presence of clonic movements, the fact that the patient is a woman of a neurotic type, and that laughing and crying are often present, aid greatly in the diagnosis. Further areas of anæsthesia are often present in hysteria and inhalations of nitrite of amyl may cause relaxation followed by sobs and tears as the spasm is relieved by the drug.

Tetany rarely presents such severe contractions, but it may do so. The spasms are often localized, and if they occur in children signs of rickets or gastric dilatation may be present. Tetany practically never causes death, and it affects chiefly the hands and feet, which tetanus does not.

Prognosis.—The prognosis in tetanus depends very greatly upon the severity of the paroxysms and upon the virulence of the infection. In virulent infections death comes as early as the second day and usually by the sixth. It is essential that two forms of the disease be recalled in studying this question. There is an acute form with a very high mortality of 80 per cent., and a chronic form in which recovery takes place in a large percentage, about 50 per cent. The mortality is very high in children in all cases.

Treatment.—Before everything else in the treatment of tetanus must be considered the use of tetanus antitoxin. Its value, however, is chiefly limited to those cases in which it can be administered as soon as the inoculating wound occurs, or within a short time after this. Its failure to be of value when employed after the symptoms are well developed is not due to any lack of power on the part of the tetanus antitoxin, but to the fact that the tetanus toxin so rapidly and firmly combines with the nervous protoplasm of the brain and spinal cord that it is impossible for it to be disassociated from this protoplasm, and therefore the antitoxin cannot combine with it and prevent it from damaging the central nervous system.

When children are wounded by means of toy pistols tetanus antitoxin should be injected at once, since the proportion of cases in which tetanus develops from this injury is very large, and by the prompt administration of the remedy the disease may be prevented from producing its characteristic symptoms. Thus in 1903, out of 56 cases of so-called Fourth of July tetanus treated without antitoxin 16 died, whereas in 1904, out of 36 cases treated with antitoxin none died. In 1216 cases of tetanus treated by antitoxin, Packard and Wilson found that the mortality was 42.2 per cent., and Moschowitz in 461 cases treated in this way found a mortality of 40.3 per cent. As the death rate of acute tetanus is about 80 per cent. and of chronic tetanus about 50 per cent., it is evident that antitoxin saves many lives. Even after tetanic symptoms have developed tetanus antitoxin should still be used, as it may be of some value.

A suggestion has been made that in severe cases the patient be trephined and the antitoxin injected by the hypodermic needle between the membranes of the brain or into a cerebral ventricle. It does not seem that this measure offers sufficient promise of usefulness to justify so serious a method of treatment. If the symptoms are very severe, and doubt exists as to the rapidity with which the antitoxin can be absorbed from the subcutaneous tissues, it may be advisable to inject it into the cerebrospinal fluid, inserting the needle between the fourth and the fifth lumbar vertebræ. In this way it will reach the spinal centres quite rapidly without exposing the patient to a surgical operation. The needle should be introduced according to the directions given in the section on Cerebrospinal Meningitis, and proof that it has entered the membranes of the cord assured by the discovery that a few drops of cerebrospinal fluid drip from its external orifice. The syringe containing the tetanus antitoxin is then attached to the needle and the injection is made. According to Luckett it is best to withdraw a considerable quantity of cerebrospinal fluid before injecting the antitoxin.

Still more recently the use of antitoxin injected into the nerve trunks supplying the part of the body through which the infection has taken place has been tried with satisfactory results, the idea being that the infection spreads along the nerve.

The wound, by means of which tetanus infection has possibly taken place, should, if small, be excised, or if it is large it must be thoroughly cleansed first with normal salt solution or some antiseptic liquid, care being taken, if an antiseptic is employed, that it is not used in sufficient strength to in any way interfere with the vitality of the tissues with which it comes in contact, since it is probable that the vitality of these tissues is of greater value in protecting the individual against infection than are the ordinary antiseptic drugs. If the wound is a punctured wound, it should be converted into an incised wound in order that the tetanus bacillus may be thoroughly washed out of it and that free drainage may be provided. This is exceedingly important. Nothing aids in the production of tetanus so certainly as the closure of such a wound in its early stages. Wounds should be allowed to heal by granulation, as this is the surest way to prevent the development of the disease.

After the disease is developed the patient must be fed with nutritious and easily digested foods in order that his nutrition may be maintained. In the rapid type of tetanus death usually comes so soon that great emaciation does not occur. But in the more chronic form the question of nutrition must be constantly kept in mind.

If the jaws are so locked that food cannot be introduced into the mouth, one or more teeth should be removed in order that a stomach tube may be passed, or a rubber tube may be passed by way of the nostril, as in feeding insane patients who refuse to take nourishment. Humphrey has gone so far as to recommend in these cases that a gastrostomy be performed, the tube introduced, and the patient nourished through the operative wound. This seems, however, an unnecessarily heroic method when the tube can be used.

The severity of the tetanic seizures can be to some extent modified by the administration of full doses of chloral and the bromides, which act as sedatives to the motor and sensory portions of the spinal cord. These remedies are, however, in no way curative, but simply symptomatic in that they diminish to some extent the force of the convulsions without in any way influencing the deleterious influence of the poison upon the system.

Care should be taken that the activity of the kidneys is maintained by the administration of mild diuretics and by providing the patient with plenty of water. The state of the bladder should also be watched, as retention of urine is not uncommon. To prevent this, repeated catheterization should be resorted to.

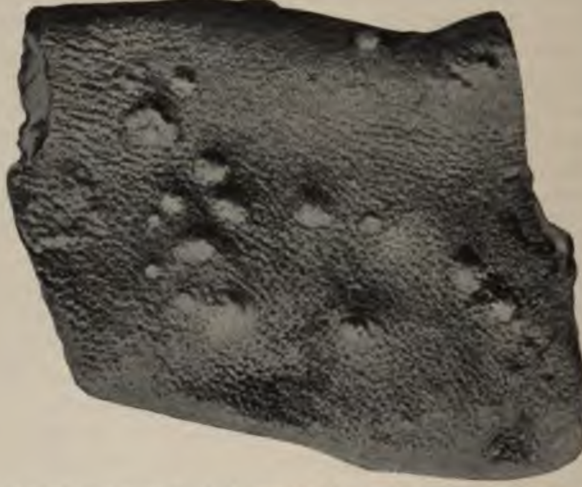
Under the name of " Kopf-tetanus," or head tetanus, a modified form of the disease sometimes occurs. It is said to be particularly apt to take place after injuries to the face. In these instances the spasm is chiefly confined to the muscles of the neck and face, but often extends to the abdominal muscles, and there is frequently spasm or paralysis of the glottis, which not rarely becomes a most serious symptom. In some instances the disease gradually spreads until it becomes like an ordinary case of tetanus. It is to be treated by the employment of antitoxin and other antitetanic measures.

GLANDERS.

Definition.—Glanders is a disease which is usually met with in the horse, but it may also affect man. It is due to the presence of the *Bacillus* *mallei.* When it appears as nodular masses in the nostrils of the horse it is called "glanders," but when these nodules are in the skin it is called "farcy." Analogous types occur in man.

Etiology.—The *Bacillus mallei* is usually conveyed to man while caring for a horse suffering from glanders, and enters his body through some break in the skin. It may also find its way into the system by way of the nasal mucous membrane. Rarely one person is infected by another by contact and through a wound. The bacillus is a slender organism, somewhat thicker in proportion to its length than the bacillus tuberculosis, with rounded ends. It is easily stained with aniline dyes, but is equally readily decolorized by feeble acids or alcohol. It can be readily cultivated outside the body.

FIG. 43



Character of the cutaneous eruption in human glanders. The variation in size and general lack of umbilication are noteworthy points in differentiating it from that of smallpox. On account of shrinkage the skin and pustules appear more wrinkled than they did before removal from the body. (Photograph, natural size, by Roman Mercado, assistant photographer of the Bureau of Animal Industry, U. S.)

Pathology and Morbid Anatomy.—The *Bacillus mallei* produces a circumscribed infiltration of the tissues with accumulations of leukocytes and connective-tissue cells, which resemble macroscopically small miliary tubercles, but, as Baumgarten has shown, these nodules histologically occupy a position midway between tubercles and miliary abscesses. The surrounding tissues are infiltrated with blood or show many, or few, petechial extravasations. After a short time they undergo necrotic changes, and as they break down abscesses are formed, which by necrosis of the overlying tissues are changed into ulcers, which may be superficial or deep. Like tuberculosis, the infection tends to spread along the lymphatics and eventually the bacilli may reach the blood and be distributed in the viscera, causing nodules in various organs. Such nodules occur in the testicles, lungs, spleen, liver, and kidneys, and sometimes the bones are affected, causing an osteomyelitis.

When the nodules break down secondary infections perpetuate the suppuration, the specific bacilli become much diminished, and it may be impossible to discover them by staining, because at this period they lose their property of being readily stained.

Symptoms.—Acute glanders develops, in about four days after inoculation, with *general wretchedness*, some *fever*, and the appearance at the site of infection of a *circumscribed red swelling*. This is followed in a few days by breaking down of the granulomatous mass, in *ulceration of the nasal mucous membrane*, and the discharge of muco-pus from the anterior nares. Secondary infection of the lymph glands in the neck may occur, and if the process is severe the nose may become necrotic. Cough and dysphagia may be present. Upon the face and about the joints there develops an array of *papules* which as they become pustules may very closely resemble the eruption of smallpox. A septic pneumonia often comes on.

Death comes to such cases almost invariably by the end of a week or ten days.

In rare instances the process becomes subacute or chronic, and the nasal discharge, unless accompanied by the severe symptoms described, may make a diagnosis difficult, if not impossible, by the ordinary methods of observation.

When the inoculation is by the skin, producing *farcy*, the same acute localized swelling takes place and the neighboring lymphatics become inflamed and swollen. Not only does this occur as it does in most acute local infections which are severe, but small nodules are found scattered along the neighboring lymphatics forming the so-called "farcy buds." These undergo necrosis, and sloughs form. A septic arthritis may develop. The nasal passages escape, as a rule, in farcy. Death takes place in the majority of these cases in from ten to twelve days.

Chronic farcy lasts, like chronic glanders, for a longer period of time than the acute disease, sometimes for years. It presents the picture of multiple abscesses and sloughs, associated with more or less general septicæmia, death taking place from this cause. Very rarely recovery occurs.

Diagnosis.—Glanders—that is, infection of the nasal mucous membrane by the *Bacillus mallei*—can scarcely be mistaken for any other disease. Farcy must be separated from multiple abscesses and carbuncles. This is done by the history of exposure, the distribution of the "farcy buds," and, finally, by the injection of mallein, which produces a reaction as does tuberculin in the tuberculous.

Treatment.—The swellings should be promptly opened and free drainage provided. If possible the local focus should be well removed by excision or by the cautery. A nutritious diet should be given and stimulants used if needed. Mallein has been used as a curative agent, but nearly all acute cases die, do what we will.

ACTINOMYCOSIS.

Definition.—This is a chronic infectious disorder produced by the *Streptothrix actinomyces*, sometimes called the "ray fungus." It is far more common in cattle than in man, and in cattle it usually affects the lower jaw, producing a tumor or growth which gives the disease the popular name "lumpy jaw." In other cases the tongue is involved, producing the so-called "wooden tongue."

Etiology.—The actinomycotic infection may be conveyed from cattle to man by the hands of the individual, or by straws used for picking the teeth, whereby infection of the jaw occurs. Direct transmission from man to man, or beast to beast, or beast to man does not appear to be of very frequent occurrence; apparently both are infected independently by some common route or source, the exact character of which often cannot be determined. Grain which has been soiled by the slobber of an infected animal may infect other cattle. Cereals are thought by some to be the most frequent carriers of the disease to man and beast.

The organism appears, in the discharges from the areas of infection, as a minute, rounded mass so tiny as to be microscopic in some instances, but in others as aggregated masses, called granules, which are as large as a pin's head. These masses are yellowish-white, resembling particles of sulphur or iodoform, grayish or drab in hue, and even with slight magnification often appear in groups or clumps of radiating filaments, which have caused the organism to be called the "ray fungus." Each terminal filament in some stage of its evolution develops a bulbous end.

Pathology and Morbid Anatomy.—In the lower animals the disease produces a slow, suppurative, and proliferative process, which results in the development of large fungous growths, which may in part become calcareous. From these growths, which are usually situated primarily in the jaw, secondary extensions occur, so that the fungus is found in the tissues of the tongue and pharynx, and even in the lungs, the intestines, and in the nearly related glands and skin. When the disease affects man it does not so commonly involve the jaw, but results in the development of abscesses which often change into ulcers or form fistulæ. These may lead to the deeper tissues, although the disease is usually superficial.

Histologically the new tissue may closely resemble sarcoma, for which it is often mistaken, but its richness in pus cells and resemblance to granulation tissue, combined with the presence of the fungus, should prevent this error.

The lower jaw is more frequently attacked than the upper; cutaneous or subcutaneous forms occur and invasion of the alimentary and respiratory organs, both primarily and as a secondary process, is not uncommon. A chronic *bronchitis actinomycotica* and cerebral actinomycosis are among the rarer manifestations of the disease.

Symptoms.—The symptoms of actinomycosis in man depend to a great extent upon the part of the body which is affected. When the infection takes place through a carious tooth or by ulcer of the gum the jaw is invaded, and the tissues covering it become swollen. To such an extent may this swelling increase that the neck and face may be involved. In these tissues suppuration ensues and pus is discharged from chronic and somewhat puckered sinuses, which heal in one place only to break out elsewhere. Rarely the disease may spread to the fauces and to the tongue.

By the swallowing of the fungus it may infect the intestines and even the liver, and in all these organs it often causes the formation of abscesses. It has been found in the stools in these cases.

Actinomycosis affects the lungs even more frequently than the alimentary tract, and produces symptoms of subacute bronchitis or bronchiectasis or even those of pulmonary abscess. The patient suffers from cough and from fever, and expectorates purulent material in which the micro-organism is often found. The pulmonary lesions are not very acute in their course, but rather chronic, life usually being prolonged in these cases for a year or even longer than this. Rare cases of brain abscess have been recorded as the result of the organism reaching this organ. Howard has been able to find only four primary cases beside his own, and thirteen secondary cases. Such cases must not be confused with those equally rare instances of streptothrix infection which Musser has recently reported.

Diagnosis.—The disease, when the jaw is affected, must be separated from ordinary necrosis and from sarcoma. In the first the swelling is not so widespread and the sinuses not so numerous. In the second condition there is no suppuration, the growth is usually more rapid, and the surface is not so fluctuating. An examination of the pus in those cases in which it escapes will decide the diagnosis by revealing the ray fungus. It is to be remembered that secondary pyogenic infection and extensive necrosis may render the detection of the specific fungus difficult if not impossible, and undoubted cases are on record in which for relatively long periods the characteristic organism was absent. Search for the germ is most likely to be rewarded during recrudescence in old lesions and in newly formed nodules or extensions, when they are freshly opened.

Treatment.—This is largely surgical when the growth is so placed as to permit of its being attacked by this means. The mass and the surrounding tissues should be excised and all dead bone and infected tissue removed, after which drainage should be maintained and the sinuses irrigated with weak solutions of odine or of iodoform in oil. When the pleura is involved iodoform injections are particularly useful. Iodide of potassium is also an effective drug when given internally in doses of from 20 to 60 grains a day, it being thought that in its liberation of iodine it acts as a specific against the ray fungus.

Periods of marked improvement and even apparent cure should not cause relaxation in treatment, nor do they justify a too hopeful prognosis, as a recrudescence of lesions long obsolescent is of frequent occurrence.

MYCETOMA (MADURA FOOT, FUNGUS FOOT OF INDIA).

This is a mycotic disease, usually invading one or both feet and rarely appearing in other parts of the body. It is most commonly observed in India. Sporadic cases occur in other parts of Asia, in Europe, and in South America. A number of cases have been reported in the United States. Two varieties of the disease are recognized: the melanoid, or mycetoma with black granules, and the ochroid, with white or yellow granules. They are due to distinct varieties of streptothrix (*Streptothrix maduræ* and *Streptothrix mycetomæ*). The disease is closely related to actinomycosis; indeed, some of the cases reported in the United States as mycetoma are undoubted cases of actinomycotic feet.

The disease commonly attacks one foot; beginning as a firm, hard nodule on the sole, which gradually softens and discharges an oily, fetid pus containing the black or yellow granules. The sinus thus formed persists. Other nodules appear and go through the same course. The foot gradually enlarges and the sole is greatly thickened. The disease attacks the deeper tissues until eventually all structures, including the bones, are converted into a greasy, yellowish mass. The appearance of the fungus foot, with the thickened sole, the toes strongly extended upward, and the plantar and dorsal surfaces covered with the button-like orifices of the sinuses, is characteristic. Occasionally, the disease shows some tendency to the formation of secondary deposits, spreading along the lymphatic vessels. The diagnosis between mycetoma and actinomycosis rests on the microscopic character of the organisms.

Treatment.—The treatment consists of conservative resection in early cases and amputation in older cases.

SYPHILIS.

Definition.—Syphilis is a contagious disease, the cause of which is as yet undiscovered. It is sometimes called "*Lues*," "*Pox*," or "*Lues Venerea.*" It occurs in two forms, the *acquired* and the *hereditary*, and is characterized in the different stages of its progress by a greater number of pathological changes in the tissues of the body than any other known malady. It has been said that he who knows the whole pathology of syphilis and tuberculosis knows all pathology. This is, of course, an exaggerated statement, but it emphasizes the fact that the disease presents lesions in many different tissues.

The acquired form is usually divided into three stages, called the primary, secondary, and tertiary, and to these Fournier has added a fourth group of affections, apparently quite separate from any of the foregoing, which he calls the parasyphilitic or metasyphilitic affections.

The primary stage is characterized by the development of a chancre or hard sore, also called the "initial lesion"; the secondary stage by the appearance of eruptions and lymphatic swellings, and by ulceration of the mucous membranes. The third stage consists in the growth of tumor-like masses, called gummata, and pathological changes in the bones and in the nervous and vascular systems. In the parasyphilitic forms there is atrophy of the cells of the part involved and overgrowth of connective tissue.

History.—The history of syphilis is not definite. Certain investigators believe that it is one of the most ancient maladies, but it was not clearly recognized as a separate affection in Europe until 1494. Those who wish to look into this question should consult *Syphilis in Ancient and Prehistoric Times*, by Buret, translated by Ohmann-Dumesnil.

Distribution.—Syphilis is found all over the world, and in its frequency and virulence is not modified materially by climate or geographical conditions, but it is worthy of note that the disease is unknown among savage peoples who have not come in contact with civilized communities.

Etiology.—No one seems to doubt the micro-organismal nature of syphilis, although a demonstrated etiological agent is wanting. A great number of organisms have at various times been regarded as the cause of syphilis, among them being the bacteria described by Lustgarten, Van Niessen, Lisle and Jullien, and the parasites studied by Döhle and by Schüller. None has been proved an etiologic factor. Siegel describes a small body, the *cytoryctes luis*, which he finds in the blood of syphilitics and regards as the cause of the disease. His findings lack confirmation. A distinct advance in the investigation of the disease was made in 1903, when Metschnikoff and Roux succeeded in producing characteristic lesions in the higher apes by inoculating syphilitic virus from human beings; this has since been many times repeated. Early in 1905 Schaudinn and Hoffmann described a small spiral organism which they found constantly in primary and secondary syphilitic lesions and which, because of its indifferent staining qualities, they termed the *Spirochæta pallida*. Recently they have given it the name *Treponema pallidum*. Their findings have since been confirmed by hundreds of observers in all parts of the world. No authentic instance of the organism in tertiary lesions is on record. It is found in the artificially produced lesions in apes and highly important is its presence in the blood and tissues of infants dead from hereditary syphilis. Levaditi has made extensive studies of these cases and finds the organism in greatest numbers in the liver, lung, and suprarenal glands respectively. It exhibits a preference for the perivascular tissues rather than for the blood stream and is found in and without the vessel walls in enormous numbers. Many are intracellular, especially in epithelial cells. The organism is 4 to 14μ long does not exceed 0.5μ in thickness and possesses numerous pronounced spirals. It is actively motile, but flagella have not been positively demonstrated. The specificity of this parasite is regarded by Metschnikoff and many others as being abundantly proven and they accept it as the undoubted cause of syphilis. Some investigators as yet hold more conservative views. It may at least be said that evidence in favor of the specificity of the organism is constantly accumulating.

In the vast majority of cases syphilis is acquired by sexual intercourse, although a large number of cases of acquired syphilis, due to non-sexual contact with syphilitic persons or their garments when infected by discharges, have been recorded. (See Bulkley on *Syphilis Insontium.*) Obstetricians, midwives, and nurses have often contracted the disease through a break in the skin of the finger. Wet-nurses have been infected through the nipples by syphilitic infants, and drinking utensils, knives, forks, spoons, pipes, and dental instruments have conveyed the poison to the mouths of innocent persons. The disease can be transmitted by kissing and by the drinking cup. Primary lesions of syphilis have also been produced in the mouth by perverted sexual practices.

The virus of the disease is active in the transmission of the malady throughout the primary and secondary stages, and during this time all secretions from the lesions of these stages are capable of producing the disease in another person, provided that they be brought in contact with a solution of continuity in the skin or mucous membrane. Infection does not take place through healthy skin or mucous membrane, but the break in the surface may be so slight as to be overlooked. The blood of the patient during the secondary stage is capable of spreading the disease by inoculation, but notwithstanding this fact it is noteworthy that the secretions of the various glands do not contain the poison unless they are contaminated by discharges from local syphilitic lesions.

The acquired disease is not conveyed by the discharges from syphilitic sores, or by the blood of a syphilitic, if five years have elapsed since the date of primary infection; indeed, in most cases the virus ceases to be capable of inoculating another person at the end of two years after infection. This rule holds true, even although the patient may be suffering from syphilitic sores or other active lesions at the time of contact. On the other hand, the spermatozoids may indirectly transfer the poison from the man to woman by the fetus.

A person who is suffering, or has suffered, from acquired syphilis is protected against a second infection in the vast majority of instances, although a few cases have been recorded which seem to throw doubt upon the statement of some syphilographers that the protection is absolute. This immunity is developed at once after primary infection, as early as the development of the primary lesion or chancre, and in some cases even earlier than this. In the case of a person who has inherited syphilis from one or both parents the protection against acquired infection is absolute, even if no signs of the hereditary disease be present.

Hereditary syphilis may come to a child through one or both parents. When the father only is syphilitic, the term "sperm inheritance" is employed, and when the mother only is syphilitic it is called "germ inheritance." A syphilitic male may transmit syphilis to his offspring without manifesting at the time of intercourse any symptoms of syphilis and without producing in the mother any signs of the disease. It is also possible for him to have a healthy child; that is, he may fail to transmit the infection. This depends largely upon the stage of the malady, its virulence and activity, and the value of any antisyphilitic treatment that may have been instituted for the father's benefit before conception, and for the benefit of the mother and child after conception.

A woman suffering from syphilis may or may not bear a syphilitic child, and if active antisyphilitic treatment during pregnancy is maintained, the child is likely to escape. It is also possible for a mother who contracts syphilis during her pregnancy to give birth to a non-syphilitic child, but it is also possible for the child to contract primary syphilis from a mucous patch as it passes through the birth canal. It is interesting to note, however, that while the syphilitic mother is not always able to confer immunity to primary infection upon her child, so that it cannot be infected by the disease before or after birth, it is possible for the syphilitic fœtus *in utero* to confer immunity upon its mother, or, to express it differently, given a child *in utero* by a syphilitic father, that child may be syphilitic at birth, but its mother may not have been infected during pregnancy, and is protected against syphilitic infection subsequently. This is known as Colles' law.

That immunity to syphilis can be so acquired is proved by the fact that if a syphilitic baby nurses at its mother's breast she will not contract syphilis, even if its mouth be filled by mucous patches, but if that infant is nursed by an innocent wet-nurse it can produce syphilis in that nurse.

From what has been said so far it is evident that a syphilitic father or syphilitic mother may be the parent of a syphilitic or non-syphilitic child. If both parents are syphilitic, the probability of the child being infected is twice as great as if one parent is affected.

Prevention.—The prevention of syphilis is one of the great social questions of the age that has not been solved. In many cities prostitution has been licensed in order that, by governmental and medical control, prostitutes suffering from syphilis might be treated and prevented from plying their vocation while capable of transmitting the disease. This plan when instituted has not checked the dissemination of syphilis, since it continues to spread through illicit intercourse carried out with unlicensed women who will not be classed as registered prostitutes.

Syphilis may also be prevented by forbidding intercourse on the part of persons suffering from the disease, and by instructing the non-syphilitic to avoid intercourse while any break exists in the mucous membrane or skin of the external genitals. Careful regard to cleanliness after intercourse is of some protective value.

Frequency.—It is almost impossible to determine the prevalence of syphilis, since the living keep its presence secret and the physician rarely returns a death as due to it, but to some indirect result of it.

In 1874 Dr. F. R. Sturgis estimated that, out of a population of 942,292 in New York City, 50,450 were suffering from syphilis.

In an appendix to Sanger's *History of Prostitution*, 1892, it was estimated that 100,000 persons out of a population of 1,800,000 had syphilis.

At the present time the population of Greater New York is about 3,560,000, and assuming that the rate of increase of the disease has kept pace with the increase in population there would be nearly 200,000 syphilitics in that city. These estimates are not made, however, on a statistical basis.

A committee appointed by the Medical Society of the County of New York for the study of measures for preventing venereal diseases, addressed a circular letter to all the physicians in Greater New York asking them to report the number of cases of gonorrhœa and syphilis which they had treated from May 1, 1900, to May 1, 1901. Of the 4750 physicians to whom the letter was sent, 678 forwarded statistics of their cases. The total number of cases of syphilis reported was 7200. Assuming that as many cases occurred in the practice of the physicians who sent no reports as in the practice of those who forwarded statistics, calculations would show that 50,400 cases of syphilis were under treatment in private practice during the period of time which the investigation covered. As many patients go from one physician to another, it is not improbable that some of the reported cases may have figured twice in the statistics; but the committee believed that the number which did so was more than offset by the large class of patients who take treatment from advertising quacks.

Of forty-five dispensaries and charitable institutions visited by the committee nine refused to give any information. An inspection of the records of the remaining thirty-six showed that 7607 cases of syphilis had been treated during the year.

Burre shows by statistics that the morbidity of syphilis among the inmates of the licensed houses of prostitution in Paris has fallen from 30 per cent. in 1873 to 0.25 in 1902. On the 1st of January, 1873, 1126 public women were registered, and during the year 338 cases of syphilis were recorded from among the number. On the 1st of January, 1902, 429 public women were registered, and only 1 case of syphilis was observed among them during the year. Burre attributes this decrease in syphilis to the more general dissemination of knowledge concerning the infectious nature of the disease and to the adoption of hygienic measures for its prevention, which at present are largely practised by all the licensed prostitutes. He also lays some stress upon the matter of obligatory elementary education, believing that it may have served to make the prostitutes more intelligent as a class than they were thirty years ago. His statistics illustrate very well the fallacy of collecting cases and drawing conclusions from them without due care. Surely no one supposes that the number of prostitutes in Paris has diminished in the proportion of nearly 75 per cent.

R. W. Taylor states that most of the cases of syphilis which he has seen in hospitals are from tenement houses and have not contracted the disease from regular prostitutes, which illustrates the difficulty of preventing its spread by licensing women of the town.

Pathology and Morbid Anatomy.—As already stated, syphilis may in its various stages of development affect almost every tissue of the body. Some of these manifestations are not distinguishable from lesions resulting from other causes, and hence their syphilitic character can only be established, if at all, by the exclusion of other factors and the associated presence of recognizable luetic phenomena.

The *primary* lesion of syphilis, called the *chancre*, develops at the point of infection, and is usually characterized by an obliterative endarteritis with spheroidal cell infiltration of the surrounding connective tissue, and by the formation of connective-tissue cells which are particularly numerous about the bloodvessels. As a result of the vascular changes and associated lessened nutrition and possibly the action of the syphilitic poison, with or without added infection, superficial and usually central necrosis occurs and an ulcer results.

Soon after the formation of the chancre, just described, the *secondary* stage develops. The lymph nodes all over the body, but notably those adjacent to the initial lesion, become enlarged and inflamed, and inflammatory and degenerative or necrotic processes develop in the skin, in the mucous membranes, and in the bones and viscera.

Following this so-called secondary period of the disease there develops the *tertiary* stage, in which the periosteum and the internal viscera suffer from peculiar growths of newly formed tissue. A most constant lesion is characterized by the formation of a new tissue consisting of spheroidal and polyhedral cells and scattered giant cells, which are commonly abnormal, poorly supplied with bloodvessels, and having a marked tendency to necrosis, especially coagulation necrosis, and hyaline degeneration in their earlier stages, and later caseation closely resembling that seen in tuberculosis. The growth of this new tissue is usually in circumscribed nodes, and it is in these masses that the necrotic and degenerative processes just named occur most markedly or are most evident. These "*gummata*" may grow to considerable size. They appear as dirty-white, firm masses which, on section, often are found to be caseous at the centre, where the new tissue has undergone necrotic change.

Syphilis produces grave changes in the *bloodvessels*, and no other pathological process impairs the general vascular system so markedly, except it be renal disease. A syphilitic arteritis develops with diffuse overgrowth of fibrous tissue in the adventitia, and even gummata may form along the vessels. The arteritis also involves the middle coat and even the endothelial lining of the vessels, and so narrows or occludes them. This of course diminishes the blood supply to the various organs and increases the labor of the heart. The heart muscle also suffers from a myocarditis characterized by overgrowth of its connective tissues, and the pericardium and endocardium may be thickened for a like reason, but gumma of the heart is very rarely produced. The changes in the heart are, therefore, almost entirely due to the effects of the disease on the vessels which supply it, and upon the changes which occur in the aorta and the peripheral vessels. In other words, while arteritis may result in *myomalacia cordis* the conspicuous change is a fibrosis of the heart muscle. The aortitis and general arteritis result in increased cardiac and vascular stress.

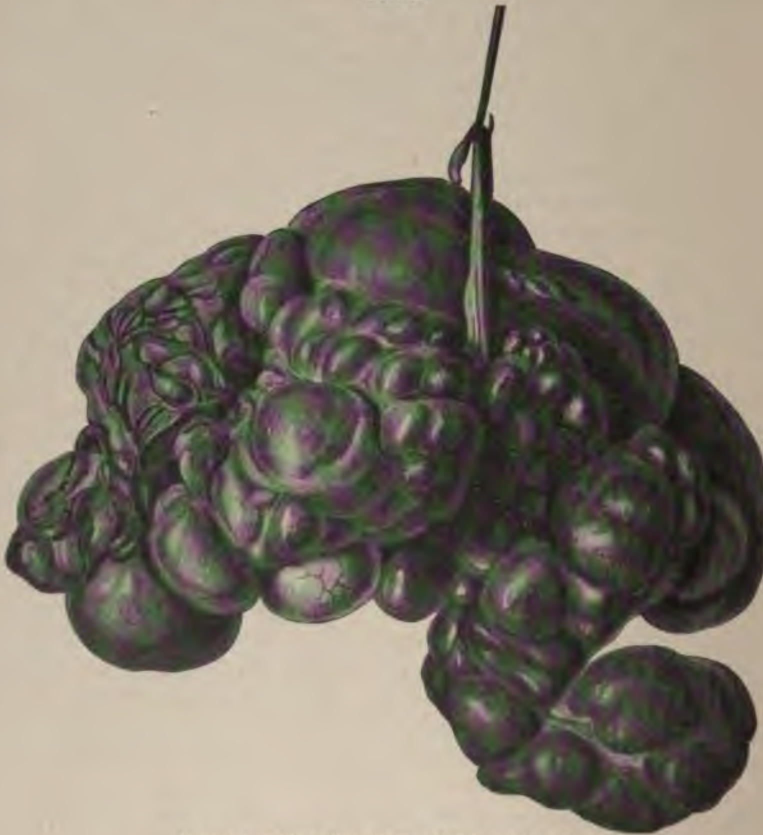
In the secondary stage of syphilis an acute *syphilitic nephritis* has been described. Later on a destructive overgrowth of connective tissue develops in association with the vascular changes just described, and gummatous growths occur in the kidneys.

The *liver* is very commonly affected by the formation of gummata or by connective-tissue proliferation, which produce grave interference with its function. These changes take place in both the acquired and in the hereditary form of the disease. This overgrowth of connective tissue occurs in two types. It is developed between the lobules constituting an interlobular or perilobular cirrhosis, and between the cellular columns forming an intralobular cirrhosis. In some instances these connective-tissue formations consist in large, firm bands which run in various directions through the liver and, in contracting, draw in the capsule of Glisson and so cause great distortion of its surface. (See Fig. 44.) Not rarely gummata are enclosed by these bands. In the earlier stages and milder forms of syphilitic hepatic cirrhosis the changes cannot be considered pathognomonic, but in the exaggerated form, just described, typical syphilitic changes occur. As secondary lesions of the liver amyloid disease and atrophy of its parenchyma are occasionally observed. (See Cirrhosis of the Liver.)

The lesions in the *lungs* consist in gummata which are often surrounded by exudative material, as in pneumonia. These gummata may contain a cheesy area as in tuberculosis and, by pressure, may cause secondary alterations. A second change is overgrowth of fibrous tissue around the bronchi which, associated with catarrhal processes involving their mucosa, distorts these tubes, causing narrowing at some points and at others bronchiectases. Infarctions may occur because of the obliterative changes in the bloodvessels.

A true syphilitic phthisis presenting symptoms resembling tuberculous pulmonary phthisis, but in which tubercle bacilli are not present, may occur, but it is exceedingly rare. It is true that cases have been reported in which gummata in the lungs have, like tubercles, undergone softening of a caseous type, and Wilks has recorded an instance in which this process had gone on to the development of a cavity. Virchow and Fowler have recorded similar cases. These cases, however, although they may produce physical signs of cavity do not present the characteristics of pulmonary phthisis in the sense of pulmonary tuberculosis, nor do those instances in which bronchiectatic cavities develop as the result of syphilitic fibroid changes in the lungs do so, even though the physical signs may be similar. The main pathological difference in the two states is this—viz., that in tuberculosis there is not only a destruction of the tubercle by softening, but the intervening tissue is infiltrated with exudate which soon becomes tuberculous

FIG. 44



Nodular syphilis of the liver. (Kast and Rumpler.)

and proceeds to necrosis. This does not occur in syphilis. In rare cases syphilis of the lung and tuberculosis may be coincident.

In the hereditary syphilis of infancy a lobar or bronchopneumonia in which the pulmonary tissues show red, gray, and white exudates, according to the stage of the local disease, is sometimes met.

The *lymph nodes* in cases of syphilis are always affected by an overgrowth of connective tissue after the primary infection. In the third stage gummatous masses may develop in them.

Next to changes produced in the organs of circulation syphilis manifests its gravest changes in the *central nervous system*. The meninges may be the seat of gummata with or without the presence of chronic, indurative overgrowth of connective tissue.

In the brain it causes gummata manifesting the symptoms of brain tumor; it also produces a syphilitic inflammation which is associated with the formation of a gelatinous tissue, and finally and most frequently it gives rise to serious degenerative changes in the arteries which interfere with the nutrition, and later by rupturing bring about cerebral hemorrhage. It also causes gumma of the cord and its membranes, which usually have their origin in the tissues of a bloodvessel or in the pia arachnoid. Rarely it affects the peripheral nerves, through pressure, as they emerge from the cerebrospinal sheaths. In the spinal cord it causes degenerative changes of cells and fibres and overgrowth of the sustentacular tissue.

The *parasyphilitic affections*, paresis, meningoencephalitis, locomotor ataxia, etc., will be discussed with diseases of the nervous system.

Symptoms.—The symptoms of acquired syphilis are best described as they appear in the three stages of the disease.

FIRST STAGE.—In from twelve to twenty-one days after exposure and infection the patient develops at the site of original contact with the virus a *small papule* or pimple which has an area of indurated tissue about its base, the so-called primary lesion or *hard chancre*. Further examination of the patient will reveal the fact that the inguinal glands are slightly enlarged.

This period of primary syphilis lasts from three to ten days or two weeks, and is followed by the development of the *secondary stage*.

SECOND STAGE.—In the secondary stage we find *fever* as an early symptom, which varies in its degree very greatly in different patients. In some instances it is so mild as to be overlooked; in others it may rise to a point as high as 104° or even 105°. The more common febrile movement is one in which the temperature for some days stays in the neighborhood of 101°. When the fever intermits, being fairly high at one period and then breaking sharply, it may mislead the physician into a diagnosis of malarial infection or acute sepsis. I have seen several cases in which a diagnosis of typhoid fever, malarial fever, or tuberculosis was made when in reality the disease was early secondary syphilis.

The *skin eruptions* of the secondary stage consist chiefly of the *roseola*, the development of which often marks the onset of the secondary stage. This *roseolous rash* may occur in limited areas or be widely distributed over the body and even involve the face. On one occasion a woman with a well-developed syphilitic roseola presented so scarlet a visage that, although she was veiled, she caused the other patients to leave my waiting-room in alarm, they thinking that she had scarlet fever. As a rule, however, the rash is not so marked on the face.

In other cases, in place of roseola there develops a *macular syphilide*, characterized by the appearance of reddish-brown or copper-like macules scattered over the trunk.

As the secondary stage advances the eruption may be *papular* and *finally pustular*, and at this time it may closely resemble that of true variola. In still other cases a *squamous* or *scaly eruption* appears which differs from psoriasis in that it is not chiefly on the extensor surfaces as is ordinary psoriasis, and in addition it is frequently copper-colored.

At the point of junction between the mucous membrane and the skin, as at the anus or at the angles of the mouth, "*mucous patches*," or ulcers, develop, and upon the skin in the neighborhood of these lesions warty growths of a flat character, the so-called *syphilitic condylomata*, appear. Mucous patches on the buccal mucous membrane and tongue also appear.

There is nearly always some *falling of the hair* in secondary syphilis. Sometimes this falling is well distributed; in other cases it is in patches—syphilitic alopecia.

A serious, oftentimes painful complication at this stage is *syphilitic iritis*. If the treatment is not active sight may be lost.

A rapid development of *anæmia*, which often becomes quite marked, not as to hæmoglobin, but as to the number of the red cells present, is often observed.

The secondary stage lasts from twelve to eighteen months, and is usually followed by a period during which the symptoms are modified or entirely disappear, the virulence of the disease seeming to have spent itself, but even if no syphilitic symptoms are present a child begotten at this time will usually suffer from hereditary syphilis.

THIRD STAGE.—In the great majority of untreated cases the malady proceeds to the so-called *tertiary stage*. This is characterized by the presence of *skin lesions*, which are more severe than those of the secondary period, such as *tuberculous* and *ulcerous* formations of a subacute or chronic character. There is an overgrowth of connective tissue in different parts of the body, as in the secondary period, and multiple *gummata* are often present in numbers, developing in the skin, in the subcutaneous tissues, in the muscles, and in the internal viscera, particularly in the liver. When in the skin they often slough and produce ulcers, and in the internal organs they become filled with fibrous tissue and undergo contraction in the manner already described.

Last, but by no means least, of the changes due to syphilis in its tertiary stage, we meet with lesions of the nervous system. These changes, as a rule, are late manifestations of the disease, occurring some years after the infection. Rarely they may appear as early as within the first six months, usually within the first ten years, seldom as late as twenty years.

Syphilis in certain cases may seem to possess great virulence and become destructive in its course, almost from the onset. The chancre may rapidly ulcerate and spread, the fever may be marked, and the anæmia severe. The skin lesions become pustular, even in the secondary stage, and form deep ulcers, which, in turn, cause scars as they heal. Destructive changes rapidly develop in the bones and viscera. The patient may die within a few months of the infection. In these cases there is usually a lowered vital resistance which permits the disease to progress unopposed.

In other cases the symptoms are remarkably mild. The chancre is so small that it is overlooked, the patient truthfully stating years afterward that he has never had a primary lesion. The rose rash may not occur, or be so faint and fleeting as not to attract notice, and the primary anæmia may be entirely absent. Syphilis may end in complete recovery at the close of the secondary stage, but such a fortunate result is rare, unless active treatment has been instituted.

It is a fact worthy of note that *nervous lesions* seem to occur more frequently in cases which have presented mild secondary symptoms than in those who have had severe secondary and tertiary lesions of internal viscera.

Three intracranial conditions due to syphilis may produce *violent headache*, namely, arteritis, meningitis, and gumma. When arteritis is the cause, giddiness, weakness of groups of muscles, difficulty of speech, and, it may be, signs of general paresis develop. On the other hand, when the ocular muscles are affected and an optic neuritis is present, meningitis is the more likely condition, particularly if there is spasmodic contraction of certain cranial muscles and fever. Neuroretinitis is present in meningitis and in gumma, but is not commonly present in arteritis. (See Meningitis.)

The symptoms of *syphilis of the brain* depend very largely upon the site of the lesion, for, as already stated in the section on the Morbid Anatomy of Syphilis, these lesions may be at the base, on the convexity, or in the membranes. By far the most common symptom of cerebral syphilis is *headache*, which is usually diffuse and constant, but if the meninges are involved, or a gumma is causing pressure, it may be exceedingly severe and characterized by what are known as "crashing pains." Patients with cerebral syphilis are often *unduly somnolent*. (See Diagnosis.)

When gummatous growths form at the base of the brain the symptoms are those due to interference with the cranial nerves, such as *squint*, *optic atrophy*, and *facial paralysis*. When the convexity of the brain is affected the symptoms are those of localized or *Jacksonian epilepsy*, or of *petit mal*. Sometimes the epileptic seizure is general. Fournier laid down as a law that epilepsy beginning in adult years is, nine times out of ten, syphilitic. A third form of cerebral syphilis is that in which there are *psychical disorders*, such as melancholia or delusions of grandeur. (See Paresis.)

Spinal syphilis manifests itself as the result of the presence of gummata or of connective-tissue changes in the cord. When the lesion is a gumma the symptoms are those of pressure on the cord. When connective-tissue changes occur the signs are those of spastic paralysis of the lower limbs, with markedly exaggerated reflexes, low muscle tension, and vesical disturbances. Often the disorder of the functions of the bladder is the first sign of spinal difficulty. The bladder may lose its expulsive power or incontinence may occur. As a parasyphilitic affection locomotor ataxia is the most common nervous disease. (See Locomotor Ataxia.)

Diagnosis.—The diagnosis of acquired syphilis is readily made if the patient presents the well-developed symptoms. In many cases, however, these are not manifested. The indurated base of the chancre is an invaluable sign if the chancre has not been cauterized, and the presence of enlargement of the lymph nodes in the groin and in the great chain of nodes in the neck is also a useful diagnostic point. The presence of secondary syphilitic roseola, and fever with sore throat, and mucous patches are diagnostic. In some cases, however, the secondary symptoms never develop or are so mild as to be overlooked, yet well-marked tertiary signs develop later. The employment of mercury or iodide of potassium, followed by the disappearance of the symptoms, is a therapeutic test, but such a result is not a pathognomonic sign of syphilis.

Prognosis.—The outlook in acquired syphilis as to severity of attack and ultimate recovery depends largely upon the state of the general health, and the promptness with which specific treatment is instituted. Much depends also upon the faithfulness of the patient in carrying out the treatment for a sufficient length of time. In the great majority of cases active and skilful treatment permits a favorable prognosis as to complete cure, and even as to the safety of future marriage. In the malignant cases, or those in which tertiary lesions have already formed, we can only hope to modify the progress of the malady, or perhaps arrest it without being able to remove all signs of its invasion. Gummatous growths may, however, be removed by treatment, even in the tertiary stage.

Hereditary Syphilis.—The symptoms of *hereditary syphilis* may be present at birth, the skin being already the site of *syphilitic eruptions*, of which *pemphigus neonatorum*—that is, a bleb-like eruption about the wrists and ankles—is typical. The *liver* and *spleen* are usually *enlarged*, and the child may be wasted and poorly nourished. In other instances the child manifests no lesions at birth, but within its first six months of life develops *syphilitic rhinitis*, or, as it is called, "snuffles." This is accompanied or followed by cutaneous lesions, of which the most frequent is *mucous patches* about the anus and in the mouth. It may waste away from so-called *syphilitic marasmus*, developing a syphilitic rosary at its costocartilaginous junctures, as in rickets. The ends of the long bones are the victims of *syphilitic epiphysitis*. If the child lives to reach the period of second dentition its teeth may be notched—the so-called "Hutchinson" or "peg" teeth. This malformation does not appear in the milk teeth. In many cases of hereditary syphilis in infancy the child looks like an old man, whereas in hereditary syphilis of early adult life the patient often looks very immature—"syphilitic infantilism." Children having hereditary syphilis are prone to suffer from *syphilitic keratitis*, from *deafness*, and from *bone lesions* which develop after several years of life. The periosteum is thickened and even nodular in its appearance, particularly on the tibia.

Another lesion is *deformity of the fingers*, in which they become thickened at the base and taper rapidly to the tip, being somewhat pear-shaped or top-shaped—the so-called *syphilitic dactylitis*.

Treatment.—Divergent views have existed in regard to the proper treatment of syphilis in its early stages. A certain number of practitioners of experience have insisted that it is unwise to administer mercury to a patient suffering from a suspicious primary lesion until, by the development of secondary symptoms, the diagnosis of syphilis is absolutely confirmed; since if we do not wait for these symptoms, the possibility exists that a patient who has not really acquired syphilis may be condemned to the belief that he has been inoculated, and this may cause him great mental suffering during the rest of his life.

Another group of practitioners have strongly urged a view directly opposed to this, claiming that we have no right to permit the disease to become thoroughly engrafted upon the patient's system without instituting measures for its relief, or at least for the diminution of the severity of the infection. The leaders in this line of thought have advocated the excision of the chancre in the belief that by so doing the primary focus of infection was removed. It must be remembered, however, that the primary lesion does not develop until two or three weeks after the actual inoculation, and, therefore, although it appears at the site of inoculation, there is good reason to believe that it is not a source from which still further infection takes place, but rather a localized manifestation that inoculation has been accomplished. Nearly always there can be found in the adjacent lymphatics evidence that they are affected as early as the chancre appears. The question as to whether the chancre should be excised must, therefore, be left to the judgment of the individual physician, with the statement that it is possible, but not probable, for the excision to have some influence for good.

It is with those who believe in the immediate administration of antisyphilitic treatment as soon as the chancre is developed that I agree. It does not seem to me rational to permit the disease to run on uncontrolled until he who runs may read that infection has taken place. While it is true that the chancre at times is not sufficiently characteristic to enable us to make a positive diagnosis that it is true syphilis, we are justified in such a case in considering that it is such and proceeding at once to the relief of the patient.

The main treatment during the primary and secondary stage of syphilis must consist in the administration of full doses of the protiodide of mercury, which should be given in the form of uncompressed tablet triturates in the dose of ¼ of a grain three times a day, increased by one or two quarters each day, until the patient manifests distinct evidences of the full systemic effect of the drug, as evidenced by some looseness of the bowels or by the development of tenderness of the teeth and slight salivation. It is important when this drug is given that tablet triturates, and not compressed tablets, are employed, as the compressed tablets are often unabsorbed, because of their hardness, and frequently cause irritation of the stomach, whereas, the properly made tablet triturate rarely does.

As soon as the patient manifests any of the symptoms, mentioned as indicative of the fact that he is using all of this drug which he can well bear, it is proper to diminish the dose one-half, and keep it at this point, provided that this dose seems competent to prevent the development of further syphilitic manifestations. If, however, this half dose is not sufficient for this purpose, the drug must be given in ascending doses the second time, and if the syphilitic manifestations are at all malignant it may be necessary to continue it, even if opium or bismuth have to be given to control diarrhœa.

It is essential in the use of the protiodide of mercury in syphilis, first, that the stomach shall not be disordered, because it is of vital importance that the patient should be able to take full quantities of highly nutritious food, in order that by maintaining his vitality his own vital processes may aid him to combat the infection.

It is also essential that great care be taken against the development of mercurial stomatitis. If this condition once develops, it is often difficult to cure it while the mercury is continued, and it frequently will prevent the patient from taking sufficient doses of the drug to favorably influence his syphilitic infection. If the patient is directed to take the greatest possible care as to cleanliness of his mouth, to use a tooth-brush and some antiseptic dentifrice after each meal, to keep particles of food from between the teeth by the use of floss silk, and, finally, if he also be given a prescription calling for 10 grains of chlorate of potassium and 10 drops of tincture of myrrh in an ounce of elixir of calisaya, which is to be diluted one-half with water, and used as a mouth-wash night and morning, it will be found that he will be able to take much larger doses of mercury than if these measures are delayed until some evidences of mercurial sore mouth present themselves.

Should the manifestations of syphilis be virulent, then it is necessary to give the drug to the patient not only in the form of the protiodide by the mouth, but to use blue ointment rubbed into the skin at least once a day, in the dose of about 1 drachm, choosing a different spot each time for the rubbing, and exercising great care that the rubbing is continued long enough to actually cause the absorption of the mercury. Usually a hot Turkish bath or, if this is impossible, an ordinary hot-water bath should be taken before the mercurial ointment is used, in order that the skin may be rendered pliable and put in such a state that the mercury can be readily taken up by the tissues beneath it. The entrance of mercurial ointment into the body may also be aided by smearing it on a flannel binder and placing this about the patient's waist.

In other instances, in addition to the internal and external use of mercury, or in place of one of them, hypodermic injections of mercury may be employed. For this purpose one of the best preparations is corrosive sublimate dissolved in normal salt solution and given in the dose of $\frac{1}{8}$ of a grain, injected deeply, but gently, into the loose cellular tissues of the buttocks or back, or, better still, into the body of the greater muscles, such as the gluteus. Great care must be exercised that antisepsis is complete, since otherwise the irritant drug, although antiseptic in itself, may cause abscess. This injection should not be given oftener than every two or three days. In other instances gray oil may be used, prepared by rubbing 2 drachms of lanolin with a sufficient quantity of chloroform to form an emulsion, continuing the rubbing until most of the chloroform is evaporated, then adding metallic mercury to the extent of 4 drachms, and rubbing again until the mixture is complete. This strong gray ointment, diluted still further by the addition of equal parts of olive oil, may be injected in the dose of 1 or 2 minims every second or third day in the same manner as corrosive sublimate.

Still another way of getting mercury into the body is by means of the sublimation of calomel. The patient, being stripped of all clothing, is wrapped in a blanket and placed upon a chair with a wooden seat. Under this chair is placed an alcohol lamp and over it a disk of metal upon a small iron stand, on which 20 grains of calomel is laid. Upon this stand is also placed a tincupful of water. The heat of the lamp vaporizes the water and sublimes the calomel, and the mercury, being deposited upon the skin of the patient, is absorbed. This method of treatment is useful for the relief not only of the systemic symptoms, but also for the syphilitic eruption of the skin. A similar plan of sublimation can be carried out with inhalations, the patient holding his face eighteen inches away from the pan and inhaling the fumes. If this is done mucous patches in the mouth are very frequently rapidly healed, but after each employment of sublimation and inhalation, the mouth should be well rinsed with water, in order that an excess of mercury may not remain there and produce stomatitis. In many cases the best results are produced by a plan of treatment in which both the iodide of potassium and protiodide of mercury are given together or alternately.

The *treatment of the tertiary stage* of syphilis consists chiefly in the administration of iodide of potassium or iodide of sodium or iodide of strontium, in as full doses as the patient can well bear, but should evidences of gummatous growth in the brain present themselves the iodide is not sufficiently active in its action, and mercury should be given with it.

The dose of the iodides varies greatly with the susceptibility of different individuals. As a rule, tertiary syphilis is not benefited by giving less than 100 grains a day. I have had a patient under my care who would take 800 grains a day with great benefit, with no other disagreeable symptoms than the development of an intense acne. But we rarely meet with instances where these enormous doses must be taken.

The proper way to administer the drug is to order a saturated solution of iodide of sodium dissolved in the strength of 1 grain to the minim of water, and direct that 10 minims of this be given in a dessertspoonful of the compound syrup of sarsaparilla three times a day an hour after meals, being increased each day from 1 to 5 minims at a dose. If careful attention is paid to the diet and to the condition of the bowels, patients who would not be able to take large doses at first soon become fairly immune so far as untoward effects are concerned, and can take effective quantities within a brief period of time.

Hereditary syphilis is to be treated by the active employment of mercury. In babies suffering from syphilis, gray powder may be given in the dose of 2 grains two or three times a day, and mercurial ointment may be rubbed into the abdomen and on the inside of the thighs and smeared upon the abdominal binder of the child. This will prove a most advantageous plan of treatment. The change in the nutrition and appearance of the infant under these circumstances is little less than marvellous.

The diet should be carefully regulated, and, if the digestion will stand it, cod-liver oil should be given internally. The employment of the mercurial ointment produces an active systemic influence without disordering digestion, and is, therefore, particularly advantageous when hereditary syphilis is being treated in infants.

TUBERCULOSIS.

Definition.—Tuberculosis is an infectious disease caused by the presence in the body of the *Bacillus tuberculosis*. It is characterized by a local inflammatory process followed by the development of areas of necrosis. While the lesions produced by the disease are varied, the typical manifestation is the formation of small nodules which appear as gray, or white, or sometimes yellowish bodies called tubercles. It is because of these tubercles that the name "tuberculosis" is applied to the malady.

Etiology.—The chief etiological factors in this disease are the specific bacillus and the presence of a favorable state in the tissues of the individual for the growth of the germ. As the disease is constantly present all over the world, except in a few scattered areas, the specific germ is always at hand, and as a large number of causes produce a condition of the tissues which is favorable to their development the disease is only too prevalent.

The bacillus of tuberculosis appears as a straight, slightly curved or bent rod with rounded ends, devoid of motility, and reproducing itself by fission; the often expressed belief that it is a spore-bearing organism is not unequivocally established. It stains with the ordinary aniline dyes and by Gram's method. (See Pathology.)

The *Bacillus tuberculosis* enters the body by several pathways, of which the most common is undoubtedly the respiratory passages. It also gains access by way of the alimentary canal with the food, and occasionally by accidental inoculation. Recent studies have shown that the tonsils and lymphoid tissues of the pharynx are portals through which the tubercle bacillus frequently enters. The position of the tonsils exposes them to both air-borne and food-borne infection, and their crypts and lymphatic communications afford favorable opportunities for the entrance and dissemination of the micro-organism. Very rarely true hereditary transmission takes place by the passage of the bacillus through the placenta or possibly by the infection of the ovum by this organism. Such instances are, however, so rare that they are curiosities in medicine.

When the infection takes place by inhalation it usually occurs by the bacilli being dissipated through the air in the form of dust, or by their expulsion in small masses of sputum which, falling on pillows, bedding, or clothing, are easily taken into the respiratory passages when the sputum dries. Flügge has shown that when a patient coughs with his mouth open the ejected air may contain droplets holding the bacillus, thereby rendering the immediate neighborhood of the sufferer especially dangerous. There can be no doubt of these facts, for they are proved by the very great frequency of the disease in the lungs, particularly when opportunity exists for infection by dust, and by the fact that susceptible animals can be infected by this disease if forced to breathe dust which has been contaminated by dried tuberculous sputum.

Kingsford found in analysis of 339 cases that 216, or 63.7 per cent., occurred by inhalation, 65, or 19.1 per cent., by ingestion, and 17 per cent. were of doubtful origin.

That tuberculous infection by way of the alimentary tract occurs very commonly as the result of drinking milk from tuberculous cows, or milk that has been contaminated by the sputum from tuberculous human beings has long been held. The infection of milk by coughing or sneezing by persons suffering from this disease occurs quite frequently. The milk of a tuberculous cow will convey the infection even if local tuberculous lesions are not present in the udder, and the bacillus may be found in butter made from such milk. The fact that tuberculosis is so frequently found in the mesenteric glands of young children is significant in this connection. Infection by the meat of a tuberculous animal can only occur if the meat actually contains the bacilli and is eaten uncooked. This form of infection is probably very rare except when sausages made from what are known as Bologna cows[1] are eaten in a raw or half-cooked state.

Medical publications have teemed during the last few years with rather heated debates as to the communicability of bovine tuberculosis to man. In the minds of some bacteriologists, in Germany in particular, this question is still *sub judice,* but the majority of those best qualified to judge now agree that no doubt can exist of its transference, particularly from the udders of tuberculous cows to the mesenteric glands of children who drink the milk from these animals.

Raw, Theobald Smith, and others have maintained that man is subject to two forms of tuberculosis, one derived from members of his own zoological group and another due to infection by the bovine bacillus. The wide distribution of tuberculosis in the animal kingdom, the morphological, cultural, and pathogenic differences in the bacillus found under different conditions, and the generally admitted possibility of ranging these organisms in a scale, or at least in closely allied groups, explain, at least in part, the different phenomena as seen in man.

The mere presence, however, of the tubercle bacillus is not the only requisite for the development of tuberculosis, for as already stated the tissues must be in a favorable state for its growth. This favorable state is produced by any cause which impairs vital resistance and prevents the body from destroying invading micro-organisms soon after they enter it. Of these causes, aside from diseases which impair the general health, we find the most potent are bad air, particularly that due to poor ventilation when large numbers of persons are crowded together; lack of exercise, so that all parts of the lungs are not thoroughly expanded; and, lastly, those conditions of air and soil which are associated with excessive humidity, particularly if there be much dust in the atmosphere, as in large cities.

In addition to these causes, which increase the susceptibility of all persons, we also find that certain individuals inherit conditions which undoubtedly predispose them to this disease. They belong to two classes: those who by inheritance possess faulty thoracic development, or bad chest configuration, so that the apices of the lungs never expand properly, and those who seem to inherit a condition of the tissues which is unable to cope with the infection when it takes place. Both these causes are often present in one case. Such persons are usually lightly built and have small bones and delicate features, with a thin skin and superficial veins about the temples. It is a mistake, however, to think that this configuration is always present, for another type exists in which the bony structures are large and the muscles powerful, the so-called "lanky" type, in which tuberculosis is very apt to run a rapid course. Every clinician of experience has been astonished to find active tuberculosis of the lungs in heavy and powerfully built men, and has seen more than one generation of the same family, though strongly built, succumb to this malady, although promising in early life to escape all danger from it. In these instances the vital resistance to infection is poor, although the physique may seem excellent.

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[1] A Bologna cow is an animal so feeble and wasted that it cannot be used for milking, breeding, or for the providing of ordinary butchers' meat. It is killed and used to make sausage.

The influence of age upon the development of the disease is distinct, but it is not sufficiently powerful to confer immunity upon any period of life. In the first ten years of life tuberculosis is quite common, affecting the lymphatic system most frequently, the bones being also commonly involved, and more rarely the membranes covering the brain. After puberty the pulmonary tissues are the parts which are affected in the majority of cases, and this predisposition of the lungs to the disease persists throughout the rest of life, although after the thirty-fifth year the frequency of pulmonary tuberculosis rapidly decreases, so that in persons over fifty years of age it is really very uncommon as a new ailment, unless they have been specially exposed by occupation to infection by the malady. The only cases I have seen of primary pulmonary tuberculosis which *began* in persons of over fifty years of age were miners and grinders.

The sexes are about equally affected by tuberculosis.

Of the races, negroes and North American Indians are very susceptible, and half-breed negroes and half-breed Indians are peculiarly prone to the malady. I have had opportunities of studying the frequency of tuberculosis among both of these classes and have been impressed by this well-recognized fact. Perhaps this susceptibility is due to the fact that the white father is usually a degenerate, or one whose vitality is impaired by alcohol and abuse.

Of the occupations which favor the development of tuberculosis may be named knife-grinding, mining, weaving, and other pursuits which cause large quantities of dust to enter the lungs. (See Pneumonoconiosis.)

All the infectious diseases which diminish the vitality of the patient predispose him to infection by this bacillus. Thus, pneumonia, particularly that of the catarrhal type, not rarely causes pulmonary tuberculosis to develop, and influenza renders the patient especially prone to its development. In many cases the catarrhal process provides the centre in which a new, or an old and slumbering, infection can become active. Among the acute infections, measles and whooping-cough are active predisposing factors, causing catarrhal pneumonia or exhaustion and diminished vitality. Diabetes mellitus very commonly ends in a rapid and fatal tuberculosis.

A very important point is the relation of injury to the development of tuberculous lesions. There can be no doubt that trauma to the chest wall may be followed by an outbreak of pleural or pulmonary tuberculosis, that injuries to the joints, even if seemingly trivial, may cause tuberculous arthritis, and blows on the abdomen may incite tuberculous peritonitis or tuberculosis of the retroperitoneal or mesenteric glands. (See also article on Pneumonia for traumatic lesions of the thorax followed by pulmonary disease.)

Prevention.—In the prevention of tuberculosis the most important factor is the destruction of the bacillus as soon as it leaves the body of the patient This is by no means as easy to accomplish as would appear at first sight, since it is often expelled in enormous numbers by sneezing and coughing. The moustache or beard of the consumptive is a veritable nest of infection, and his bed-clothing may be equally virulent unless he holds something in front of his face when he coughs.

All sputum should be received into rags,which should be burned in a hot fire before they become dry, or into a paper spit-cup which can be burned. If a china cup is used, it should always contain bichloride of mercury solution.

Largely through the efforts of Dr. Herman Biggs, of New York, the health department of that city has been active during the last ten years in stamping out tuberculosis, or consumption. Under an ordinance, physicians are required to report every case of this disease that comes under their care. In the poorer districts of the city this is followed by inspection, and, if necessary, disinfection of the quarters occupied by the sick man, and this again has been supplemented by the distribution of circulars in which directions are given whereby the patient can take precautions against the infection of his family, and the family can protect themselves.

The second great preventive of tuberculosis is sunlight, for sunlight destroys the bacillus. If this were not the case our streets would infect more thousands than they do. Sunlight not only destroys the bacillus, but increases the vital resistance of the patient and of the uninfected as well. The absence of sunlight and the presence of bad air are the most potent auxiliaries to the disease. This is shown by the prevalence of the malady in tenement houses, in prisons, and in asylums which are badly arranged or managed. These facts have not only been proved on a gigantic scale by unintentional tests with human beings, but experimentally as well, particularly by Trudeau, who inoculated two sets of rabbits with the bacillus tuberculosis. He kept one set in a dark cellar and these animals suffered an unusually high mortality. The other set he turned out-of-doors, and these animals survived or were affected only by a modified form of the disease.

In those who have an hereditary predisposition to the disease or who have a faulty thoracic development, out-door life is in many cases an absolute necessity to prevent the disease.

It is proven that tuberculosis can be conveyed from animals to man, and it would seem unwise to give up such precautionary measures as careful inspection of cattle in slaughter-houses and the proper control of cows supplying milk, and especially when the latter is to be used as an infant food.

Frequency.—The prevalence of tuberculosis in its various forms is very great. About one death in every seven is due to this cause, and when we add to this fact the additional statement that a very large proportion of those who die of other diseases show more or less well-developed tuberculous lesions, it becomes evident that tuberculosis contributes to the death of a still larger proportion of persons. Thus Schlenker in 100 autopsies made on adults and children dying of various diseases found that 65 per cent. had tuberculosis. Biggs found it in 60 per cent. of his postmortems, and out of 4000 consecutive autopsies in Breslau about 1300 showed tuberculosis. These statistics, which give some conception of the ordinary prevalence of the disease, are outclassed in an extreme degree by the reports of Naegeli in Zurich, who found in the Pathological Institute of that Canton that 500 consecutive autopsies revealed tuberculosis in some form in 97 per cent. This percentage held true of adults as well as of children. Naegeli also found that tuberculosis is very rare in the first twelve months of life, uncommon up to the age of five years, but so frequent from five to fourteen years that it was found in one-third of all bodies examined. In studying these statistics of Naegeli it must be remembered that in many of the autopsies tuberculosis was not the cause of death, and in some cases was present in such a very slight degree that only careful search revealed its presence. Burrell believes that about 70 to 80 per cent. of all persons who reach the age of forty years have or have had some form of the disease.

The far greater frequency of tuberculosis in cities as compared to country districts and villages is shown by the statistics of Paris, in which the number of cases per thousand is 4.9 per cent.; whereas in 662 villages in France it is only 1.81 per cent.

Notwithstanding these facts it is interesting to note that in many parts of the world tuberculosis has undergone a most remarkable decrease in its frequency, although the mortality rate of 1 in 7 still holds true for nearly all cities. In New York the mortality has decreased from 4.6 to 2.6 per thousand in ten years, and a similar fall of about 40 per cent. has occurred in Philadelphia in that time. Abbott has shown that in 1853 the mortality of pulmonary tuberculosis in Massachusetts was 42 per 10,000 inhabitants, whereas in 1895 it was 21.8 per 10,000 inhabitants. The decrease in the entire United States has been from 25 per 10,000 in 1890 to 19 per 10,000 in 1900. Hiller has shown that at the present rate of decrease the disease will be extinct in Prussia in 1927 and in England about 1947. In Prussia the mortality fell from 31 per 10,000 in 1886 to 19 per 10,000 in 1901, and in England it has fallen 50 per cent. in the last forty years.

A very great difference in frequency is found in different races. Thus, in the United States the death rate in those of English descent is 15 per 10,000, whereas for the Irish it is 43, and 59 for the colored race.

Occupation also makes great differences; thus, the death rate among tradespeople is 17 per 10,000, among barbers 33 per 10,000, book-keepers 40, and stone-cutters 54 per 10,000.

The average age at death from pulmonary tuberculosis is thirty-five years, but the actual incidence of the disease is from fifteen to thirty-five years.

The relative frequency of the different forms of tuberculosis is difficult to determine. Statistics of deaths from tuberculosis in Ireland from the years 1891 to 1901 show the following figures as to the relative frequency:

Pulmonary tuberculosis	21.35 per 10,000
Tuberculosis of the mesenteric glands	2.2 " "
Tuberculous meningitis	2.25 " "
Other forms of tuberculosis	2.3 " "

Some difference exists, however, between the frequency of primary and secondary lesions. Thus, Heller, of Kiel, found but 1.45 per cent. of primary intestinal tuberculosis, but 37.8 per cent. in which the principal lesion was abdominal.

Pathology and Morbid Anatomy.—As already stated, when discussing the etiology of tuberculosis, the bacillus enters the body usually through the respiratory mucous membrane, or through that of the alimentary canal. It is possible, however, for infection to take place through the skin, but this is usually followed by a localized lesion and rarely by visceral disease. The results which accrue from the entrance of the bacillus, in the manner indicated, vary greatly with the virulence of the micro-organism, the vital resistance of the individual and the organ or part in which the primary localization of the bacillus takes place. The effect of the bacillus upon the local tissues is to cause an accumulation of cells in the immediate neighborhood, followed in favorable cases by repair or, under less promising conditions, by necrosis. This aggregation of cells, composed of lymphoid, hyaline, endothelial, and, it may be, giant cells, and containing the bacillus, is the histological or rather anatomical characteristic of the disease, and is called a *tubercle.*

In the great majority of instances the pathological process which is induced is inflammatory in type, and, as already stated, the lungs and their adjacent lymph nodes are the parts which are usually affected in adults, whereas in young children the gastrointestinal tract and its adjacent glands are commonly involved either alone or with the structures just mentioned. When the collection of the cells is small the growth is said to be a *miliary tubercle,* because it is thought to be the size of a millet-seed; but when a single tubercle grows to be so large as to be called a nodule its growth to these proportions is accomplished by the addition of a number of miliary tubercles. This growth usually is limited, in the miliary tubercle or nodule, by the fact that an exudate takes place, as part of the inflammatory process which the bacilli produce, which prevents the spread of the bacilli to adjacent areas, and so limits the field occupied by the micro-organisms.

As the disease progresses this exudate becomes organized and is finally developed into a dense fibroid or cicatricial tissue, which acts as a protective barrier against the spread from that particular area of invasion. This barrier in a great number of cases remains effective and in a sense imprisons or restricts the production of poisons and lessens their dissemination. Within this limited zone of action the bacterial products cause necrosis and in the dead tissue the germ is suppressed or actually destroyed. After the local necrotic process is complete the necrotic contents are more or less fully absorbed and only a cicatrix remains to mark the site of the original lesion, or if this does not take place the caseous and degenerated mass undergoes calcification. In either instance a natural process tends to bring about a cure.

If for any cause this protective barrier is removed by absorption while the imprisoned bacilli are still alive, or if the wall which is formed is incomplete, the bacilli escape and speedily infect adjoining or distant areas, being conveyed by the lymph or blood streams. Such is the explanation of those cases in which a patient who has suffered from some acute infection, like pneumonia, typhoid fever, or influenza, speedily develops tuberculosis during convalescence, although the acute illness may, by confining him to a healthful and well-ventilated room,have protected him from any recent infection.

In still other cases the protective barrier of surrounding exudate is not formed and the amalgamation of tubercles produces a nodule which undergoes necrosis and softening and its bacterial contents become diffused into the surrounding tissues, thus spreading the infection. In still a third type of cases the lesions consist in a diffuse exudative process, with little or no tubercle formation, and as a consequence we find that it is possible for the entrance of the tubercle bacillus to be followed by a tuberculous pneumonia or pulmonary consolidation, tuberculous serositis, or lymphadenitis, the lesion not containing the characteristic tubercle. In other words, in certain instances the tuberculous inflammation is so intense and the poison formed by the bacilli is so abundant or virulent, or the resistance of the soil so inadequate, that no attempt at protection is made, but instead there occurs a profuse exudative process which is extremely liable to undergo necrosis, and this results in rapid breaking down not only of the exudate itself, but of the involved tissues as well.

Under certain admittedly unusual conditions the tubercle bacillus becomes distinctly pyogenic and, rapidly developing in the lung, produces not only the degenerative and necrotic changes peculiar to tubercle formation, but fills the air vesicles with pus, serum, and dead epithelial cells and leukocytes, a state in which the part involved speedily goes on to widespread destruction. It is also to be recalled that in nearly all cases of tuberculous disease infection by other pyogenic organisms, such as the staphylococcus and streptococcus, aids in producing local inflammation and pus, and leads to the formation of toxins which cause local and general impairment of vitality.

In some cases, on the other hand, the bacilli, in the presence of the resistance offered, do not seem capable of originating an acute inflammatory process, nor do they cause the formation of tubercles with caseation, but produce a condition in which an excessive formation of connective tissue occurs, which prevents the rapid spread of the disease and constitutes a form of infection called *chronic hyperplastic tuberculosis* in the intestine or in the lung which is closely allied to fibroid phthisis, so called; but this fibroid process is by no means entirely dependent upon the presence of the bacillus tuberculosis, since other causes may produce it.

It is fully established that the action of the tubercle bacillus in the human organism is due to its poisons, a number of which have been described. The early coagulation necrosis and subsequent caseation are clearly the result of bacillary toxins. The tendency to fibrosis seen in many cases has been thought to be due to a sclerogenous toxin and the frequent cheesy disintegration, so commonly present, to a caseogenous poison; it is probable, however, that the same noxious agent in some individuals induces caseation and in those more resistant to its action a fibroid or fibrocalcareous change. Progressing caseation may be looked upon as an evidence to low resistance, while fibrosis, with or without calcification, indicates strong reparative and antagonistic powers on the part of the affected tissues.

With these preliminary remarks we may proceed to a discussion of the various manifestations, pathological and clinical, which are to be met with in persons affected by this disease.

Acute Miliary Tuberculosis.—By acute miliary tuberculosis is meant a condition in which a single organ, or a number of organs, or perhaps the whole body, is infected by the *Bacillus tuberculosis*, causing the formation of innumerable tubercles of the type already described. It arises not by the inhalation of dust laden by bacilli, but by the escape of bacilli in large numbers from some infected focus, as, for example, a caseous lymphatic gland. The escape takes place into a bloodvessel, and in a few hours at most the bloodvessels of the neighboring parts, or perhaps of the entire body, are swarming with bacilli, so that in a very brief space of time the lungs, the liver, and other parts are found studded, or, as it has been well expressed, "stuffed," with miliary tubercles. The caseous gland or primary cheesy nodule which gives origin to this acute secondary infection is usually so situated that it is adherent by inflammatory products to a vein, commonly the pulmonary vein or one of its branches, or to the thoracic duct, or the superior cava. By a process of extending necrosis the soft contents of the gland, laden with tubercle bacilli, break into the vessel or duct. Sometimes an active tuberculosisof the wall of the bloodvessel is present, so that tubercles may be found in the intima. Councilman has observed a tuberculous aortitis apparently resulting from infection through the vasovasorum.

Weigert has divided the results of this vascular invasion by the bacilli into three classes: (1) that in which all the organs of the body become filled with tubercles of the miliary type; (2) that in which the disease, though widely disseminated, nevertheless appears in multiple but widely separated foci; and (3) that in which the tubercles are not so numerous, their growth is more chronic, and their size causes them to be classed as nodules. This is manifestly an artificial division which is not closely adhered to under natural conditions, for the process may represent all these types in one case, while in other instances it may be impossible to tell to which class the case belongs because the lesions shade into one another.

Symptoms.—The symptoms of widely diffused miliary tuberculosis are to be carefully studied because they simulate those of enteric fever and other typhoid states so closely that not rarely an erroneous diagnosis is made. The chief manifestations may be said to be those of profound toxæmia without any localized lesion to explain the illness, which begins with the *general wretchedness* common to the early stages of all acute infections and which is followed by *fever*, rising each evening to 102° or 103°. The *pulse* is *unduly rapid*, and there are often *profuse sweats*. In such cases there are three symptoms which, while not pathognomonic by any means, are nevertheless of some value in separating this condition from typhoid fever. The fever is often irregular, sometimes breaking with a profuse sweat. In other cases it is higher in the morning than at night. The pulse is often exceedingly rapid in the early stages, a phenomenon which, as a rule, is not observed in typhoid fever. The temperature does not resist cold sponging in the first week as does that of typhoid fever, but, on the contrary, falls with great rapidity to below normal. Later there is the absence of rose spots to lead one to a correct opinion, and careful examination of the lungs may reveal some area of infiltration or softening, or of dulness on percussion which should arouse suspicion.

Additional differential factors are as follows: There is absence of the Widal reaction. This reaction, however, often does not appear in typhoid fever until after the tenth day. Tubercle bacilli may, by spinal puncture, be found in the cerebrospinal fluid, and in some instances the ophthalmoscope will reveal tubercles in the choroid. Rarely the bacillus can be demonstrated in the blood or urine. Enlargement of the spleen, the diazo reaction in the urine, the presence of active diarrhœa or severe constipation, and the fever are not differential points in favor of typhoid, for they all appear in miliary tuberculosis. Unlike typhoid fever, *herpes labialis* may be present in acute miliary tuberculosis.

In other instances the disease has a much more *abrupt onset.* The patient is seized with a chill followed by *high fever*, or *rapid pulse, profound prostration*, and *copious sweats*. Emaciation proceeds with remarkable rapidity. The aspect of the patient is profoundly toxic or septic and his expression anxious. The tongue is dry and the cheeks flushed.

When *miliary tuberculosis* involves the *lungs* the pulmonary symptoms are chiefly those of diffuse acute bronchitis, although careful examination may reveal at one apex, or at both, some impairment of resonance due perhaps to an ancient infection. The general symptoms are distinctly asthenic, as already described, and added to them there are fine rales over the greater part of the chest and a degree of *dyspnœa* far out of proportion to the lesions which can be discovered. The respirations may be unduly rapid. The *cyanosis* is very pronounced, the *cough* constant, and the patient may seem surprisingly ill considering that no cause can be discovered. The *sputum* may be *rusty or blood-streaked*, or a true *hæmoptysis* may develop. Auscultation may reveal *pleural friction* due to tuberculosis of the pleura, and as the case progresses widely distributed rales may be heard in the back and front of the chest. In these cases great mental anxiety is often a marked symptom unless the disease attacks a child, when the patient usually lies limp and apathetic and perhaps stuporous.

These patients usually die in from one to three months, but cases are occasionally met with in which death ensues as early as the fourteenth day. In still other instances the case becomes less fulminating in character, the symptoms moderate, and the patient passes into ordinary subacute pulmonary tuberculosis. Acute tuberculous bronchopneumonia is more frequently seen in children than in adults.

Diagnosis.—The diagnosis is not difficult in the pulmonary form if there is a history of an old tuberculous lesion elsewhere, or if the marked cyanosis as compared to the apparent limited area of disease is considered. Here again the presence of tuberculous foci may, if found, show that an acute condition is imposed upon an older one. The physician must not be led into the belief that the lungs are normal because he is able to elicit a clear and resonant percussion note on the chest wall, for a compensatory emphysema often is present in these cases.

When the miliary process chiefly or entirely involves the meninges the symptoms are of course cephalic in large degree, and we have that grave state known as acute tuberculous meningitis present.

Spinal puncture to determine the cause of the disease is a most valuable aid. If tuberculosis is present the cerebrospinal fluid will be turbid and occasionally, if it is placed in a centrifuge, the bacilli can be found, or some of the fluid may be injected into a guinea-pig, which will develop tuberculosis, if this be the cause of the illness.

From typhoid fever of the meningeal type tuberculous meningitis is separated by the presence of spots, by the diazo reaction, and the Widal test. Again, the diagnosis of tuberculous meningitis may be confirmed if a focus of primary tuberculosis can be found in the other organs as in the lungs, the bones, or the mesenteric glands.

Prognosis.—The prognosis is always fatal, although cases said to have recovered have been reported

Treatment.—The treatment consists in the use of nutritious food and stimulants and in the relief of restlessness by chloral or the bromides.

Glandular Tuberculosis.—Glandular tuberculosis, or tuberculosis of the lymphatic glands, is the condition which was formerly called "scrofula" before Koch demonstrated the existence of the tubercle bacillus. It is now known that no such disease exists as scrofula, or scrofulosis, in the sense of a separate entity.

Tuberculosis of the lymph glands is often a very mild form of the infection and the mortality from its presence is very low. Indeed, it may be said that if the infection does not escape to other parts of the body life will not be seriously jeopardized.

In studying this state it must be recalled that one of the important functions of the lymph nodes is to arrest and perhaps destroy such micro-organisms as may endeavor to enter the general system. As soon as pathogenic germs enter a healthy gland one of several processes takes place. In a strong individual with great vital resistance the gland becomes enlarged and active in an evident endeavor to destroy the invaders. In this it may succeed, or the few bacilli which escape are caught and destroyed by other adjacent glands. If the infection is virulent and the vital resistance is below par, the battle is more prolonged, the inflammation in and about the gland is more active, and the general system may be saved by the additional safeguard of a wall of protective tissue thrown around the infected gland to protect the rest of the body. In still other cases the glands go on to caseation and the necrotic contents escape externally or even internally. It is a noteworthy fact, however, that this so-called pus is usually sterile or contains bacilli in such small numbers as to be demonstrable only by inoculation experiments. Again, if the infection wins the battle and the gland undergoes caseation, it is still possible for the area to be surrounded by a fibrous barrier which walls up the caseous mass and its bacilli and protects the body even though the gland is destroyed.

When the protective processes fail the bacilli pass the lymph nodes, or a caseous gland in juxtaposition to a bloodvessel breaks, and general tuberculosis ensues as already described.

Tuberculous infection of the lymph nodes takes place in four chief areas:

the cervical glands, the mediastinal glands, and the mesenteric and retroperitoneal glands. In the first class the infection takes place through the tonsils, in the nasopharynx, or because of the presence of bad teeth or a break in the gums. The mediastinal glands suffer by the entrance of the bacillus through the mucous membrane of the larynx, bronchial tubes, or smaller bronchioles, while the abdominal lymphatics receive their infection from the intestines.

The diagnosis of *cervical adenitis* is not difficult, although occasionally, when the disease is bilateral and the swelling is great, the possibility of Hodgkin's disease may have to be considered.

The involvement of the *mediastinal glands* by tuberculous infection results in a spread of the disease to the retroperitoneal lymphatics or in the growth of the tissues affected to such a degree that lymphatic tumors may be formed which cause serious symptoms by pressure. Thus, the recurrent laryngeal nerve may be pressed upon and laryngeal spasm result, or the superior vena cava or pulmonary vein suffers from compression, and in a similar manner bronchial obstruction may ensue. More important, however, than the pressure symptoms are possible perforation by ulceration of the bronchi or trachea, or even of the bloodvessels with rapid diffusion of the infection all through the body. So, too, it is possible for bacilli to enter the lung, to pass to the lymphatics, to cause disease in these glands, and finally cause pulmonary tuberculosis, pleural tuberculosis, or pericardial tuberculosis by softening and rupture into these parts through the adhesions which are formed.

In diseases of the retrobronchial glands auscultation over the upper end of the sternum, when the head is well thrown back, may reveal a tracheal hum, and careful percussion may elicit some dulness.

When the *mesenteric* and *retroperitoneal glands* are involved, producing what is called "tabes mesenterica," the child is anæmic, poorly nourished, has constipation alternating with diarrhœa, and presents an enlarged abdomen ("pot-belly"). The size of the belly as compared to the rather wasted arms, legs, and thorax is noteworthy, and careful palpation may occasionally reveal enlarged glands deeply situated in the abdominal cavity. This condition is to be separated from tuberculosis of the peritoneum and from consumption of the bowels, for both of these structures are usually free from the disease in these cases, although they may be infected by softening of the glands themselves. These lesions probably exist in a far larger proportion of cases than is generally thought, and end by a process of fibrosis and calcification, for the involvement of these glands is met with in many cases at autopsy when death is due to another cause, and when no suspicion of tuberculous infection has been present.

Treatment of Glandular Tuberculosis.—Tuberculosis of the retrobronchial glands and of the retroperitoneal glands can be treated only by sunshine and fresh air with residence by the sea, and by the internal use of tonics, of syrup of the iodide of iron and cod-liver oil to combat anæmia.

Persistent enlargement of the *cervical glands* demands their surgical removal, not their incision, but their excision, because, as has already been stated, a tuberculous focus is always a threatening focus. On the other hand, it cannot be denied that large numbers of very healthy adults bear scars showing that they have had cervical adenitis in early life. In these cases the battle between vital resistance and tuberculous infection has been won by the individual.

Tuberculosis of the Serous Membranes.—Tuberculosis of the serous membranes may be divided into the acute and chronic forms. The acute is further subdivided into (*a*) an acute serofibrinous form, macroscopically identical or indistinguishable from serofibrinous serositis arising from other causes; (*b*) an acute miliary tuberculous serositis due to the invasion of the serous membrane by tubercle bacilli and the formation of miliary tubercles. The two forms just mentioned may be distinct or coincident. The chronic tuberculous serositis may be (*a*) fibrocaseous or (*b*) fibrohyaline. The former results from the formation of tuberculous exudates, in which extensive caseation gives rise to cheesy accumulations of various sizes surrounded by granulations or more fully organized fibrous tissue. Marked calcareous change is frequently associated with this form. The fibrohyaline type is characterized by marked thickening, and the formation of adhesions by newly developed fibrous tissue of a peculiar, grayish, translucent form. Both the chronic forms may occur together, and the caseous masses may be enclosed by hyaline fibrous tissue of the type just mentioned.

Acute Meningeal Tuberculosis.—Meningeal tuberculosis is an inflammation of the pia mater produced by an infection of this membrane with the bacillus tuberculosis and accompanied by an effusion of lymph, it may be the formation of pus, and the development of tubercles. These tubercles are usually very minute, but occasionally are large from the amalgamation of several tubercles into one. They are most profuse at the base of the brain, hence the name *basilar meningitis*, and extend upward on its sides following chiefly the vascular pathways. In some instances, however, the pia mater on the convexity of the brain contains more tubercles than exist at the base. Nearly always at the base there is a copious exudate of lymph which produces a pearly, gelatinous appearance. The lateral ventricles are distended with fluid. Minot states that Robert Whytt, of Edinburgh, in 1768, first accurately described this condition, although he had no clear conception of its cause. Guersant, in 1827, reported that the pathological appearances of the membranes were of so peculiar a type that he suggested the name "granular meningitis." In 1830 Papavoine described the disease as a true tuberculous lesion, and the condition of moderate hydrocephalus which existed with the meningitis was recognized as having its origin in the tuberculous infection. It was reserved for W. W. Gerhard, of Philadelphia, in 1833, to show not only that this type of meningitis was tuberculous, but that it was practically in every instance secondary to some tuberculous lesion elsewhere.

SYMPTOMS.—The symptoms of acute meningeal tuberculosis are very characteristic, whether it occurs in children or adults. It is much more frequently seen, however, in children between two and seven years. These symptoms are best divided into three stages for study. At first the parent notices that the child is unusually *peevish* and *irritable*, or in other cases *peculiarly languid* and indisposed to play. There is little restful sleep and the child often has *night terrors*. The *appetite is capricious* and the bowels irregular. After these symptoms have lasted for some days, during which time the tubercles have probably been deposited in the pia mater, the well-developed symptoms of the disease appear. *Headache* may be constant and is characterized by *sharp exacerbations of pain* which cause the child to give a peculiar *high-pitched scream* which is quite characteristic. Sudden attacks of *vomiting* of an explosive character may occur. At times a fleeting *delirium* may be present.

It is almost impossible in many of these cases to exclude early typhoid fever, for a similar train of symptoms may be presented in its early stages.

The *temperature* is usually elevated, rising as high as 102° or 103°. The *pulse* is *slow* and the *respirations* are *irregular* and *sighing*. *Rapid emaciation* takes place, and if the child be very young, so that the fontanelle is open, there may be distinct hydrocephalic enlargement. The patient now lies *stuporous* or *somnolent*, with the eyes half closed. Indeed, the appearance may be that of deep sleep with sighing breathing.

In some cases in which the onset of the affection is rather acute, the patient suffers from a series of *convulsions*. I have seen such a case with Dr. Brouwer, of Tom's River, New Jersey, while preparing this article, in which the child had as many as sixty convulsions in twenty-four hours.

As the exudate increases symptoms of intracranial pressure and signs of interference with the cranial nerves appear, so that squint, twitching of the facial muscles, and chewing movements of the lips and jaws develop.

Even as late as this the patient may be aroused and will seem so much better for a time that the friends are much encouraged, but a relapse inevitably occurs. The child is now too stuporous to be roused, the eyes are filled with sticky secretion, and the parents find solace in the belief that even if hopelessly ill the patient does not suffer. This solace is, however, occasionally rudely dispelled by a shrill, piercing cry which, interrupting the profound stillness, is more than usually startling. As death approaches the pulse becomes very rapid, probably from vagal paralysis, the pupils no longer react to light, and the eyeballs are rotated upward.

The duration of the entire illness is about ten to eighteen days, as a rule, but cases may die as early as the end of five days, in a convulsive seizure, or they may last for several weeks.

DIAGNOSIS.—The diagnosis of acute miliary tuberculous meningitis must be made with the recollection that the following conditions simulate it: Acute meningitis not due to tubercle is rare in children, has a more sudden onset, as a rule, and ends in a week in most cases. The delirium accompanying it is more marked, the febrile movement is more sharp, and there is usually no history of tuberculosis in the parents, as there is in the tuberculous case in many instances.

From cerebrospinal meningitis of the epidemic type it is separated by the sudden onset of that disease, by the absence of its eruption, and by the fact that no cases of cerebrospinal meningitis have occurred in the vicinity. By spinal puncture the meningococcus may be obtained in one case and the tubercle bacillus in the other, although the latter is not frequently discovered.

The fluid in cerebrospinal fever contains polymorphonuclear cells and hyaline leukocytes, while in tuberculous meningitis recent studies seem to show that a lymphocytosis is the rule. (See Cerebral Spinal Fever.)

Tuberculous Pleurisy.—Tuberculous pleurisy is, in the vast majority of cases, secondary to tuberculous infection in other parts. Most commonly the primary focus is in the lungs or in the mediastinal glands. In some instances the process is the result of a general infection which results in miliary tuberculosis, and in these instances it not infrequently happens that the pleura is involved without there being any tuberculous process in the lungs. Thus Hodenpyl, in 91 autopsies on persons in whom the lungs were free from tubercle, found miliary tuberculosis of the pleura in 41, both the parietal and visceral layers being affected. He also believes that miliary tuberculosis of the pleura is apt to undergo fibrous changes.

The tuberculous lesions are of three types. In the *first* type we find scattered patches of tuberculous deposit which are the continuation of a tuberculous process in the lung beneath the visceral layer of the pleura, or similar patches are found which are independent of lung involvement, or again patches appear upon the parietal pleura. In the *second* type the lesions are simply those of a widespread miliary tuberculosis of the pleura, and in the *third* type, which is representative of a more chronic or slow process, there is great thickening of the pleura, partly as the result of the organization of formed exudates and also of proliferative changes in the primitive serous layers. Throughout this tissue and exudate miliary tubercles, or masses of miliary tubercles, appear. These undergo coagulation necrosis in some instances.

The presence of any one of these processes usually results in the outpouring of a certain amount of *effusion* which is often serous and by no means rarely purulent. When it is serous it is lacking in fibrin and it may be tinged with blood. The physician should recall the important clinical fact, in connection with these tuberculous pleural effusions, that whether they be serous or purulent, an examination of the fluid will rarely reveal the tubercle bacillus unless in some manner these organisms are dislodged from the pleural surface by scraping. In some cases the effusion is not due to the bacillus tuberculosis alone, but to an associated infection. Thus, in the purulent type the pneumococcus, streptococcus, or staphylococcus pyogenes are often found. (See Empyema.)

Reference has already been made to the fact that the visceral layer of the pleura is often infected by a tuberculous process in the lung as a result of direct extension. It may be added that in nearly every case an inflammatory area—that is, a localized pleuritis—exists over the seat of the disease in the pulmonary tissues. This condition often gives rise to *pain in the chest* and not rarely causes adhesions between the layers of the pleura. The inflammatory process, while tuberculous in origin, is not necessarily tuberculous in character, but it often becomes tuberculous as already stated.

Sometimes when the tuberculous mass in the lung softens and breaks down the visceral layer of the pleura is perforated and *sudden dyspnœa* and *pain* ensue, with the production of *pneumothorax*. (See Pneumothorax and complications of Pulmonary Tuberculosis.) Through this opening infection with pyogenic bacilli occurs or the *Bacillus tuberculosis* becomes pyogenic, and as a consequence pyopneumothorax develops. (See Pyopneumothorax.)

Tuberculosis of the Pericardium.—Like tuberculosis of the pleura, pericardial tuberculosis is usually secondary to primary infection elsewhere. It may, however, be primary. It occurs in two forms: the miliary, in which the small tubercles are scattered or profuse, and in a form in which the entire pericardium, both in its visceral and parietal layers, is thickened by an inflammatory exudate which is associated with the development of tuberculous masses which undergo cheesy change. In this type the pericardial space may be nearly obliterated by the adhesions which are formed between its layers. It is a noteworthy fact that although the pericardium is so near the lungs and pleura it is, comparatively speaking, rarely infected. Out of 1048 autopsies Wells found tuberculous pericarditis only 16 times and in 4500 autopsies Baginsky found it 15 times. In 1317 autopsies on phthisical patients Willigk found tuberculosis of the pericardium 11 times. Leudet found it 8 times in 299 autopsies. In 1000 autopsies Osler found 7 cases of tuberculous pericarditis. Ellis has recently reported from the laboratories of the Jefferson Medical College a case in which the heart, the pericardium, and the mediastinal tissues formed one large, adherent mass of tuberculous nodules.

The *symptoms* presented by tuberculosis of the pericardium may be so slight that no suspicion of the pericardial disease exists during life, or they may resemble those of mediastinopericarditis, or adherent pericardium. (See Adherent Pericardium).

Tuberculosis of the Peritoneum.—Aside from tuberculosis of the lungs, tuberculosis of the peritoneum is the most frequent and most important manifestation of tuberculous infection met by the physician. The statistics of Grawitz and Brunn show that in 13,422 autopsies tuberculosis of the peritoneum was found 284 times.

In 2802 autopsies on tuberculous subjects, collected from various sources, the peritoneum was involved in 571, a percentage of 20.36. These figures represent all ages. Steiner found the peritoneum affected in 92 out of 800 cases of tuberculosis occurring in children, or in 11.5 per cent.

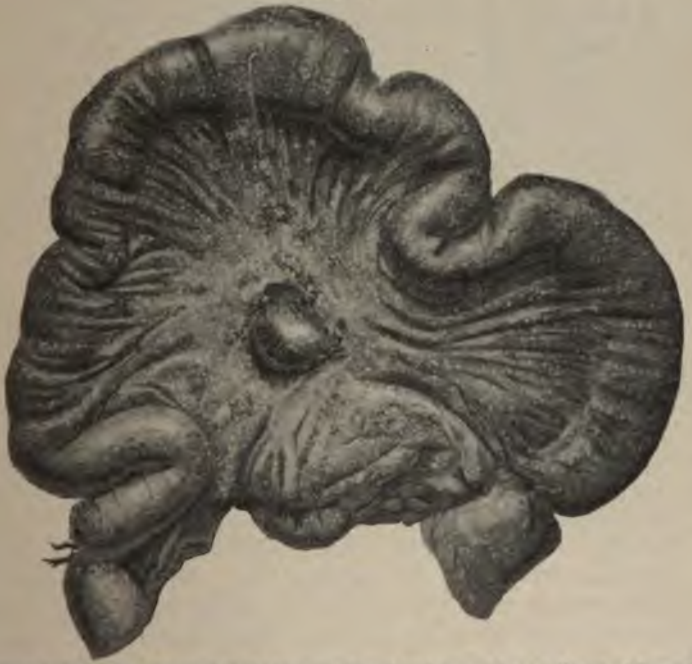
As to the relative frequency of the disease in adults and children Aldibert's statistics, based on 326 cases, are of interest. Of these 326 cases, 274, or 84.05 per cent., occurred in adults and the remaining 52, or 15.95 per cent., occurred in children. It is in a very large proportion of cases secondary to tuberculous foci elsewhere. The combined statistics of Munstermann, Borschke, and Pribram, comprising 437 cases of tuberculous peritonitis examined postmortem, showed that only 3 were primary.

A tuberculous family history is present in 53 per cent. of cases. The disease is much more common in the female than in the male; according to Nothnagel 90 per cent. of the cases are females. König's statistics make it 78 per cent.

Tuberculous peritonitis is of especial interest not only because of its frequency and gravity, but because it is, in one type at least, more readily cured than any other well-developed form of internal tuberculosis. It occurs in three chief varieties, viz., as an acute miliary tuberculosis, as a chronic tuberculosis with large nodules and adhesions, and as a still more chronic form with fibroid changes. The relative frequency of the different forms of tuberculous peritonitis is shown by the following facts: In 46 cases which came under the observation of Munstermann, 25 were exudative, 21 were plastic, and 8 were chiefly caseous. Of the 21 plastic cases 8 were fibrous. Herringham found fibrous adhesions in 18 out of 50 cases. Borschke found the miliary form in 16 out of 226 cases which came to autopsy.

The *acute miliary form* is usually secondary to infection of the mesenteric and retroperitoneal glands, but occasionally in women the infection comes from the Fallopian tubes, or in males from the bladder or other part of the genito-urinary apparatus. The miliary tubercles are scattered widely over the peritoneum on both its visceral and parietal layers (Fig. 45), and the surface of the liver is often profusely peppered by these formations. In these cases there is a serous effusion into the peritoneum which in some instances is very profuse, particularly if the case is rather subacute in the rapidity of its course.

FIG. 45



Miliary tubercles of the surface of the small bowel and mesentery. (Kast and Rumpler.)

The mode of its development is well illustrated by a case I saw some years since with my associate, Dr. Thornton. A girl of about twelve or thirteen years, while in apparently perfect health, was bathing in a pond and playing with a small row-boat, the sharp prow of which struck her a severe blow on the epigastrium, which made her nauseated and faint. She speedily began to lose weight and strength, became distinctly emaciated, and rapidly developed a marked ascites. The abdomen was opened and every peritoneal surface was found literally covered with tubercles. The fluid was allowed to escape, drainage was permitted, and perfect recovery followed. Undoubtedly the blow on the belly ruptured a tuberculous mesenteric or retroperitoneal gland, and so produced general peritoneal infection. In such a case no nodules can be found in the belly on palpation, but the presence of ascites in a child, or in an adult, without any signs of hepatic disease should lead the physician to suspect tuberculosis of this type, particularly if in addition there is present some fever of an irregular type, which is commonly moderate, but which may rise at times as high as 103° or even 104°

How opening the belly and permitting drainage cures these cases is not known, but the clinical fact that such a result is often achieved is not to be denied, and this holds true even if this condition develops in adults. Walter's statistics show that 50 per cent. of adults who are subjected to laparotomy recover, and Herzfeld's statistics give a recovery percentage of 62 per cent. for children. Marganecci gives 85 per cent., von Krencki 71.5 per cent. Thomas 73 per cent. Hall reports 94 cures out of 110 operations.

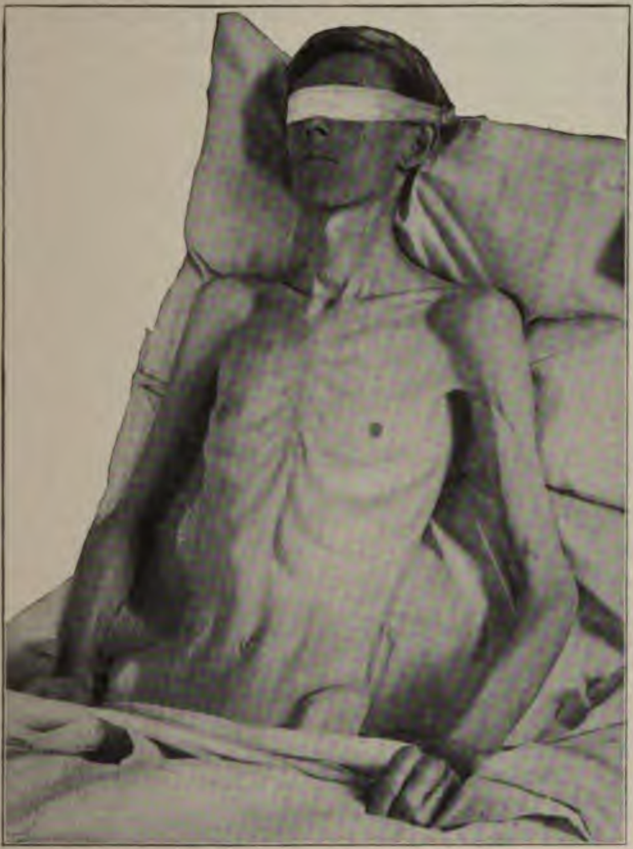
The second *caseous type* of peritoneal tuberculosis with nodules is characterized by the presence of caseous masses of tubercle which tend to ulcerate, which are associated with seropurulent, or purulent, effusion in moderate amount, and in which the belly cavity is not distended by fluid nor the intestines by gas, as in the miliary form just described, but is apt to present a peculiar pasteboard rigidity. The effusion in these cases is often sacculated by reason of the fact that there are formed adhesions which wall off spaces in which the fluid collects. These spaces may be between coils of intestine or contiguous mesentery, between the intestine and omentum, and between the omentum and the parietal peritoneum.

In some instances these sacculations are capable of containing but a few drachms of fluid, but in those cases in which fairly large peritoneal areas are separated from the general peritoneum by adhesions very large accumulations of fluid may be present. If this takes place in the flanks or lower zone of the abdomen, where it usually occurs, the symptoms may very closely resemble ovarian cyst, and many cases have been operated upon with the idea that ovarian disease was the cause of the fluctuating mass. Careful palpation under ether may reveal an irregular nodular edge to the growth, or nodules elsewhere may explain the real state.

The *chronic fibroid type* may resemble the nodular type just spoken of, but in it the matting of the abdominal contents into a small compact mass is quite extraordinary, the intestines and omentum being glued together in an adhesive bundle which cannot be separated. In this type the belly is often remarkably scaphoid and the degree of general emaciation extraordinary. Some idea of its degree may be gathered from the illustrations shown in Figs. 46 and 47.

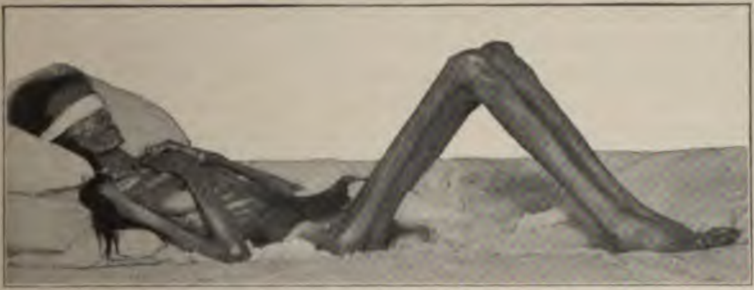
The *symptoms* of chronic fibroid peritoneal tuberculosis are characteristic. In addition to the *general emaciation* it will often be found that the *skin* over the abdomen looks and feels peculiarly *rough* and *scurf-like*, or as if there was marked "goose flesh" over this part. In addition it is often stained a

FIG. 46



A case of peritoneal tuberculosis of the fibroid type in a man aged twenty-one years, with great general atrophy and scaphoid belly.

FIG. 47



Extreme emaciation in a woman due to thoracic and peritoneal tuberculosis of the fibroid type.

curious dirty yellow or is light brownish in hue. The *abdominal wall* is not only *hard*, but the abdominal muscles are readily felt by the finger-tips, while deep *palpation* reveals *nodules* or gives the sense of abdominal vacancy as if the patient had been eviscerated. The *temperature range* is not markedly febrile, and often is subnormal, ranging from 96° in the morning to 99° at night.

It is not, however, in these well-advanced forms of the disease that the physician has difficulty in making a diagnosis, but in those cases in which there is general impairment of health without marked general emaciation; and it may be without distinct abdominal symptoms save *obstinate constipation*, with occasionally attacks of *active purging*, or in those instances in which the patient is still hale and robust, but suffers from some abdominal distress. These cases often present a *distinct abdominal tumor*, or *tumors*, composed of tuberculous nodules, or of nodules combined with thickened knuckles of intestine which may be so firmly held by adhesions of exuded lymph that gas and feces produce a tumor that cannot be readily dispelled by pressure.

Sometimes a tumor is met with in the epigastric area extending across the abdomen at this level, or just above the umbilicus, formed by a peculiar rolling of the omentum as a workman would roll up his apron and stow it under his belt. A similar condition is sometimes found in carcinomatosis of the peritoneum, but such a roll is usually due to tuberculosis.

When the mesenteric glands or retroperitoneal glands are gravely infected, a single nodule or several nodules may be easily palpated. When multiple they are usually tuberculous, but where it is single a careful exclusion of malignant growth must be made by finding a tuberculous focus elsewhere or by the tuberculin test.

Treatment of Tuberculosis of the Peritoneum.—The *treatment* of the subacute or chronic forms of peritoneal tuberculosis consists in operation. The first thing to be looked for at operation in peritoneal tuberculosis in the female is the Fallopian tubes, for they are the cause in the majority of cases. If diseased they must be removed, for such tubes will persistently infect the peritoneum.

The operative treatment is most successful, as a rule, when the state is characterized by sufficient effusion to keep the intestinal coils apart, and so prevent adhesions. The operation consists in a single opening of the peritoneal cavity and a free entrance into it of atmospheric air. Any attempt to remove the tuberculous masses is useless, unless a single mass can be excised without damaging the tissues and without the danger of setting free bacilli to cause infection elsewhere. In sacculated cases the sac should be incised, drained, and packed with iodoform gauze. In the chronic fibroid type operation will not be productive of much good, for it cannot result in the loosening of the shrivelled omentum or of the cicatricial contractions about the intestines, but in these cases cœliotomy may arrest the disease. When there are sacculations with accumulations of fluid or pus, the operation is of value in that it evacuates these collections and may arrest the process, but it does not promise complete cure as in the cases with large ascites. (For statistics see page 306.)

The medical treatment consists in active feeding with easily assimilated foodstuffs and in the use of cod-liver oil. If anæmia is marked the syrup of the iodide of iron may be given in alternate weeks with the oil, and I have certainly seen good results follow the use of nightly iodoform inunctions over the abdomen, a mixture of olive oil and iodoform in the proportion of 10 grains to the ounce being used. Iodoform suppositories, 5 grains each, may also be employed.

Pulmonary Tuberculosis.—Pulmonary tuberculosis, or pulmonary phthisis, as it is sometimes called because it causes such emaciation or wasting, is the most prevalent disease to which man is susceptible. It affects, as a rule, young adults or adolescents (see Frequency of Tuberculosis), but it may occur at any period of life, being comparatively rare in the first five years of existence and in the period of well-developed old age.

As a result of infection of the lung by the *Bacillus tuberculosis* we find three types of pulmonary disease: the *miliary*, the *chronic* or *caseating, ulcerative* type, and the so-called *fibroid type*. Of these the second form is by far the most common and the most important from the clinical standpoint. The infection takes place as a primary process through the entrance of the bacillus by the respiratory passages, or secondarily as a result of the transference of the bacillus from some primary focus by the bloodvessels or lymphatics.

Much discussion has arisen as to the mode by which the first focus of tuberculosis in the lung is produced. Birch-Hirschfeld proved that, in many cases at least, the bacilli gain their primary lodgement in a bronchiole, where the lung is least able to get rid of foreign matter by coughing, and that, from a primary tuberculous lesion at this point the rest of the lung becomes infected. Aufrecht also proved that the primary infection sometimes takes place through the circulation, to which the bacilli gain access by the tonsils and the alimentary canal, the pulmonary focus being due to a plugging of a vessel by their presence.

Whether the means of infection be respiratory or vascular, the ultimate lesions are often the same; but the early lesions differ, and the prognosis may be governed to some extent by the finding of a primary focus elsewhere which is responsible for the pulmonary lesions.

The early lesions of pulmonary tuberculosis, due to *infection by inhalation* (aërogenous infection), are found chiefly in the wall of a bronchiole and in the alveoli grouped around it and forming lobules. Either by extension from this infected lobule or by the fusion of a number of similarly affected lobules, large tuberculous masses are speedily formed. They are also characterized by the extension of the tuberculous infection to the tissues around the bronchioles, giving rise to an extending bronchopneumonia which is nodular in its character owing to the primary lobular limitations.

The early lesions of the form of tuberculosis of the lung which is due to infection by way of the *bloodvessels* or *lymphatics* are found in the walls of the alveoli—that is to say, in the connective tissue between the alveoli and in the interlobular capillaries. The disease may be well scattered through both lungs in either instance, but in the first type the patches are larger, involving, it may be, a lobule at a time, whereas in the second form they are diminutive and more of the nature of miliary tubercles as observed elsewhere. This we would naturally expect from what has been said of the cause of miliary tuberculosis. After the disease has existed some time the areas of tuberculous deposit in either case may attain the same size.

The discovery that there is a primary seat of tuberculosis, which has given rise to the pulmonary lesions, requires a graver prognosis because it indicates that there is more than one focus, and because such a primary lesion which has caused pulmonary disease by infection through the vessels may have caused other foci of infection elsewhere by the same means. Again, the disease is more apt to be generalized throughout the lung in this case than in the inspiratory form of infection.

Tuberculous infection of the lung, therefore, produces the following changes in the pulmonary tissues: The gray and yellow tubercles, which differ in no way from those tubercles already described as occurring elsewhere, become amalgamated and form caseous masses, with, sooner or later, the characteristic softening of the growth; if the tubercles do not undergo necrosis and fail to coalesce, the accompanying low-grade irritation or inflammation may lead to fibroid changes. Along with these changes in the tubercles themselves there is always associated a considerable amount of inflammation, which often results in the formation of an exudate which fills the air vesicles just as it does in croupous or catarrhal pneumonia. Side by side with the development of the tubercles in the lungs, and of the pneumonic exudate, there develops in the interstitial tissues themselves a process which is tuberculous and which causes thickening. This is the so-called tuberculous infiltration of Laennec. The lung, therefore, becomes solidified, partly as the result of the tuberculous growth, and partly as a result of the inflammation caused by the bacillus.

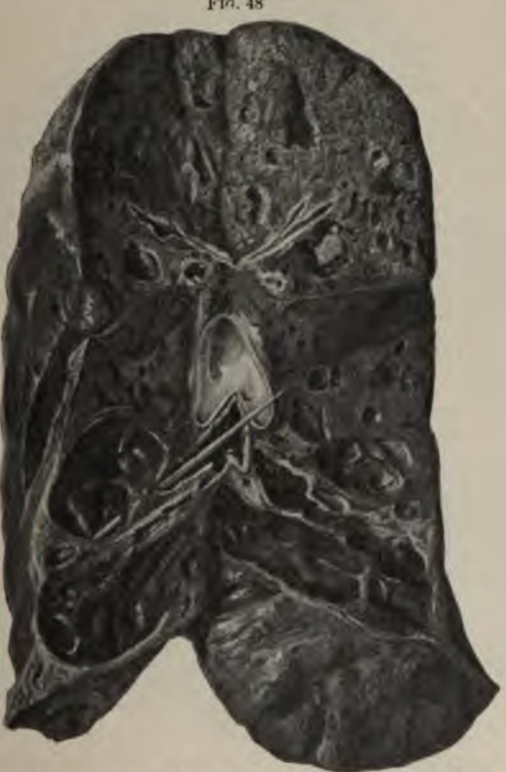
Sooner or later a large part of the infiltrated area undergoes caseation. Around this focus or area of active tuberculous process inflammatory changes occur, which may cause the neighboring parts of the lung to present lesions like those of catarrhal pneumonia. On making a section of such an area the lung presents a smooth, homogeneous surface, as does a piece of Castile soap or cheese (Figs. 48 and 49), but if the process is not far advanced in caseation it may show a peculiar gelatinous appearance, the so-called gelatinous pneumonia.

The fourth condition, which is noteworthy, is the lack of bloodvessels in the diseased portion of the lung, for no new ones are formed with the morbid growth and the ones naturally present are occluded by the disease which involves their coats and causes thrombosis; the resulting thrombus in turn undergoes caseation so that the vessels disappear in the tuberculous mass. These vascular changes possess great interest for this reason, and also because by this means the tubercle bacillus may enter the blood and infect other points, or by a process of ulceration of the vessel wall hemorrhages may occur.

All tuberculous lesions in the lungs are, therefore, very similar in character; all manifest a disposition to undergo similar reparative or degenerative changes, the alterations being differences in degree rather than in kind.

When the restrictive efforts of the affected organ are inadequate caseation extends until a bronchus is reached, through which the products of necrosis are removed by drainage and expectoration. Air takes the place of the material removed, and so a cavity is formed, the walls of which are lined by broken-down tubercle-containing material, which continually softens (caseation) and melts down, thereby enlarging the cavity. This cheesy material is loaded with bacilli in far greater numbers than they exist in the solidified part of the lung. The cavity is also infected by the pathogenic bacteria inhaled in the air, and these aid in the destructive local process and increase the general toxæmia.

FIG. 48



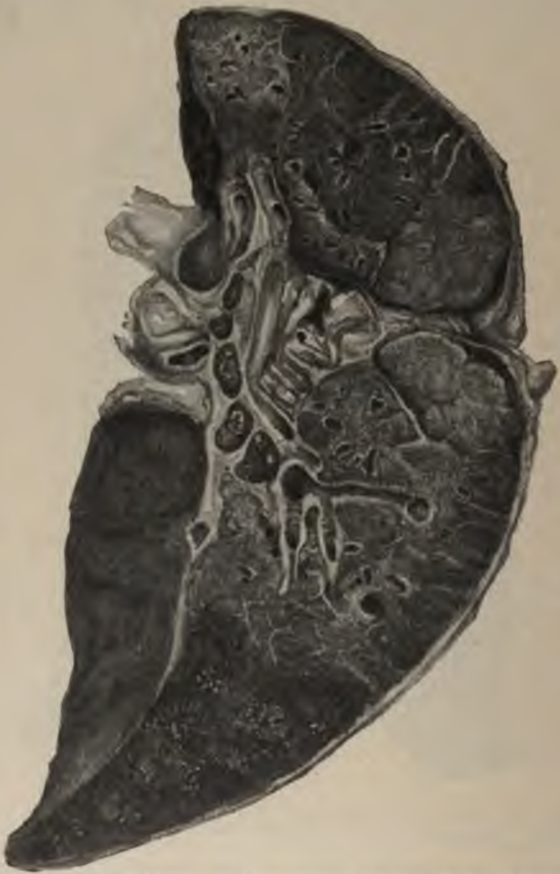
Caseous consolidation in the upper lobe and bronchiectasis in the lower lobe. (Kast and Rumpler.)

It is interesting to note that the smaller bronchioles are usually closed by tuberculous infiltration as the disease progresses and only the larger ones remain patulous. These communicate with the cavities by small lateral orifices as the tube courses along the wall of the excavation, or open into the cavity like the small papilla of a duct. The walls of the bronchial tubes which provide drainage for the cavities are often the site of tuberculous ulceration.

Tuberculous cavities are of two classes, moist or secreting, and dry. The first is that met with in the acute types of the disease and it often increases in size very rapidly. The contents of this cavity are usually composed of caseous matter, broken-down lung-tissue, pus cells, and tubercle bacilli, and the walls of the cavity suffer from active ulceration.

The dry cavity, on the other hand, is found in the chronic cases which often last for years; efforts at repair smooth the wall, in which fibrous tissue develops, and it not rarely happens that by the fibroid process already described as occurring in this disease the size of the cavity is greatly decreased. These cavities contain but little material beyond a small amount of pus, and from their walls hemorrhage may rarely occur as the result of erosion of large vessels. Secondary cavities are due to the spread of the infection by the vessels or bronchi, and follow the secondary caseation process already described.

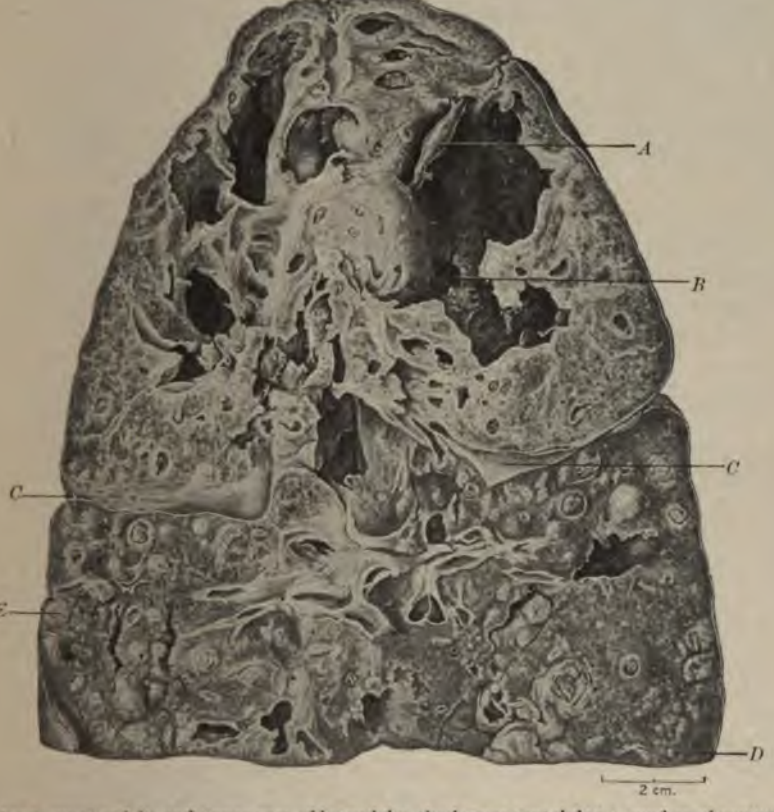
FIG. 49



Caseous consolidation above. Red hepatization below. (Kast and Rumpler.)

We have now passed over the stage of pulmonary infection, consolidation, caseation, and disintegration, and come to the study of the processes often instituted in *reparation*. This is not a part of the tuberculous process. The small-cell infiltration and exudation in the inflammatory zone surrounding the area of infection sometimes escape speedy involvement in the tuberculous process, and instead of degenerating rapidly aids in the production of fibrous tissue. At first it is immature and imperfect in character, but as time passes it becomes firm, dense, and fully-formed fibrous tissue. If the tuberculous focus is small it may be completely encapsulated by this fibrous covering, with the result that the caseous mass becomes calcareous or is gradually absorbed so that only a puckered scar results.

Fig. 50



Left lung, superior lobe and upper part of lower lobe, the former containing a number of communicating caverns, brought about by tuberculous infiltration, caseation, and evacuation of the contents through the bronchi: *A*, aneurysmal dilatation of an artery spanning one margin of a large cavity; *B*, communication with another cavity; *C*, *C*, thickened and adherent pleura between the two involved lobes. The pleura over both lobes is thickened, and at the autopsy the cavity had been obliterated by universal adhesion; *D*, the pointer from the letter *D* leads to a small group of tubercles in which caseation is just beginning; *E*, a fused group of tubercles, further advanced than at *D*.

When a *cavity heals* its walls undergo cicatricial contraction, but it is probably never obliterated unless it has been exceedingly small before the healing process began.

The growth of fibrous tissue is most marked in those parts of the lung which ordinarily possess the greatest amount of connective tissue, as in the interlobar and interlobular portions of the pulmonary tissues.

If a large cavity is present bands of fibrous tissue may persist, and, stretching across it, form trabeculæ (Fig. 50). At times these trabeculæ consist largely of good-sized bloodvessels which have not been plugged by the tuberculous process. If they are perforated by ulceration, so violent a hemorrhage may occur as to cause death, even though the process of advancing cicatrization is endeavoring to limit the progress of the disease. At times the lesion in the bloodvessel develops into an aneurysm, and this may rupture, causing hæmoptysis.

Finally, we find still another process designed to arrest the disease and save the patient, namely, thickening of the pleura which protects the pleural cavity from pneumothorax and which, as it undergoes fibroid change, contracts and so acts as a sort of fibrous capsule of the entire diseased lung.

Fibroid tuberculosis of the lungs is a very chronic condition, already described from the pathological standpoint, and characterized by marked overgrowth of fibroid tissue in the affected organ. A somewhat similar state exists when no tubercles have been present. The primary areas of tuberculous invasion become invested by fibrous tissues so that tuberculous bronchopneumonia becomes gradually changed into one of fibrous overgrowth, with shrinkage of the parts so that the lung becomes much decreased in size and even the chest may be sunken and deformed. These thoracic changes are, however, more marked in those cases in which the visceral pleura is also involved in the cicatricial or fibroid process. The disease loses many of the symptoms of ordinary pulmonary tuberculosis, and while the constricted cavities may contain bacilli and the bronchial tubes provide copious material for expectoration, the process of general wasting goes on very slowly and the strength does not decrease with any speed. Such conditions are usually seen in patients of middle life and may last for ten to twenty years. Death finally comes from dilatation of the heart or from an acute complicating pneumonia or a hemorrhage from an ulcerated bronchial vessel.

Symptoms of Pulmonary Tuberculosis.—The onset of this, the most common type of the disease, is often such as to mislead the physician. In some cases there is no cough, but only a slight *rise of evening temperature* preceded by chilly sensations. If the number of cases in which these symptoms have given rise to the diagnosis of "malarial poisoning" could be gathered together they would be a " multitude which none can number." In many instances this error has been a deserved reproach to the physician who made it, because he has not searched for tuberculosis as a cause but has simply prescribed quinine.

Another type of onset is found in those cases which present insidious pleural effusion. (See Pleurisy with Effusion.) In still a third series the primary symptoms are *laryngeal.* As these lines are written I am sending a case of active tuberculosis of the lungs to New Mexico. He was told by several skilled laryngologists that his husky voice was due to gout of the larynx, whereas if they had examined his chest marked signs of phthisis would have made the diagnosis evident. All cases presenting signs of persistent hoarseness should cause the physician to search for tuberculosis, syphilis, papilloma of the larynx, and aneurysm of the aorta.

In the fourth type the very earliest sign of the disease is *spitting of blood.* There can be no doubt that in the vast majority of instances the bringing up of blood from the bronchial tubes means tuberculous infection. The only other causes which are at all frequent in the production of hæmoptysis are acute pneumonia or pulmonary infarction due to cardiac lesions. It not infrequently occurs that hæmoptysis in the stage of onset is scanty and associated with no demonstrable physical signs, the lesion being situated in such a position that it readily perforates a vessel. (See p. 316.)

The symptoms of pulmonary tuberculosis may be divided for study into those which are complained of by the patient, those which can be readily observed by the physician, and those which can be demonstrated by the aid of physical diagnosis. It must be remembered, however, that the severity of the symptoms of all kinds varies to an extraordinary degree in different cases and at different times in the same case. It is necessary, therefore, in speaking of the symptoms to adhere to the description of the three types of the disease named when discussing its pathology. At the outset, however, it may be said that two symptoms are present in all cases at some period, namely, *loss of flesh* and *fever.*

It may be said of the *fever of tuberculosis* that it is usually moderate, varying from 100° to 102°, although at times it may reach 103°. When the temperature reaches higher than this it is probably not due solely to the tuberculous infection, but to septic or hectic fever, depending upon associated staphylococcic, pneumococcic, or streptococcic infection. In all instances in which the fever is high it is prone to run a very uncertain and aberrant course, save that it is high at night and low in the morning, as in nearly all fevers, particularly that due to sepsis. It is very easily broken, as a rule, by the use of any antipyretic medicine, but this effect of drugs is, of course, very temporary.

The *loss of weight* depends upon several causes for its existence. The loss of appetite, the cough, which is exhausting and sleep destroying, the sweats, the disorders of digestion, and the anæmia are all active factors in decreasing flesh. Last, but by no means least, as a cause of loss of weight, is to be considered the *toxæmia* of the disease itself.

The rapidity of loss of flesh is sometimes remarkable, amounting to as much as four or five pounds a week. This rapidity of loss is a good guide to the activity of the tuberculous process, for if it be rapid the outlook as to the progress of the patient's illness is gloomy. On the other hand, gain in weight is correspondingly encouraging in that it indicates a very slow, or arrested, progress of the disease.

A third symptom, often of very great annoyance to the patient, is *sweating,* which is particularly prone to occur at night. These sweats vary greatly in severity, and seem to occur because of the relaxation of sleep, but in many cases their true cause is the hectic or septic state of the patient. If they are not severe enough to exhaust the patient or disturb his rest, they are to be regarded as an effort to diminish toxæmia, but if they become so profuse as to be called colliquative they are deleterious. At times the sweat follows a sharp rise of septic temperature. It is hardly necessary to add that profuse night sweats, while a common symptom in well-developed phthisis, are by no means pathognomonic of this disease.

Aside from the loss of flesh, fever, and sweats, the most constant symptom of pulmonary tuberculosis is *cough.* It varies in its character and in its degree in different cases of the subacute or chronic form of the disease. In the early stages it is apt to be worse on going to bed or on getting up in the morning, and in the early stages is usually annoyingly unproductive and persistent. As the disease advances and the process of softening begins to take place in the consolidated part of the lung, the cough becomes less dry and more productive. When cavities are formed, marked increase in morning cough is very prone to occur in order that the cavity may be well cleared of the accumulations which have occurred in it during the night. Cough is to be regarded as a useful attempt on the part of the system to keep the lungs clear. Only when it provokes hemorrhage or is so excessive as to cause exhaustion, loss of sleep, or vomiting is it to be regarded as an evil.

Aside from the general symptoms of tuberculosis already described, patients with pulmonary tuberculosis often have *severe pain* in the chest, which is due to an extension of the inflammation to the visceral layer of the pleura. They also suffer from *dyspnœa* on exertion partly because of the diminished area of lung and lessened ability of the blood to carry oxygen, partly from cardiac feebleness, and partly because the general nervous system and the muscles are so feeble that any exercise leads to exhaustion.

The *sputum* is composed of mucopurulent material from the associated chronic bronchitis, or if the lung is beginning to undergo softening the expectorated material is thin, with small, pale and greenish-looking masses in it—the so-called "nummular sputum." Sometimes when a cavity is being emptied or there is a marked bronchorrhœa the sputum is very purulent.

The quantity of sputum varies greatly. The average amount in an active case varies from 1 to 4 ounces a day, but I have known a patient with several cavities to raise a pint or more in twenty-four hours.

Complications.—A frequent symptom of the ulcerative type of pulmonary tuberculosis is *hæmoptysis,* but a large number of cases pass through all stages of the disease without bringing up a particle of blood. It is absent, according to West, in from 20 to 30 per cent. of cases. Hæmoptysis is more than three times as frequent in males as in females. The quantity of blood lost varies from a mere streak in the sputum to 3 ounces in the average case. Occasionally it amounts to 4 or 6 ounces, but a little blood "goes a great way," and patients will state that they have spit a quart when only a few ounces have been raised. It is rare for as large an amount as a pint to be coughed up in twenty-four hours. Very rarely a large gush causes death by suffocation. Sometimes the hemorrhage is concealed and unaccompanied by blood-tinged sputum. If of a dribbling type it may inundate a large part of the lung or even fill a cavity and cause death without any external manifestation; such cases are rare. A free hemorrhage nearly always means the presence of an ulcerating cavity. The blood in hæmoptysis may come from the pulmonary vessels or from the bronchial vessels, but it is usually from the former. Flick, Ravenel and Irwin believe that hæmoptysis is usually due to pneumococcus infection.

All ages may suffer from hemorrhage from the lungs, but the period from eighteen to thirty-five is of course that of greatest frequency. Hoffnung has recorded a case in a child of ten months and Powell one in a child at seven months of age.

The blood which is expelled in true hæmoptysis is usually frothy and is brought up by coughing. It is also usually red except in instances of slow oozing into a cavity, when it may appear as a dark clot or clots. When the bleeding is profuse the blood gushes out of the mouth. Often before the spitting of blood actually takes place a salty, or bloody, taste in the mouth is persistently present for some time.

Hæmoptysis due to pulmonary tuberculosis is to be separated from hæmatemesis by the fact that the first occurs with coughing and the second with retching or vomiting. It is further differentiated by the fact that the blood is frothy and filled with mucus and bubbles and is usually bright red in hæmoptysis, whereas in hæmatemesis it is usually pure or discolored by contact with the gastric juice. In one state the history of pulmonary disease, or the discovery of lesions in the lungs, reveals the seat of the hemorrhage; in the other gastric symptoms are present. In hæmoptysis the blood is often brought up in small degree for several days, whereas in hæmatemesis it is usually brought up once or twice on one day and then the bleeding ceases.

In this connection it must not be forgotten that hæmoptysis, or blood spitting, is not always due to tuberculosis of the lungs. It is sometimes present in the stage of onset in acute croupous pneumonia. It is not rarely met with in thoracic aneurysm, and its occurrence, unless it be very profuse, does not necessarily mean immediate death in the latter type of cases, since it not rarely happens that the vessel oozes blood for several days before it finally completely gives way. Sometimes by the pressure of the aneurysmal sac some small vessel may be eroded so that the blood escapes from it alone. Slight hæmoptysis occurs in some cases of malignant intrathoracic growth, and swollen glands by pressure may rupture a neighboring vessel and cause leakage of blood. Again, hæmoptysis often develops in mild degree as a result of pulmonary infarction.

Among the other causes of hæmoptysis may be mentioned hemorrhage from a superficial vessel in a bronchial tube in bronchiectasis, and from the larynx in malignant and non-malignant growth or tuberculosis of this organ. Hæmoptysis is sometimes due to a varicose condition of the veins at the root of the tongue.

A peculiar form of hæmoptysis which lasts in some cases for years is seen in Formosa and Japan, due to the presence of the parasite *Paragonimus Westermanni.* (See Parasitic Hæmoptysis.)

Although hæmoptysis in the great majority of cases indicates pulmonary tuberculosis, it must not be forgotten that this symptom sometimes occurs for years without any other signs of the disease appearing. I have one case in mind in which the young wife of a student at the Jefferson College had repeated hemorrhages during an entire winter without any physical signs being present, and continued to have them for many years afterward. Eight years after they began she still had them on exertion, but was the picture of health, had no signs of aneurysm or tuberculosis, and had gained thirty-five pounds.

In regard to the *distribution of the cavities* Ewart has collected the following interesting statistics. In 791 cases cavity occurred at the apex in 282 cases, in the dorsoaxillary region in 227, in the mammary region in 189, in the sternal region in 61, and at the base in 32.

Many of the other complications of pulmonary tuberculosis in addition to vomiting, diarrhœa, and hæmoptysis have already been considered when discussing the disease as it affects serous membranes, as in pleurisy and pericarditis. The most important is *pneumothorax,* which follows the perforation of the tuberculous lesion into the pleura. It occurs in from 3 to 10 per cent. of all cases, is often fatal if sudden in onset, and may cause distressing dyspnœa. West says that of 39 cases, 2 died in an hour, 8 others in twenty-four hours, and 29 out of the 39 inside of two weeks. The mortality is about 90 per cent. Effusion usually speedily develops. I have seen great relief follow gentle aspiration of the air from the thorax, but aspiration is to be avoided save when the pressure produces urgent dyspnœa. Very rarely the pneumothorax develops insidiously without severe symptoms, producing what is called "latent pneumothorax." (See Pneumothorax.)

A still more rare affection seen in some cases of the chronic forms of pulmonary tuberculosis is pulmonary osteoarthropathy (which see).

Diagnosis.—Before the physical signs of pulmonary tuberculosis are dealt with the sites of the lesions usually present may be discussed, so that they may be examined with particular interest in every case. The apices are the parts affected in the vast majority of cases, and it is here that the primary lesion is usually found, even if other parts become more severely diseased later on. The process as it extends is prone to travel backward rather than forward. No satisfactory explanation of this fact is obtainable.

Next to the apices the upper part of the middle lobe on the right side is most frequently the site of infection, or the upper part of the lower lobe on the left side is diseased. The area of the upper part of the middle lobe on the right side is one which is often overlooked, owing to the fact that it is covered by the right scapula. Only when this scapula is raised by the hand being placed on the top of the head is the spot of impaired resonance exposed at its lower margin.

Tuberculosis of the bases rarely occurs except after the disease has lasted long enough to involve the whole lung.

PHYSICAL SIGNS.—The two methods of physical diagnosis which give us the most information in cases of pulmonary tuberculosis are percussion and auscultation. Throughout that period in which there is consolidation of the lung *percussion* gives impaired resonance or dulness over all the part affected, unless the lesion be deep seated, in which case light percussion over this part may produce a sound which is high pitched or slightly tympanitic. With the development of cavity the percussion note over it undergoes a change and there is developed a high-pitched tympanitic resonance, which careful percussion will show to be surrounded by an area, or ring, of impaired resonance representing the surrounding area of infiltrated lung-tissue. At times in the neighborhood of tuberculous lesions in the lungs hyperresonance is developed on percussion, as the result of a compensatory emphysema of the lung. If the cavity communicate with a bronchus and the patient takes a breath and holds it, with the mouth open, percussion may develop the so-called "cracked-pot sound."

Auscultation reveals, in the earliest stages of infiltration, prolongation of expiration in the part involved. This is a physical sign of very great importance. Again, it may reveal some harshness of the inspiratory murmur and both inspiration and expiration may be more distinct and rougher than in health.

Occasionally careful auscultation will also reveal a few very fine rales on forced inspiration. In lesions of the apex on the left side such a forced inspiration not rarely produces an inspiratory sound which is interrupted by the action of the heart three or four times during the act of drawing air into the lung.

It must not be forgotten that negative signs may be as valuable as positive ones, and therefore if the infiltration produces an absence of breath sounds at the infected spot this may indicate disease as surely as do the more positive signs already named.

If the physician listens carefully over the area of consolidation with his disengaged ear closed by his finger-tip, and the patient will say one, two, three in a stage whisper, the area of consolidation will give greater vocal resonance than the same area in the healthy lung.

With the development of softening the fine dry rales which have been heard at first become coarse and moist, and as a cavity is formed they may become even bubbling or gurgling. These rales sometimes possess a curious metallic sound.

As the cavity is formed the vocal resonance over it increases and may become startlingly clear, so that when the patient speaks the sound of the voice is transmitted with great clearness through the chest wall. This is called pectoriloquy.

Over such a cavity cavernous breathing is often heard, or, if the cavity is a small one, the breathing may be hollow, tubular, or amphoric, as if he patient were blowing with his lips over the mouth of an open bottle.

Moist cavities also present on auscultation, in addition to large moist rales, metallic tinkling due to the dropping of fluid from their walls. This metallic tinkling is to be separated from the metallic tinkling of hydropneumothorax by the absence of the physical signs of fluid in a dependent part of the chest, and by the fact that such a cavity is near the upper part of the lung and so produces this sign in the upper zone of the chest.

The sounds arising from a dry cavity are blowing or amphoric. When such a cavity has existed long enough for marked fibroid change to occur it often happens that the chest over the affected part is greatly flattened, and it may be decreased in all its diameters. Compensatory hypertrophy of the opposite lung causes an increase in the size of the chest on that side, and this emphasizes the difference between the two sides. Further than this, the contraction process may greatly displace nearby organs. Thus, if the left lung undergoes this change, the right lung, partly from enlargement and partly from traction, may extend as far as three inches to the left of the sternum, the heart may be drawn upward and tilted to the left of the nipple as high as the third interspace. Even the stomach may be drawn upward. On the other hand, when the right lung is affected by the disease the heart may be drawn to the right under the sternum, the liver may be pulled upward, and the left lung drawn well over to the right side of the chest. Most of these marked changes are due to associated pleural adhesions, and these may cause deformity of the entire chest.

The diagnosis of cavity from bronchiectasis is made by the recollection that a cavity is usually near the apex, and bronchiectatic spaces are at the base, as a rule, although they may develop as high as the third or fourth rib. If so, they are nearer the sternum than is the cavity. Again, in cases of cavity the area around the hollow space is usually dull on percussion, whereas in bronchiectasis it is usually hyperresonant from emphysema.

It is to be remembered that tuberculosis may occur as a complication of bronchiectasis, but that well-developed bronchiectasis rarely occurs in tuberculosis except in old chronic cases with much contraction due to fibroid change.

Palpation.—Palpation over that portion of the chest which is infiltrated by a tuberculous process, or in which a cavity has already formed, also presents very definite physical signs when the patient speaks, namely, a marked increase in vocal fremitus. If the cavity is superficial and of any size it may be possible to feel the bubbling rales which are produced by its contents.

Inspection.—Inspection of a well-advanced case of pulmonary tuberculosis occurring in one whose configuration is naturally phthisical reveals a very typical picture, but in those with well-developed chests very advanced lesions of the lungs may be present before any change in the appearance of the chest is manifest.

The *physical signs of acute pneumonic tuberculosis* are at first the same as those of acute pneumonia. There are bronchial or tubular breathing, dulness on percussion, and fine crepitant rales. As the disease progresses these signs become modified to the extent that the rales become coarse and more moist in character, and signs of softening are therefore developed.

In the *diagnosis* of a case of this character it may not be possible to state accurately the true cause of the disease for several days, but the following points are of some value, namely, the discovery in the history of the patient, or in the body at the time, of a tuberculous infection, as of enlarged cervical glands or of tuberculous masses elsewhere, as in a Fallopian tube or in a testicle or joint; the general appearance of the patient as to nutrition, for the pale, anæmic patient with the typical slim bones and large joints, large orbital spaces, and delicate features is more apt to succumb to the bacillus tuberculosis than to the pneumococcus. On the other hand, it is to be remembered that robust and hearty persons may develop acute tuberculous pneumonia and die in a short period. The presence of the pneumococcus in the sputum is of little diagnostic value, but the discovery of the tubercle bacillus will be of great aid in determining the cause of the illness.

MICROSCOPIC DIAGNOSIS.—An examination of the sputum by the aid of the microscope reveals shreds of mucus mixed with particles of caseous substance and small round cells, leukocytes, and pus corpuscles. Crystals of the triple phosphates, oxalates, and of tyrosin and leucin are often present. All these constituents of the sputum are, however, of little importance as compared to two others, namely, the presence of *elastic-tissue fibres* possessing the morphology and arrangement of pulmonary reticulum, showing that breaking down of the lung is taking place, and *tubercle bacilli*, the presence of which reveals the fact that they are the cause of this condition. The bacilli are indisputable evidence of the presence of the disease, but their absence from a specimen of sputum does not exclude tuberculosis, because they may happen to be absent from that individual sample, or they are absent because the sputum does not come from a part of the lung in which breaking down is taking place. Even an old cavity, if it is well drained, may not provide bacilli constantly.

Yellow, elastic fibres are to be sought for by spreading the sputum in a thin layer on a pane of glass placed over a blackened surface. A second sheet of glass is placed over this and the sputum smeared by moving the upper piece laterally. The particles of elastic tissue are usually contained in small masses of yellowish-gray material, which, if crushed and placed under the microscope, are found to consist of characteristic, double-contoured, interlacing, yellow, elastic fibres, having the arrangement of the pulmonary elastica. Elastic-tissue stains are of value in the hands of experienced microscopists. As many meats are rich in elastic tissue which may lodge in the mouth, the mere finding of such structures in the sputum does not justify the diagnosis of " breaking down " of the lung. The recognition of pulmonary elastica must be based on the shape and arrangement indicated. Occasionally small pieces of calcareous matter are found in the sputum.

The bacilli are sought for in the following manner: A microscope slide is thoroughly cleansed and dried. From the sputum spread out on a glass plate, or in a Petri dish, nummular particles if present are selected, or if absent the thicker portion of the sputum is spread in a thin layer over the surface of the slide and allowed to dry spontaneously. The dried film on the slide, surface upward, is passed three times through the flame in order to fix the thin layer firmly to the slide. Cover-glasses may be used instead of slides, but possess no special advantages. Of the many stains recommended Ziehl's carbol-fuchsin gives satisfactory results. It is prepared by dissolving 1 gm. of powdered fuchsin in 10 c.c. of alcohol; to this solution 90 c.c. of 5 per cent. aqueous solution of carbolic acid is added; the stain is ready for immediate use, and if prepared from proper ingredients keeps well. The prepared stain is poured over the slide, which is then heated over a Bunsen burner or alcohol lamp until steam begins to rise, when the heat is withdrawn. After staining five minutes the excess is poured off and the slide freely washed in clean water. It is then flooded with Gabbett's solution, which consists of 1.5 gm. of methylene blue dissolved in 100 c.c. of a 25 per cent. aqueous solution of sulphuric acid. This is allowed to act for one minute; it is then poured off and the slide washed in water; if any of the red dye be retained the application of the Gabbett solution is repeated until all macroscopic evidence of the fuchsin has disappeared from the thoroughly washed slide, which is then stood on end and allowed to dry.

A drop of immersion oil is placed on the stained film and the specimen examined with a one-twelfth-inch immersion lens. If it be desired to preserve the specimen, balsam is applied to the dried slide and a cover-glass placed on it. In properly prepared films the cellular elements and bacteria, other than the tubercle bacillus, will have selected the blue dye; the tubercle bacillus, however, will appear red on the bluish background.

X-Ray.—A valuable aid in determining the presence of consolidation of the lung in tuberculosis is the use of the *fluoroscope* or *x-ray photograph*, for such a lesion often reveals a very distinct opacity.

Tuberculin.—In the diagnosis of pulmonary tuberculosis there can be no doubt that tuberculin when properly employed is a valuable agent, although I believe that in the majority of instances it should not be used, since careful examination of the patient and consideration of his history will in most instances reveal the presence of tuberculosis, or point to its presence with such a degree of certainty that the patient should certainly be sent away for his health on the ground that he is a fair mark for a tuberculous infection, and that his lung is in such a condition that he is at all times liable to the rapid development of a true tuberculous process.

Tuberculin, which is used hypodermically, for these diagnostic purposes is usually given to adults in the dose of a milligram, and if tuberculosis is present it causes a reaction in the form of a rise in temperature of two or three degrees within a few hours. If the dose is larger than this, susceptible persons may have a violent reaction. If the patient fails to react to the smaller doses, before deciding that the tuberculin test has proved him free from tuberculosis, doses of 2 mg. or 3 mg. or even more should be given at intervals of three or four days, or a week.

The use of tuberculin for diagnostic purposes has a larger field in cases of suspected renal or abdominal tuberculosis than it has in the diagnosis of pulmonary lesions, in which the physical signs can usually be demonstrated.

It is to be remembered that the tuberculin reaction sometimes occurs in persons who have syphilis, and it is thought by some that the reaction which is produced may actually increase the rapidity of the tuberculous process.

In order that a careful record of its effects may be obtained the temperature of the patient should be taken after the injection at intervals of every two hours for six hours, and after that every hour for twelve hours. Before the test is made it must be determined that the patient is afebrile by a careful record of his temperature for several days, as otherwise the usual fever may be mistaken for a reaction.

Tuberculin may be used in hypodermic tablets, each of which contains 1 mg., or in the form of the tuberculin (Koch), which is a liquid preparation. If the tablet is used it is dissolved in water as is an ordinary hypodermic tablet. If the liquid tuberculin is employed the bottle containing it is uncorked and a small pipette, which is graduated, is dipped into the contents and then withdrawn, with the finger-tip over the outside end to hold the fluid in the pipette, as in testing urine. The quantity of the fluid as expressed in milligrams is then allowed to run out into a sterile porcelain dish, and pure water is drawn up in the pipette to wash it free of any residue of the original fluid. This wash-water is then added to the contents of the dish, and, after mixing, the fluid is drawn up into a hypodermic syringe and given to the patient through an ordinary hypodermic needle.

It has been urged against Tuberculin R. that there is a possibility of its containing living bacilli which may infect the patient. For this reason many clinicians employ a tuberculin prepared by filtration and subsequent concentration of sterilized bouillon cultures, whereby a germ-free product is assured. (See Treatment.)

Prognosis.—It is not long since it was almost universally thought that subacute pulmonary tuberculosis was an utterly hopeless and incurable disease. At present we know that it is in many instances a readily curable affection, even when it is not possible to obtain the very best conditions for cure. Further, we know that hundreds of persons have the disease and get well without even knowing that they have had it. It is manifest, however, that only those cases can recover in which the disease is not far advanced and in which the vital resistance of the individual can be maintained at such a level that the protective processes of combat and repair, already described may be carried out to completion.

The degree of vital resistance of the patient is of very great importance in deciding the prognosis. Often the most powerfully built individual falls a victim to rapid phthisis while his comparatively feeble comrade manifests the most remarkable vitality. Additional factors in determining the outlook in an individual case are the maintenance of the body weight, the absence of anæmia (but red cheeks do not necessarily mean good blood), and the presence of a good digestion, particularly in respect to starches and fats. A good family history is not as important a factor for good as a bad history is important for evil.

A large number of statistics as to the curability of this disease by climate and feeding and by out-door life are now obtainable, and some statistics will be found discussed under Treatment.

F. C. Wood believes that the diazo reaction can be used to aid in determining the question of prognosis, stating that if no reaction occurs and the kidneys are intact the outlook is favorable, but that if the urine reveals a strong and persistent reaction the outlook is evil. (For the diazo test, see article on Typhoid Fever.)

The average duration of life in a case of pulmonary phthisis is limited to two years.

Marriage should be forbidden for either sex if suffering from tuberculosis, even if it be in a mild form. The woman who is tuberculous may survive her first pregnancy only to pass into a hurried decline after the birth of her child or during lactation. Several pregnancies almost always destroy her. The man not infrequently loses nervous vigor by marriage, and this is the more prone to occur, as it is notorious that tuberculous men are cursed with a degree of sexual desire which is in excess of that of health.

The prognosis of hæmoptysis so far as its causing immediate death is concerned is favorable. Patients rarely die during the hemorrhage unless it takes place in those who are already very feeble and anæmic. Rarely the hemorrhage is so profuse as to cause death by suffocation. West gives the proportion of deaths from this cause as but 1 or 2 out of every 100 cases that die of this disease, whereas 60 per cent. of tuberculous cases are supposed to suffer from hæmoptysis at some period of the malady.

The secondary effects of hemorrhage may, however, be disastrous, for if the neighboring part of the lung is inundated with blood and with bacilli, the bronchioles in that part become filled with the extravasation and a traumatic tuberculous pneumonia speedily ensues.

Treatment of Pulmonary Tuberculosis.—The treatment of pulmonary tuberculosis in its subacute or chronic forms may be considered in several parts.

1. Its treatment by proper diet, proper exercise, and rest.
2. Its management by suitable out-door life, and particularly by climate.
3. The employment of drugs to control or modify symptoms which are severe enough to demand attention.
4. The use of tuberculin as a curative remedy.
5. The avoidance of the use of drugs with the idea that they can cure the disease; for he who tries to cure pulmonary tuberculosis by drugs does not know the morbid anatomy of the malady.

It is of vital importance in the treatment of pulmonary tuberculosis that the disease be recognized at the earliest possible moment and that curative measures be immediately instituted. The possibility of cure depends solely upon the limitation of the lesion, and this is difficult to accomplish in direct proportion to its size and the degree to which degenerative changes have advanced.

DIET.—There can be no doubt that the proper nourishment of the patient is the most important matter demanding the attention of the physician; for tuberculosis is not only a disease in which emaciation progresses rapidly, but it is one in which the outlook depends entirely upon the ability of the patient to carry out protective processes through which alone he can hope to recover his health. Under these circumstances it is evident that the physician must do everything in his power to keep the digestion in the best possible order, to administer foods which are easily digested and readily absorbed, and, equally important, to prescribe no drugs or foods which by disordering the stomach will interfere with the function of this important viscus. It must also be remembered that the digestion of food requires nervous energy just as does the performance of any other vital function, and care must be taken that food is not ingested at a time when, by reason of exercise or other cause, a considerable quantity of nervous energy has been recently expended. The physician is, therefore, in the difficult position of knowing that the patient must take large quantities of nutriment if recovery is to be expected, and at the same time be careful that the digestion is not overburdened by the too frequent administration, or too free employment, of nutritious articles. If the patient's digestion is moderately strong, he may follow a line of diet about as follows:

Before getting up in the morning he should receive a teacupful of hot milk, which should be sipped and not gulped down in one or two large swallows. After taking this, he should rest in bed for fifteen or twenty minutes; should then bathe, or be bathed, and clothed, and for his breakfast have wheaten-grits, oatmeal, or some of the more modern cereal preparations which are known to possess real nutritive value. If his appetite is good he may also have at this time a tender chop or a small piece of steak, and if accustomed to the use of tea or coffee, these beverages may be allowed unless it is found that they increase nervous irritability. In some instances the patient may desire to take an orange or some other fruit with his breakfast, and to this there can be no objection. The meal should be adequate, but not large enough to be heavy.

Half-way between his breakfast and his mid-day meal the patient should receive some light luncheon, consisting of a cup of broth, a piece of toast, a glass of koumyss, or a sandwich made of scraped beef; or, if he tires of this, one made with toast and anchovy or caviar. Often an egg, cooked or raw, may be taken between meals with advantage. If desired, a glass of sherry or some red wine may also be taken at this time; or, in its place, Scotch or rye whiskey may be given.

The dinner should be the heaviest meal of the twenty-four hours, and should be taken between twelve and two o'clock. It should consist of a nutritious and somewhat stimulating soup which is easily digested and absorbed; one of the clear soups being preferred rather than a purée, unless it is known that the patient readily digests thickened and rich soups. This may be followed by a small piece of fresh fish, great care being taken that the fish is really fresh, and then by a hearty course of any one of the roast or broiled meats, accompanied by two or three wholesome vegetables, such as potatoes, string beans, asparagus, spinach, carrots, macaroni, and similar substances. With this meal it may be well for the patient to take a little sherry wine or whiskey and water, particularly if he is accustomed to stimulants with his meals. Some plain, nutritious dessert like cornstarch or rice-pudding may also be taken.

During the afternoon a light luncheon should be given him, somewhat similar to that which has been taken in the middle of the forenoon, two or three hours after his dinner. In the evening another light meal should be taken, which should consist of arrow-root or an egg cooked in some simple form, or a few stewed oysters or milk-toast may be used, and again before going to bed at night a cup of broth, a glass of koumyss, a cup of hot milk, or some curds and whey may be given.

The patient who is able to take the quantity of food which has just been described is, of course, one whose digestion is in fairly good condition. But if careful attention is paid to the digestive tract by the administration of aids to digestion, such as pepsin, pancreatin, and taka-diastase, if the bowels are moved regularly by the use of proper laxatives, and if, above all, the patient is required to conserve his nervous energy in order to expend it upon his digestive apparatus, it is quite remarkable what large quantities of food may be taken, even by the consumptive who otherwise seems quite feeble.

The actual quantity of the food at each feeding must be varied from day to day with the patient's appetite and with the conditions which may arise. If the patient has passed a restless and feverish night, the quantity of food at each feeding should be small; whereas, if he has had a restful night, and therefore has been able to gain nervous energy, larger quantities may be given. So, too, limited quantities should be ordered when the tongue is at all foul, and larger quantities ordered when it is comparatively clean. It is of vital importance that these daily variations should be made in the diet, for the digestive apparatus of no one is prepared day in and day out to take exactly the same quantity of food, and digest it satisfactorily. Both the physician and the patient must remember that professional advice as to food and digestion is much more important for the patient than advice as to the treatment by drugs.

EXERCISE.—The majority of cases of pulmonary tuberculosis do not require much exercise provided they are supplied with sufficient fresh air. Patients, however, differ very greatly in regard to this matter. Some of them seem capable of taking moderate exercise with great advantage, and others cannot take any exercise without suffering either from a disordered digestion or from a restless night caused by inordinate fatigue. In many instances the patient's health can best be preserved by giving him fresh air, and supplanting exercise by massage and gentle Swedish movements. Of course, these remarks do not hold true of those cases in which a small area of the lung is involved, with almost no impairment of the general health and muscular strength. These patients should take healthy exercise, being careful to avoid excessive fatigue, and they should be impressed with the idea that exercise in sufficient degree to approach exhaustion is not only bad on general principles, but actually diminishes their ability to prevent the spread of the infection in their lungs. The whole question of exercise must, therefore, be gauged in each case by the real strength of the individual rather than by his ambition to be up and about.

CLIMATE AND OUT-DOOR LIFE.—In these two factors we have our greatest aid in the treatment of pulmonary tuberculosis, although, of course, these agents must be prescribed with the same care as governs our employment of ordinary remedies. There can be no doubt whatever that an out-door existence is capable of curing pulmonary tuberculosis under certain circumstances, even when the climate is by no means theoretically suitable for pulmonary cases. This is a matter of importance when it is remembered that a very large proportion of consumptives find it impossible to travel great distances to obtain those climatic conditions which are most favorable to them.

At every modern resort for consumptives every measure is taken to keep the patients for many hours each day in the open air, the essentials being that they shall be exposed to sunlight, and, if possible, to the direct rays of the sun, and protected from high winds. These conditions can be obtained by the erection of suitable sheds facing the sun, and providing wind guards which will place the patient in a quiet atmosphere. Even should the patient be unable to go to the country for fresh air, good results have been found to follow this plan of treatment while he remains in a city residence, either in a suitably arranged room or in a tent or shed erected upon the roof of his house.

The climate to which the patient should resort, if it is possible for him to travel, should, in the great majority of instances, be one which is found at an altitude varying from 3000 to 6000 feet. There are two great essentials in such a climate: first, that there shall be an unusual number of hours of sunshine in the course of the year, and, second, that the atmosphere shall be dry. A third point of importance, but by no means an essential one, is that the atmosphere shall be quiet, in order that there may be little dust. The temperature is of comparatively little importance, provided it is not accompanied by humidity, for it is quite remarkable how patients suffering from this disease often thrive in temperatures which in winter are far below the freezing point, and in summer are often as high as 90°. At those altitudes of from 5000 to 6000 feet which are suited to this class of patients the atmosphere is so clear that the sun's rays are not interfered with, and even if the thermometer shows that a zero temperature is present, the patient, if properly clad and protected from wind, can very frequently lie out-of-doors all day, warmed by the heat of the sun. This is beneficial to an extraordinary degree.

A high altitude is advantageous for the tuberculous patient, not only for the reasons which we have given, but also because the rarity of the atmosphere requires that he use all possible portions of the lung tissue, and, this being the case, he gradually expands and calls into functional activity all those parts of the lung in the neighborhood of the tuberculous lesion which have a tendency to become functionally inactive. This development of active circulation of air and blood does much toward aiding nature in walling off the tuberculous focus and preventing its further spread. A high altitude is also advantageous because it seems to increase the quantity of hæmoglobin in the blood. Whether it increases the number of red blood cells is still a matter of debate, some asserting that the increased number of corpuscles, found in the superficial bloodvessels after a patient has been some weeks at a high altitude, depends more upon an altered distribution of blood than upon any actual increase.

Of the high-altitude resorts which are most popular because of their excellent climate for consumptives may be mentioned Colorado Springs, Colorado, certain parts of Arizona and New Mexico, and parts adjacent in America, and the so-called Engadine, in Switzerland, of which the most celebrated places are Davos, Pontresina, and San Moritz.

A high, dry climate is contraindicated in tuberculous patients who are suffering from tuberculous laryngitis, since the dry air increases the laryngeal irritation. It is also contraindicated in patients who have dilatation or degeneration of the heart muscle, and great care should be taken to determine the state of the heart in all tuberculous patients before sending them away from home. A persistently high pulse rate is a distinct contraindication to altitudes. If valvular disease exists, and there is a tendency to failure of compensation, a high altitude is also contraindicated; but where compensation is adequate, the mere presence of a murmur does not necessarily contraindicate resort to an altitude, provided that the patient is cautious not to overexert himself.

Emphysema associated with pulmonary tuberculosis usually contraindicates a high altitude.

It has been taught by some that a tendency to hæmoptysis also renders a high altitude inadvisable, but very eminent phthisiologists, on the contrary, have asserted that a tendency to hemorrhage does not contraindicate health resorts of this character. My own personal feeling is that the patient who has a tendency to hæmoptysis should approach a high altitude very gradually in order that his heart and lungs may become accustomed to the altered atmospheric conditions, and certainly, for the first few weeks after his arrival, should rest constantly.

Still another contraindication to such an altitude, unless perchance the climate is very mild and the temperature fairly constant, is renal disease of any kind, or tuberculosis of the genito-urinary tract. Should any of these contraindications exist, the climate of choice is one which is represented by San Diego, California, where the air is pure, where sudden changes of temperature do not occur, and where a flood of sunshine is constantly obtainable.

In those cases which have a great deal of secretion, excessive cough, and excessive expectoration, dry climates, such as are found near Phœnix, Arizona, and Silver City, New Mexico, are the climates of election.

For those who are unable to take a long journey, and for those who are not thought to be suitable cases for high altitudes, the Adirondacks in the neighborhood of Saranac Lake, New York; White Haven, Pennsylvania; or Asheville, North Carolina, can be recommended. These altitudes are in the neighborhood of 2500 feet. Lower altitudes which have been found advantageous for these cases exist at Thomasville, Georgia, and Lakewood, New Jersey, where the curative elements are the sunshine and pure air.

It is interesting to note, in regard to the treatment of pulmonary tuberculosis by fresh air, proper diet, and moderate or high altitudes, that a large percentage of cases can be cured. Thus, Trudeau reported, at the Adirondack Cottage Sanitarium, where the altitude is less than 2500 feet, for the years 1897, 1898, and 1899, cures in 72 per cent. of incipient cases and 17.8 per cent. in advanced cases; and Clapp and Bowditch, of the sanatorium at Rutland, report for the year ending September 1, 1901, cures in 72.5 per cent. of incipient cases and 46.11 per cent. in advanced cases.

Still more recently Trudeau has presented the results obtained by an analysis and study of all the cases under his observation in the last seventeen years.

Of the 1500 cases under consideration, which have been discharged from two to seventeen years, 434 could not be traced, leaving 1066 which have been traced. Of these 1066, 46.7 are still living, 31 per cent. are known to be well at present, in 6.5 per cent. the disease is still arrested, 4 per cent. have relapsed, 5.2 per cent. are chronic invalids, and 53.3 per cent. are dead. As to the influence of the stage of the disease on the permanency of the results obtained, he found 66 per cent. of the 258 incipient cases discharged are well at present. Of the 563 advanced cases 28.6 per cent. are well, and of the far-advanced cases 2.5 per cent. only remain cured. Thus we learn that 31 per cent. of all cases discharged from two to seventeen years ago have remained well, and that 66 per cent. of the incipient cases discharged during the same time continue well at present. Surely these results are encouraging and he has shown us the way in a great work. Thirty years ago physicians were of the opinion that cures did not take place from pulmonary tuberculosis in more than 2 per cent. of cases.

Sea voyages, which at one time were very popular in the treatment of tuberculosis of the lungs, are no longer regarded with much favor. The possibilities of seasickness, bad weather, and of consequent close confinement are naturally not looked upon with favor, when we consider that free feeding and fresh air are absolutely essential for these patients. Further than this, the atmosphere at sea is never dry, but always more or less damp. Again, there are practically no comfortable sailing ships at the present time, and steamers make such rapid voyages that the patient is not long enough at sea to be materially benefited.

Before the physician decides to send his patient away from home for the climatic treatment of his disease, he should determine whether such treatment really offers fair chance of benefit; for it is a vital mistake to exhaust the strength and finances of the individual in a vain endeavor to arrest an inevitable process. If it is decided that the disease has advanced so little that such a trip can promise good, the next question which arises is as to whether the patient is strong enough to stand the journey, and, again, if he can stay away long enough to be benefited; for in all instances it is useless for a patient to leave home with any expectation of returning in less than six months or a year, and usually he had better give up the hope of returning except for a visit, if he wishes to preserve his health after the climate has done its good work. It is also a grave mistake to send such patients far from home unless they can be accompanied by some relative or friend, as homesickness exercises a deleterious influence upon vitality.

DRUGS.—I have already mentioned the use of the various digestants when speaking of the diet. The ever-present anæmia of many of these patients is to be overcome by the careful administration of iron and arsenic. Arsenic for many years has had the reputation of being a drug of great value in tuberculosis. Iron is also very useful, but it should not be given in the large doses ordinarily employed. As I have repeatedly pointed out, the quantity of iron in the body is exceedingly small, not more than about 10 grains, and therefore the administration of 2 or 3 grains of reduced iron two or three times a day provides in twenty-four hours far more iron than can possibly be utilized, and at the same time tends to produce constipation and so disturb digestion. Most cases will do better if they are given in the neighborhood of $\frac{1}{10}$ to $\frac{1}{2}$ grain of reduced iron three times a day.

Strychnine may be used in moderation as a bitter and as a nervous tonic, but it is a mistake to use it as a circulatory stimulant, as it is only an irritant to the nervous system, and produces fictitious strength. Quinine cannot be given in doses which are adequate to control hectic fever, and if any attempt of this kind is made it disorders the stomach and produces headache; 2 or 3 grains, twice or thrice a day as a bitter tonic, are quite sufficient for the average patient.

The syrups of the hypophosphites and lactophosphates have been popular with the profession for many years. In many instances the improvement which follows their use depends upon the iron or strychnine which they contain, and too frequently these syrupy preparations disorder the stomach and spoil the appetite. If it is desired to administer calcium, potash, and soda to such patients, it is better, in the writer's experience, to give an elixir of the glycerophosphates in the dose of a dessertspoonful three times a day to an adult.

When the heart is weak, particular care should be taken in regard to rest, and digitalis may be given, but it should be used in small doses over a long period of time rather than in full doses for a short period. It is quite remarkable what good results follow the use of 2 or 3 minims of a physiologically tested tincture of digitalis three times a day. These doses, maintained for some time, produce a true improvement in the heart muscle and do not disorder the stomach; whereas, larger doses soon upset the cardiac balance, and almost certainly disturb digestion, and even produce vomiting.

For the control of *night sweats* no remedy equals camphoric acid in my experience, given in the dose of 15 to 20 grains, two or three hours before the time that the sweat usually comes on. The best way to administer it is in cachet or capsule, or dissolved in a little brandy. The difficulty of the use of atropine for this purpose is that it checks other secretions and sometimes by so doing renders the cough more dry and annoying.

Fever, as a rule, requires treatment only if it becomes excessive. The patient may be sponged with tepid water and alcohol, and even cool water may be used. But care must be taken that the temperature does not break rapidly and become subnormal. The use of the coal-tar antipyretics is entirely inexcusable. They diminish vital resistance, are apt to produce profuse sweats, and increase cyanosis and dyspnœa when the pulmonary lesions are well developed.

The use of creosote or of any of its derivatives, with the idea that they are beneficial for pulmonary tuberculosis, is based upon an utterly erroneous view of the disease and of the action of these drugs. When *bronchitis* is present as a complication their value as stimulating expectorants is worthy of consideration, and under these circumstances, by improving the condition of the bronchial mucous membrane and aiding expectoration, they may eventually help the patient, but they certainly do not exercise any influence upon the tuberculous process itself. Worse than this, in many instances they do not even act as expectorants, unless given in doses so large as to disorder the stomach. The number of unfortunate consumptives whose struggle with their disease has been lost through disorder of the digestion arising from the administration of expectorants is not comforting to contemplate.

Cough is to be controlled by the use of such cough sedatives as heroin in the dose of $\frac{1}{24}$ to $\frac{1}{16}$ of a grain three or four times in twenty-four hours. A very useful plan of treatment under these circumstances is the administration of the elixir of terpin hydrate and heroin in the dose of a teaspoonful every four hours. In some instances cannabis indica is useful as a cough sedative. In still other cases, if the cough seems to be due to a dry and irritable condition of the bronchial tubes, quiet and sleep is obtainable if there is disengaged in the air of a room, from a bronchitis kettle, steam which arises from water into which is poured creosote, oil of pine, and oil of eucalyptus, equal parts, to the extent of $\frac{1}{2}$ to 1 drachm. Care should be taken that the patient does not go out-of-doors into the cold atmosphere after inhaling the warm steam-laden atmosphere.

When a *laryngeal tuberculosis* develops, these steam inhalations are often exceedingly valuable.

Sometimes an excessive cough at night can be stopped by giving the patient a drachm of Hoffmann's anodyne, or a little spirit of camphor. In other instances morphine in the dose of $\frac{1}{20}$ to $\frac{1}{12}$ of a grain is required, but opiates are to be carefully avoided if other measures of relief are sufficient.

When the cough depends upon the accumulation of large quantities of material in the bronchial tubes or in cavities, it is of vital importance that it should not be arrested by the administration of drugs; for it is an effort on the part of nature to get rid of materials which, if they are retained in the lung, will greatly increase septic poisoning. This is particularly true of morning cough, which, though it is often exceedingly annoying, is really an effort to empty a cavity which has become filled during the night. Such coughing can frequently be aided by placing the patient in such a position that the cavity will readily drain into its supplying bronchus.

Whenever the physician in the treatment of pulmonary tuberculosis is tempted to employ a drug, the question of its influence upon the stomach and digestion should be carefully decided, even though the indications for the use of the remedy which exist in other portions of the body seem very clear and conclusive. Thus, the use of cod-liver oil in the treatment of pulmonary tuberculosis is undoubtedly to be commended, provided that the patient can digest it, and at the same time take ordinary food. Even a healthy person cannot exist on cod-liver oil alone, and it is a vital mistake to impair the appetite and digestion by giving full doses of this sometimes valuable drug. Any sign of indigestion of the oil, as in eructations, or in the passing of oily stools, should be the signal for stopping its use at once. The digestion of good food does more for a patient's vitality than the digestion of good oil.

SERUM THERAPY.—An endeavor has been made to treat tuberculosis by means of antitubercle serum, but so far the results which have been obtained have not been sufficiently encouraging to cause the plan to be very popular, although in the early stages of the disease the use of this serum may act as an aid to the control of the tuberculous process.

Antistreptococcic serum has been used on the ground that nearly all cases of tuberculous cavity are infected by the streptococcus as well as the tubercle bacillus, and that if the former malign organism was removed, or its toxins antagonized, the patient could the better combat the original cause of the illness. If streptococci are found in the sputum in large numbers it may be used to aid the patient, but otherwise its use is futile.

TUBERCULIN.—The employment of tuberculin as a specific remedy for pulmonary tuberculosis has not as yet received general professional endorsement. It is true that a very large number of physicians who are especially engaged in the treatment of tuberculous patients have written papers in which they have highly praised the employment of this substance, and that statistics are numerous which tend to show that it produces advantageous results. It must be admitted, therefore, that at times it does good. But, on the other hand, it is a good rule in practice to follow the majority in the use of new remedies; for new remedies which really are advantageous are taken up and constantly employed by everyone. Probably the conclusions as reached by Trudeau, in regard to tuberculin, represent the real facts of the case when he says: "My experience with tuberculin treatment at the Sanitarium thus far has led me to believe that when carefully tried, in suitable cases, it has proved apparently free from danger, and that it has seemed to have some favorable influence in bringing about healing of the lesions, probably by inciting the formation of fibrous tissues."

The tuberculin which should be employed is that which is prepared by the more modern methods. It is now made by a number of reliable concerns, and it can be obtained both in this country and abroad. This remedy is not one which is suitable to all cases and should only be given by an expert in its use.

When tuberculin is used for curative purposes the so-called "Tuberculin R." is always employed. It is a very much more powerful product than ordinary tuberculin. This preparation bears this name because of the following facts: Tubercle bacilli are coated with fatty acid. This fatty acid is removed by triturating them in a mortar and washing the mass with distilled water. This mixture is then placed in a centrifuge and it is found that the fluid separates into two layers. The upper layer contains no tubercle bacilli, and it is called "Tuberculin Oberst" (T. O.), but the lower layer, which contains bacilli, is called "Tuberculin Residuatum," or T. R. This lower layer is repeatedly treated by triturating and centrifuging until all the bacilli are removed. The advantage of this product, T. R., is that it does not cause suppuration, but does cause immunization. By care in grading the dose it may be possible to produce a curative effect without causing reaction.

When the physician desires to use T. R. as a curative agent he begins by employing $\frac{1}{500}$ milligram or less if that dose produces a reaction. This dose is prepared by adding 1 c.c. of Tuberculin R. to 500 c.c. of normal saline solution (0.8 per cent.). One c.c., therefore, represents one dose of $\frac{1}{500}$ milligram. As the solution when used should not be more than twenty-four hours old, this method necessarily involves wasting a great deal of the product.

The dose is injected into the tissues of the back by means of a sterilized syringe on every alternate day. It is desirable to avoid reaction, and all febrile movement that may be induced by one injection must have disappeared before another dose is given. After repeated doses the patient may be able to stand very large doses without any reaction and with good effect.

When *vomiting* complicates pulmonary tuberculosis, its cause must be discovered. If it follows excessive cough, the cough must be controlled in the manner already described. If it arises from gastric irritability, 2 to 5 grains of subnitrate of bismuth and 1 to 2 grains of oxalate of cerium may be given an hour before meals. In other instances, where the stomach is depressed rather than irritated, 1 or 2 drops of Fowler's solution before meals is advantageous.

The *treatment of hæmoptysis* consists in the administration of a hypodermic injection of $\frac{1}{8}$ of a grain of morphine if the patient shows great mental perturbation because of the hemorrhage. It does not have any direct influence upon the flow of blood by producing nervous quiet, but it relieves the patient's mind and so quiets the circulation. If the flow of blood is profuse, the patient should be allowed to occupy that position in which it is most easy for him to rid his bronchial tubes of fluid. I have seen relief produced by permitting him to lie flat on his chest with his head resting on the edge of the mattress in such a way that the blood readily flowed from his mouth without violent efforts at coughing.

A multitude of measures have been recommended for the control of the hemorrhage. Manifestly, none of them can exercise much power for good. No one would think of attempting to control the hemorrhage from a ruptured varicose vein in the leg, or from a small artery on the surface of the body, by the internal administration of any drug of which we have knowledge. Such indirect styptics as tannic and gallic acid are useless. When the hemorrhage ceases after the administration of these, or other styptics, by the stomach, it is evident that the arrest must be due to the natural clotting of the blood rather than to any effect of drugs. That this is the correct view of the case is still further emphasized by the fact that the pulmonary bloodvessels are very poorly supplied with vasomotor nerves and with muscular fibres, and therefore drugs which act by contracting bloodvessels cannot exercise any powerful influence in this area. Finally, absorption is so slow from the stomach that it is incredible that styptics can exercise a material effect before the hemorrhage destroys the patient or is stopped by clotting. If the circulation is bounding, a dose of chloral or aconite may be given as a circulatory sedative. Nitroglycerin is also of value.

The use of adrenalin by the stomach is of doubtful value, first, because, as just pointed out, the pulmonary bloodvessels are poorly supplied with muscular fibres upon which the adrenalin can act, and, second, because it is very doubtful whether when adrenalin is placed in the stomach it is not decomposed, or at least largely prevented from exercising its constricting influence upon bloodvessels at distant parts. The use of astringent substances in atomizers is equally futile. All of the fluid strikes against the pharyngeal wall, and may run down into the stomach, but it does not reach the lungs. Only fluid which can be used in a vaporizer, such as that which is driven by a cylinder of compressed air, can by any possibility reach the bleeding spot.

Some practitioners have recommended the application of ice upon the perineum in cases of hæmoptysis, believing that in some reflex manner it diminishes hemorrhage from the lungs, and others have applied a small ice-bag over the cavity from which the hemorrhage takes place. There is much more danger of these measures adding to shock by chilling the patient than there is chance of their doing good. As already stated, hæmoptysis rarely produces death as the immediate result of the loss of blood, and remedies which receive credit for arresting the flow are probably unworthy of the confidence imposed in them.

Tuberculosis of the Alimentary Canal.—Tuberculosis of the alimentary canal may occur in any of its parts from the tonsils to the anus, and, while its development is a comparatively rare primary form of the infection, it is nevertheless met within sufficient frequency to make it of importance. In an analysis of 5142 autopsies William Hunter, the Government Bacteriologist of Hong Kong, found that this condition was rarely present in children under five years, notwithstanding the very great prevalence of tuberculosis among the Chinese.

Tuberculosis of the Tonsils.—The tonsils may contain tubercle bacilli, on their way to the infection of neighboring lymphatic glands, or they may be actually tuberculous themselves, containing in their substance miliary tubercles or caseous foci. These lesions are more frequently met with in children than in adults, and may depend upon autoinfection—that is, the tonsils may be infected by tuberculous sputum which is expectorated (secondary), or they may become infected by the entrance of tubercle bacilli in dust by the nose or mouth or perhaps in the milk of tuberculous cows (primary). Koplik has recently made an interesting report on this subject in the *American Journal of the Medical Sciences.*

Even more important than tuberculosis of the tonsils is tuberculosis of the so-called third or pharyngeal tonsil, constituting the "postnasal adenoid." As is well known, these growths are not rarely tuberculous in origin. From these adenoids the bacilli may pass through the lymphatics and so cause tuberculosis of the mediastinal and bronchial lymph nodes.

Tuberculosis of the Pharynx and Œsophagus.—The pharyngeal wall is not uncommonly the site of miliary tubercles, in the course of chronic pulmonary tuberculosis, and even more commonly tuberculous ulceration extends from the larynx and epiglottis to the pharynx and adds greatly to the discomfort of the patient. Tuberculosis of the œsophagus is exceedingly rare, but some cases have been recorded. It may complicate general miliary tuberculosis, being a part of the systemic infection, or it may arise from the swallowing of tuberculous sputum, or, again, from the extension of the disease from a tuberculous lymph node or vertebra. The ulceration may lead to perforation from within or the reverse.

Tuberculosis of the Stomach.—Tuberculosis of the stomach rarely occurs, probably because its juices protect it from infection. When it does occur, it appears as a miliary tuberculosis due to circulatory infection, or as single or multiple tuberculous ulcers involving the mucous membrane. These ulcers are usually the result of a process starting from an ulcerating gland which becomes attached to the stomach and so causes disease by the extension of the inflammatory process. Van Wart has recently reported an instance of solitary tubercle in the muscular layer of the stomach which is believed to be unique.

Tuberculosis of the Intestines.—Tuberculosis of the intestines is a much more common condition, and in the great majority of instances is secondary to infection elsewhere. Primary intestinal tuberculosis occurs usually in children, and as the result of the ingestion of milk which is infected by the specific bacillus. This primary form has been denied an existence by such excellent men as Leube and others, but so many other pathologists, of whom Bollinger may be taken as a leader, have observed it that its existence is proved.

The secondary or common type of intestinal tuberculosis is usually the result of pulmonary tuberculosis, and arises from the swallowing of tuberculous sputum. When the pulmonary lesions have lasted for a long time intestinal infection will be found at autopsy in a large proportion of cases, about 25 per cent. Statistics have been published by certain pathologists giving over 50 per cent. The lesions are found chiefly in the ileum (80 per cent. of the intestinal cases) just before it joins the cæcum, or in the ileum and colon 45 per cent., in the colon alone 3 per cent., and in the rectum 7 per cent., according to Frerichs. These statistics as to the relative frequency of the various lesions hold true of the disease, as it appears in children as well, even when the malady appears as a primary affection.

Tuberculous infection of the intestine primarily involves the lymph nodes of the bowel, causing them to become swollen by reason of the characteristic cell proliferation which the tubercle bacillus always produces. The solitary glands project markedly above the surface as yellowish-white masses which finally undergo caseation and softening, and then the mucous membrane covering them breaks down, forming an ulcer which is surrounded by somewhat overhanging edges. The ulcers are not very numerous; at times only one node may be involved. If the agminated glands, or Peyer's patches, are infected, separate caseous masses develop, several ulcers form, and finally coalesce, forming a large necrotic surface of very irregular outline. It is interesting to note that this condition is quite different from the process in enteric fever, in which disease the glands are affected generally and the individual agminated mass is uniformly infiltrated. Tuberculous ulcers of the agminated glands usually extend transversely across the gut, whereas the lesion of enteric fever extends longitudinally.

The overlying serosa is commonly thickened, it may contain distinct tubercles, and at operation the diagnosis of tuberculous ulcer may be made without opening the bowel. Fibrosis and thickening with associated contraction may cause stricture and symptoms of obstruction; perforation is not common and is usually overlooked.

Tuberculosis of the cæcum in the neighborhood of the appendix may give rise to the belief that appendicitis or tumor of the bowel is present. (See Symptoms.)

When the rectum is affected the ulceration is preceded by infiltration and caseation in the submucosa, it often encircles the bowel, and the tissues near the seat of the ulcer are frequently dotted with small, yellowish or whitish tubercles, which add to the area of the ulcer as they undergo degenerative change. They also give rise to tuberculous infection of the peritoneum and perirectal tissues and to tuberculous abscesses and fistulæ.

With the distinct and specific lesions just described tuberculous disease of the bowel presents an associated condition of acute and chronic catarrh or, in other words, a true enterocolitis.

Of recent years much has been written of *chronic hyperplastic tuberculosis* of the intestine. In this form the intestinal wall is greatly thickened, the lumen commonly narrowed and sausage-like. Distinct cylindrical segments of the involved tissue may be recognized through the thin abdominal walls. Neoplastic masses which may attain the size of a fetal head occasionally develop, and polypoid growths may occur on the interior of the bowel. The condition is most common in the ileocæcal region, and is rarely restricted to the small intestine, but may involve one or more parts or the whole of the colon, causing strictures with interposed dilated areas, although actual dilatation is rare. The stenosis may be almost complete. Histologically there is marked hyperplasia of the connective tissue of the intestinal wall, and the great thickening, sometimes attaining 5 cm., is due largely to this cause. Caseous areas and even typical tubercles may be absent. The newly formed tissue is often but scantily supplied with bacilli.

SYMPTOMS OF INTESTINAL TUBERCULOSIS.—The symptoms of intestinal tuberculosis consist chiefly in the manifestations met with in most cases of enterocolitis. The patient complains of *looseness of the bowels*, or *diarrhœa*, and a considerable amount of *colicky pain*. With these signs there is *wasting* and *decrease in strength*. The *tongue* may be *coated*, but it is often *unduly clean* and the normal roughness of its mucous membrane is replaced by a *raw-beef appearance*. *Palpation* of the abdomen may reveal *tenderness* at certain points, which is not, however, very well marked, and *auscultation* will show an excessive amount of *peristaltic movement* and *rumbling*. At times the appetite may be excessive owing to the fact that the diarrhœa causes starvation of the tissues, which is recognized by the system and shown in a desire for more food. At such times, in particular, the stools may contain *undigested particles of food*. There are, however, no symptoms in this early stage that can be considered typical, and the presence of tuberculosis elsewhere may be the chief reason for believing that the alimentary canal is involved.

When ulceration occurs the presence of mucopus, blood, and, more important, the discovery that tubercle bacilli are in the stools make it possible for us to state positively the cause of the disease. If the disease develops farther, as it is prone to do if life is prolonged a sufficient length of time, the peritoneal coat of the intestine is involved and gradually a general adhesive peritonitis, such as was described in the article on peritoneal tuberculosis, is produced, with its characteristic thickening of the peritoneum and cicatricial contraction of the omentum and mesentery. This produces *constrictions in the intestine*, which may be due to the peritonitis or to the ulcerative process inside the bowel.

In some cases the inflammatory process produced by tuberculosis of the caput coli is so intense that pain in the region of the appendix may give rise to the belief that an *acute appendicitis* or *appendicular abscess* is present. In a case known to be tuberculous the possibility of this condition is manifest, but in one which has a small and unrecognized tuberculous focus elsewhere, as in the lungs, operative procedures for appendicitis may be hurriedly resorted to when no necessity for them exists.

So, too, the finding of a mass in this region, without sharp pain, may mislead the physician into a diagnosis of malignant growth if the rest of the body be not well investigated for a tuberculous focus. Such a mass may be differentiated from carcinoma by the fact that there is a focus of tuberculosis elsewhere. If the growth be slow it is probably tuberculous; whereas if rapid it is probably cancerous, for cæcal tuberculosis may last two years and cæcal cancer rarely lasts longer than eight months. Further than this, if the patient is below thirty years of age tuberculosis is more likely than cancer; whereas after forty years the reverse is true. The tumor when outlined by palpation in tuberculosis is elongated and the thickened intestine can be felt, whereas in cancer it is usually sharply circumscribed and the rest of the bowel cannot be outlined. Fever is usually present in tuberculosis and absent in cancer. The presence of tubercle bacilli in the stools will, of course, decide the diagnosis, and even if they cannot be found, the presence of a tuberculous focus elsewhere, in a person below forty years, should be considered as pointing strongly to this bacillus as the cause of the growth.

At times the tumor found at the ileocæcal region results in obstruction of the ileocæcal valve, and the colon becomes greatly distended with gas, appearing as a large mass in the sides and in the epigastrium. In other cases the colon undergoes atrophy, and can be felt through the emaciated belly wall as a narrow, thickened band. I have seen the entire colon in a case of this character shrunken to such an extent that it was smaller than the ileum. In doubtful cases resort may be had to tuberculin to determine the true nature of the mass.

PROGNOSIS IN INTESTINAL TUBERCULOSIS.—The prognosis in intestinal tuberculosis is not as grave as in pulmonary tuberculosis, as far as early death is concerned. In the majority of instances the patient dies of the primary focus before the state of the bowel is grave enough to cause death. Such cases often last for several years and have periods of improvement followed by relapse, and characterized by gradual loss of vitality. If death is caused by the intestinal state it comes as a direct result of profound feebleness and exhaustion.

TREATMENT OF INTESTINAL TUBERCULOSIS.—The treatment of intestinal tuberculosis, as in tuberculosis of the lungs, consists to a great extent in the maintenance of the greatest degree of nutrition and vitality that is possible, and this can only be accomplished by an out-door life, plenty of sunshine, the avoidance of fatigue, and the use of such foods as are easily digested in the stomach or in the duodenum, so that the greater part of the nourishment will be absorbed before the lower part of the ileum is reached. In those cases in which excessive peristalsis rapidly carries the contents of the small intestine to the large bowel before absorption can occur, it is needful to insist on small quantities of food being taken at a time, and to order that no water be taken at meals. It is also essential that the patient shall immediately after taking food lie down and rest in order to apply all the nervous energy possible to the process of digestion and to prevent stimulation of the bowel to active movement. For the purpose of arresting peristalsis and diarrhœa the salicylate of bismuth in the dose of 10 to 15 grains three or four times a day may be given, or salol and the subnitrate of bismuth may be used. Another valuable drug is eudoxine in the dose of 10 to 20 grains three times a day in capsule, or bismuth subgallate may be given in the same dose. In some instances iodoform given in keratin-coated pills in the dose of 5 grains four times a day to exercise the influence of this substance on the tuberculous lesions; or if the disease is in the rectum or colon, 20 grains may be injected dissolved in 4 ounces of olive oil, or 5 to 10 grains may be given in suppository. Some relief and comfort can also be obtained by the use of a hot-water bag over the abdomen and by painting the belly wall every few days with tincture of iodine.

Tuberculosis of the Liver.—Tuberculosis of the liver occurs as part of a general miliary tuberculosis, as a form characterized by the formation of fairly large aggregations of tubercles in which the nodules may be as large as a walnut. Tuberculosis of this organ is practically always secondary to disease elsewhere. (See Tuberculosis of the Peritoneum.)

The miliary form is characterized by the formation of miliary tubercles which are intralobular or interlobular in position. They may even be in the walls of the biliary ducts, and vary in size from those so small that they cannot be seen with the naked eye to others which are several millimetres in diameter. When the tubercles are massed together so that they form small nodules, the cells of the liver are of course destroyed, the surrounding cells suffer coagulation necrosis and infiltration with spheroidal cells, and tubercle bacilli may be found in large numbers in the cheesy masses.

Tuberculosis of the Genito-urinary System.—Tuberculosis may involve any part of the genito-urinary tract, and is by no means rarely met with in the testicle, the Fallopian tube, the bladder, and the kidneys.

Tuberculosis of the Testicle.—When tuberculosis appears in the testicle it develops in one or two forms. In one of these the onset is abrupt and accompanied by acute inflammation, and in the other type the disease develops slowly, with no inflammation and without pain. When the acute inflammation of the first type disappears the testicle presents irregular nodules, which also develop in the chronic form. In a large proportion of cases the disease is secondary to lesions elsewhere, but it may be primary, particularly if it begins in the epididymis. Verneuil believes with others that infection may occur during coitus if tuberculous disease of the uterus exists, but that state is very uncommon. Babés has found tubercle bacilli in the vagina.

In nearly all cases, whether the disease be primary or secondary, the lesion begins in the head of the epididymis, forming nodules which undergo caseous changes. The infection spreads to the vas, which becomes thickened and nodular, and to the testicle, the vaginal tunic of which is infected. In more than three-fourths of the cases this secondary infection of the testicle takes place.

When primary infection of the testicle occurs the tubercles also produce nodules, which soften and may form a sac of cheesy matter. Sinuses may form after adhesion to the scrotum has taken place and discharge externally.

SYMPTOMS OF TUBERCULOSIS OF THE TESTICLE.—In the form of the disease characterized by *sudden onset* the symptoms closely resemble those caused by *gonorrhœal orchitis*, for *sickening pain* and *swelling* are present. Instead of subsiding in the course of a week or ten days, the swelling persists, although the pain disappears; but before many days have passed softening occurs and the so-called *abscess* is formed escaping by one or more sinuses. The *swelling* is often *bilateral*, and in some instances massive caseation does not take place, but *hydrocele* develops. In the *chronic painless form* there is *gradual enlargement*, usually of one testicle, with the development of one or more nodules and a sense of weight. In place of caseation a fibroid process may develop.

DIAGNOSIS.—The diagnosis of acute tuberculosis of the testicle can be made only after care has been exercised to exclude the possibility of injury, metastasis in mumps, gonorrhœa, syphilis, and the orchitis of some of the acute infectious diseases such as typhoid fever. A previous history of gonorrhœal orchitis is, however, of importance, for this condition predisposes to tuberculosis of this part. The absence of any of these causes, the presence of tuberculous lesions elsewhere, as in the seminal vesicles or prostate, or in organs farther removed, and the fact that the patient is in young adult life, all favor the diagnosis of this disease being present. The development of suppuration and the finding of the bacilli in the cheesy pus will, of course, decide the diagnosis.

The chronic type must be separated from sarcoma and from the thickening following gonorrhœal orchitis. The absence of any recent history of gonorrhœa, or of urethral discharge, and the presence of an irregular tumor which increases in size, all point to tuberculosis as the cause. The finding of the bacilli proves tuberculosis, but the presence of the gonococcus does not prove the absence of tuberculosis, for obvious reasons. If hydrocele is present the injection of some of the fluid into the peritoneal cavity of a guinea-pig may decide the diagnosis by producing tuberculosis in that animal.

TREATMENT.—In most cases it is far safer to remove the gland. The palliative treatment consists in the ordinary hygienic measures used in tuberculosis and, if the disease is localized, in incision and drainage with iodoform gauze; or in other cases, after the abscess is evacuated, the cavity may be injected with iodoform in glycerin or in olive oil to the extent of 15 drops. In other cases a few drops of this mixture may be injected into the gland at different points every three or four days, care being taken that antisepsis is preserved.

Tuberculosis of the Bladder.—This condition may be either primary or secondary, and when secondary it may be due to infection through the bloodvessels from a distant point, or by direct extension from the prostatic urethra, which is diseased through infection of the prostate gland, which is affected in 97 per cent. of cases of genito-urinary tuberculosis according to Kazywicki. In other instances the tuberculous lesions are transferred from the kidney by the ureter to the bladder. In still other instances the infection passes from the vas deferens or epididymis or seminal vesicles to this viscus. Primary vesical tuberculosis is quite rare, and when it occurs is due to infection from tuberculous female genitals (Fournier). In many instances a case of tuberculosis of the bladder which is seemingly primary is really secondary to an unrecognized infection of the kidney. This renal condition in other instances may be known to exist during life, but it may develop so synchronously with the vesical lesions that it is difficult to tell which organs were first affected. In many other cases the primary lesion may really exist in the prostate or in the seminal vesicles.

The bladder, when affected by tuberculosis, develops grayish miliary tubercles in its epithelial lining, which can rarely be seen through the cystoscope as small gray spots, which, like all other tuberculous growths of small size, tend to amalgamate and form patches which in turn may ulcerate, and so destroy the mucous membrane. The ulcers may be single or multiple, and, like tuberculous ulcers of the bowel, may have irregular outlines with the base covered by greenish or grayish pus. Sometimes they are deep, at others superficial, and in the severe cases they may penetrate the walls of the bladder and cause abscesses, which in turn may perforate the rectum, the vagina, or even the tissues in the suprapubic area. The chief lesions are usually in the area of the trigonum. Tubercle bacilli may be found in the pus in the urine.

SYMPTOMS.—The symptoms of tuberculosis of the bladder are usually not well marked in the early stages, and the *onset* of the malady may be so *gradual* that the disease is well developed before it is recognized. At first nothing more than a little *vesical irritability* may appear, and the urine remains clear and normal in appearance. The microscope may, however, reveal a few red blood cells, and later distinct *hæmaturia* develops, which is characterized by the appearance of a few drops of clear blood at the end of urination. As soon as the mucous membrane of the bladder becomes eroded infection is prone to occur and cystitis develops, and with the appearance of cystitis *pain* comes to be a prominent symptom, associated with tenesmus and a *constant desire to urinate*, which exhausts the patient and prevents sleep. The earliness with which these symptoms develop depends upon the seat of the disease. If it be in the trigonum, they arise promptly; if elsewhere, they may be postponed for months. *Retention of urine* may follow ulceration, or in other cases as the neck of the bladder ulcerates incontinence is produced.

DIAGNOSIS.—The diagnosis of vesical tuberculosis depends upon the presence of these symptoms and the finding of the bacillus in the urine, or by inoculation of a rabbit or guinea-pig with the urine, with the subsequent development of the disease in that animal; but the failure of either of these tests does not exclude tuberculosis. If gonorrhœa, stone in the bladder, stricture of the urethra, or a history of the use of irritating drugs can be excluded, and if no spinal disease exists to cause secondary bladder trouble, tuberculosis should be suspected. The presence of tuberculosis elsewhere, of course, suggests that this disease is the cause of the bladder trouble.

TREATMENT.—For the general plan of treatment in these cases reference must be made to treatises on genito-urinary disease. The bladder must be soothed by alkaline diuretics if the urine is acid, hyoscyamus may be given for vesical irritability, and if the disease is active iodoform in olive oil may be injected into the bladder every day, using a 10 per cent. solution. The bladder should be carefully emptied before the iodoform is injected. In other cases corrosive sublimate 1:5000 may be employed by injection. In severe cases perineal drainage is to be resorted to.

Tuberculosis of the Kidneys.—With regard to the pathway by which the bacillus reaches the kidney two views have generally been held. Hæmatogenous infection is admitted. Until recently an ascending infection has been thought not uncommon, but there is at present a decided tendency to doubt that infection travels from below upward; it has been shown by many observers that tubercle bacilli are occasionally present in the urine of tuberculous patients, even when subsequent examination at autopsy discloses no tuberculosis of the genito-urinary organs, and hence it is not necessary to invoke ascending infection to explain renal lesions secondary to tuberculosis elsewhere.

Tuberculosis of the kidneys appears in an acute and chronic form. The former is of the miliary type and is associated with the signs of tuberculous infection elsewhere, and cannot be treated separately from the general state. The chronic form may arise as a primary lesion, or, far more commonly, as a secondary process due to disease of the lower genital tract. When the disease is primary the bacillus probably gains access to the kidney through the blood; this type is that usually met with in children. But the form ascending from the genitals is that met with in adults, as a rule. Males are more frequently affected than females. The disease is most frequent between twenty-five and forty years of age, but it has occurred in an infant at the breast and in very old men. The lesions are often bilateral.

The pathological process in primary and secondary renal tuberculosis is quite different. In the primary form, in which the infection comes by the blood, the bacilli, resting in the vessels of the tufts and tubules, form small tubercles, which gradually undergo necrosis and so cause a spread of the disease to other parts of the kidney, particularly the calices and the pelvis. The necrosis of tuberculous nodes gives rise to areas of softening or abscess cavities, and these are filled with cheesy material which rarely contains blood and urine, although lime salts are frequently present in the dead tissue. Not only tubercle bacilli but pyogenic and other microorganisms are often present. The capsule of the kidney is thickened and may show scattered tubercles. The size of the organ is considerably increased by the growth of the tubercles and the associated inflammation, forming the so-called massive tuberculosis of the kidney. Finally, the kidney may be shrunken.

As the tuberculous process in the pelvis of the kidney increases the ureter is usually infected, and as a result is often partly occluded. This produces a retention of urine in the kidney and so a secondary *hydronephrosis* develops, or it may be a *pyonephrosis*. Sometimes in the early, and much more frequently in the later, stages of the affection the tissues surrounding the kidney become more or less affected and a perinephritic tuberculosis may appear from rupture of a softened area through the capsule of the kidney.

When that form of renal tuberculosis which is secondary to tuberculosis in the lower genito-urinary tract develops, the ureter is first involved, and thence the pelvis of the kidney. The disease then attacks the tips of the pyramids and so gradually the entire gland is involved, but to a less degree than in the form first described. *Pyonephrosis* is very common in these cases, and obstruction in the flow of urine is usual.

SYMPTOMS.—Often no symptoms appear until the pelvis of the kidney is diseased, when *pain* becomes a marked symptom. This pain may be dull or acute, as if due to a renal calculus, with the characteristic radiation of the pain to the penis and inner side of the thigh. The urinary symptoms are *frequent urination, slight incontinence*, and, later, distinct signs of cystitis develop. Before the pelvis of the kidney is affected the urine may be normal, but afterward it contains pus and blood, the pus coming from this area or from the necrotic ulcerating tubercle. Attacks of *violent pain*, arising from acute hydronephrosis due to blocking of the ureter by cheesy masses, may occur, followed by a free flow of *purulent urine* as the obstruction gives way. Tubercle bacilli can usually be found in the urine, but care must be taken that the smegma bacillus is not mistaken for the specific bacillus, and, as indicated above, the demonstration of the tubercle bacillus does not prove that the infection is in the genito-urinary organs.

The associated symptoms are those of *anæmia, debility*, and *loss of flesh*. A lumbar tumor may also appear.

When the case is grave the question of operation must be considered, and it is important to discover if the disease is bilateral or unilateral before operating. This may be done by unilateral catheterization.

Tuberculosis of the Fallopian Tubes, Ovaries, and Uterus.—The frequency of tuberculosis of the Fallopian tubes is notable. It forms the largest part of all statistics involving the female genitalia, for, as stated below, the ovary and uterus are rarely affected. Tuberculosis of these parts was recognized and reported as early as 1744 by Morgagni. In 1886 Hegar published an important paper on this subject which marked an epoch in its study. To illustrate the great frequency of tuberculosis of the female genital tract the statistics of eight European pathologists may be cited. In 8627 cases of tuberculosis in females, this disease had infected the genitals 208 times. These relative proportions are probably too small.

Unlike tuberculous disease elsewhere, tuberculosis is quite frequently a primary lesion in these parts, the infection being received in some cases from the male during coitus (Verneuil, Cohnheim), but in the majority of instances taking place through the blood or lymphatics. According to the statistics of Schramm, Spaeth, Mosler, and Frerichs, genital tuberculosis is found to be primary in about 18 per cent. of cases of genital tuberculous disease.

Genital tuberculosis is most common during the period of sexual life. Pathologically the condition under these circumstances is like that of an ordinary salpingitis, the tubes being thickened and filled with cheesy material. Because of the inflammation associated with the tuberculous process the fimbriated extremity of the tubes becomes adherent to the ovaries and the uterus may become infected. This condition may develop in children as well as in adults, and it is usually bilateral. True abscess of the tube may arise from this cause and a tuberculous parametritis and peritonitis often start from this nidus. In some cases a miliary tuberculosis of the tube develops.

The ovary is rarely involved, but when this occurs it is always a secondary infection from an infected tube or other adjacent parts or from the blood. The uterus is affected only in very rare instances.

Prognosis.—The prognosis in tuberculosis of the female genitalia is more favorable than would be imagined, provided an early diagnosis is made and operative treatment resorted to. It is, of course, more favorable in these instances if the lesion be a primary one, for if severe disease is present elsewhere, operation may be contraindicated and general recovery impossible.

Treatment.—The treatment is entirely surgical.

Tuberculosis of the Heart.—Ferrend and Rathery have reported a case of tuberculous vegetative endocarditis following primary tuberculosis of the spleen. Tubercle bacilli were found in these vegetations and in the clotted heart blood.

Tuberculosis of the myocardium is very rare. In 1902 Anders collected 71 cases of tuberculosis of the myocardium, and reported one of his own, which were all he could find in literature. Out of 3999 autopsies reported by Valentin and Sangelli this condition was found in only 9 instances. Weigert, however, states that he has found minute tubercles in different portions of the heart in nearly all his autopsies on patients who died from acute miliary tuberculosis.

Tuberculosis of the Thyroid Gland.—Fraenkel and Chiari in 480 autopsies on tuberculous subjects found the thyroid gland affected 13 times.

Tuberculosis of the Brain and Cord.—Tuberculosis of the meninges of the brain and cord has already been mentioned when discussing the tuberculous infection of the serous membranes. The tissues of the brain and cord are, comparatively speaking, very rarely affected. When tuberculous lesions occur in these parts they are practically always secondary to tuberculous lesions elsewhere, but there are a few exceptions to this rule. Thus, Demme has recorded a unique case of tuberculous tumor of the cerebellum in a child of twenty-three days, and he has also had a case in which infection seemingly took place through the nose.

When *tuberculous tumors* develop in these parts of the nervous system they appear as solid or caseous, rounded masses, which resemble the ordinary tuberculous growth as it is seen elsewhere. They vary in size from a millet-seed to an orange. When incised they are caseous, fibrocaseous, or hyaline and calcareous, or all of these changes may be found associated. The surface of the growth is sometimes soft and translucent, and the adjacent brain-tissue may be filled with miliary tubercles, which, coalescing with the main growth, in this way increase its size. The growth does not undergo the rapid changes usually met with elsewhere. Sometimes these nodules become encapsulated by fibrous tissue just as does the ordinary tuberculous growth in the lung, or rapid softening in the surrounding tissues develops and suppuration takes place.

These tuberculous growths do not tend to infiltrate the surrounding tissues. They generally occur in the brain tissue itself, and while it has been asserted that they always spring from the pia mater, this view is of doubtful value. As they often project above the surface, the cerebrospinal fluid is readily infected. One-third of these tuberculous growths occur in the cerebellum, in one of its hemispheres or in the middle lobe. After the cerebellum, the cerebrum is the most common site, and after this Gowers gives the following order: the pons, the cerebral ganglia, the quadrigeminal bodies. As a rule, more than 1 growth exists; but sometimes 2 and sometimes as many as 10 or 12 are present. Thus, Trevelyan found them multiple in 17 out of 33 cases, which is a smaller proportion than is usual, and the largest number in any one case was 4. In a case reported by Middleton there were 20, and in a case reported by Homen there were 12. West and Henoch have each reported a case in which there were 12 tuberculous tumors. There are a few cases recorded in which recovery has taken place notwithstanding the presence of a tuberculous tumor in the brain, and without operation. (For literature see Trevelyan's article in the *Lancet* for November 7, 1903.) The symptoms and treatment of tubercle of the brain are discussed under Brain Tumor.

LEPROSY.

Definition.—Leprosy is a chronic infectious disease caused by a specific bacillus, and is characterized by the occurrence of granulomatous new-growths in the skin, mucous membrane, peripheral nerves, and viscera. The lesions are partly anæsthetic and there is a marked tendency to destructive ulceration and trophic lesions.

History.—The history of leprosy is as old as the written history of the human race. The earliest known records are in two Egyptian papyri of 4260 B.C. and 2400 B.C. The detailed description of leprosy in the third book of Moses is familiar to all. In India and China the earliest writings that unmistakably describe leprosy appeared about 700 B.C. The disease appears much later in European history. It was not mentioned by Hippocrates, and we may assume that it was unknown in his time. It appeared in Greece before 375 B.C. and gradually spread over all Europe, its extensions being generally along the track of conquering armies. Its extensive distribution in the Middle Ages finally brought about stringent restrictive regulations which, beginning in the thirteenth century, served to gradually decrease the disease until now it occurs only in isolated centres of infection.

Distribution.—At present the distribution of leprosy is very extensive, principally in tropical and subtropical countries. It is a mistake to consider that leprosy is essentially a disease of warm climates. In Europe it appears only in small, scattered centres or in isolated cases. It prevails in greatest numbers in Finland, Sweden, Iceland, and Norway, particularly the latter. In Russia it is found in forty-nine provinces, most frequently in the Baltic provinces of Lifu with 609 and Kurland with 201 cases. Isolated cases occur in England, Germany, Brittany, and Italy. The total number of cases in Western Europe at present is estimated at 3000. The disease is found all over tropical Asia. In British India the number of lepers is estimated at 105,000, or one in every two thousand of population. It is believed that leprosy prevails in the southern provinces of China more than anywhere else, although no accurate figures are available. It is very common in Japan, the number of cases being estimated at 23,660 (Souton), and in Ceylon, Persia, Arabia, and the Malayan country. In the Philippines there are probably 15,000 lepers. The disease is widely distributed in Africa, particularly along the upper Nile and the countries bordering along the Red Sea and the Mediterranean. Leprosy was introduced into the Sandwich Islands in 1859, and in 1891 one in thirty of the population was affected.

Much has been said of the early existence of leprosy in America. There is no evidence to show that the disease existed prior to the Spanish discovery. The so-called evidence of pre-Columbian leprosy in America is entirely too vague to justify any deductions. In point of fact it suggests syphilis or sacrificial mutilation more strongly than leprosy. At present lepers are found in large numbers in Mexico and many countries of South America. In Colombia there are said to be seven in every one thousand in the population. There are some cases in New Brunswick and British Columbia. Cuba and the Antilles are severely infected; the latest figures give 1297 lepers in Cuba. In the United States the disease is generally distributed. A recent official report shows that nearly every large city has at least one case, the aggregate number for the United States reaching over 900. The disease occurs in three main foci, namely, one in Louisiana, which has existed since 1785, and has lately been estimated as containing about 500 cases; another in California, the infection having been brought in by the Chinese, and a third in Minnesota, where it is estimated that there are 170 lepers, the number being almost entirely made up of emigrants from the infected districts in Norway, from which region infected persons also carried the disease into the Mormon settlements of Utah. These settlements in Utah have also been infected from Hawaii.

Etiology.—The specific cause of leprosy is the *Bacillus lepræ*, discovered by Hansen in 1871. This bacillus is about the same size and has the same morphology as the tubercle bacillus. Like it it is also acid fast; that is to say, when stained with an aniline dye it does not decolorize readily in the presence of mineral acid. It stains a little more easily than the tubercle bacillus and decolorizes more rapidly. It has not been grown successfully on artificial culture media. It is found packed in very great numbers inside the leprosy cells, but it does not invade the nucleus. It is also found in zoöglea masses in the lymph spaces, in the granulomatous lesions, and in the infiltrated nerve tissues. A number of cases have been reported in which the bacillus has been found in the circulating blood.

Manner of Infection.—There is one case of experimental inoculation on record in the person of a Hawaiian convict reported by Arning. Four weeks after inoculation the disease began to develop with acute pain and thickening of the nerve trunks, and a little while after a typical leprous nodule appeared at site of inoculation. The patient died of the disease in six years. Unfortunately this experiment was made in a leprosy country and the man had a leprous family history, so that the evidence derived from it cannot be regarded as absolutely conclusive. There can be no doubt that in a large number of instances the bacillus gains admission through abrasions of the skin and mucous membrane, and possibly also from the friction of infected clothing.

Corroboration of the idea that the bacilli gain entrance through local lesions is found in the fact that where people go habitually without shoes, as in tropical countries, the disease first appears in the feet in a large proportion of cases. Ehler states that in Iceland the face and hands are most frequently attacked because the remainder of the body is so fully protected by clothing. Boinet, in Hanoi, considered infected earth to be the probable carrier of the disease to the feet of the natives. He found the earth saturated with sputa, crusts, and discharges of the lepers. He was able to demonstrate that the soil of the cemetery at Hanoi was highly charged with bacilli, but this evidence is of little value, as acid-fast bacilli are widely distributed. Carasaquilla believes that leprosy may be conveyed by the bite of fleas, and infection by the mosquito must also be borne in mind, since the bacilli are sometimes found in the blood and have also been demonstrated in mosquitoes. Scabies may transfer the infection. A large proportion of cases are undoubtedly infected in sexual intercourse. The contagion of leprosy is, however, feeble.

The immunity of physicians and nurses is proverbial, although several striking instances, as that of Father Damien, are on record in which attendants on lepers have fallen victims to the disease. It would seem that long intimate contact were necessary to contract it. Hutchinson suggests that leprosy may be conveyed from person to person by commensal contagion; that is to say, by eating food prepared by the sore hands of a leper, and by eating out of infected dishes and utensils. Von Bergmann, in a study of 106 cases of leprosy in Riga, found that 60 per cent. occurred in people who had lived in intimate contact with lepers. In the workhouse at Riga there were 23 cases: 4 who entered with it, 19 who contracted it in the house; 9 cases developed in women whose neighbors in the next beds had leprosy.

Great significance was formerly attached to the factor of heredity in leprosy, but recent studies of the epidemiology of the disease have disproved its importance. Boinet cites a case in which grandfather and grandmother were lepers, while the father and five children escaped although living in a leprous community. Children of lepers removed soon after birth from the infected districts do not develop leprosy, while their brothers and sisters who continue to live in the leper community may contract it. None of the children of Norwegian lepers who have emigrated to the United States have developed the disease. Tonkin, in a careful study of lepers in Algeria, found that only 10 per cent. of the cases had any leprous taint in their ancestry; so that 90 per cent. at least must have derived the disease from other sources. He found, further, that less than 10 per cent. of the children of lepers developed the disease, which is certainly a low percentage of contagion for persons living in close intercourse with lepers, even disregarding the question of heredity. There is nowhere a record of a leprous fœtus, although one or two cases of infants born with leprosy has been reported.

The disease is exceedingly rare under one year; and, in fact, before the fifth or sixth year. It must be recognized as very feebly contagious, therefore, when close and prolonged contact is eliminated. Of the various types the tuberculous is far more contagious than the anæsthetic.

Many writers have maintained that defective nutrition and diet play an important role in this disease. Hutchinson is at present the foremost exponent of the theory that the disease is conveyed by food and that the germs gain entrance to the body through the stomach. He believes that tainted fish carry the infection, although leper bacilli have never been found in them. This idea of fish serving as the medium for the infection of leprosy is not a new one. They have been suspected in all ages, and it is true that leprosy occurs chiefly in countries where fish forms a staple article of food, and where a large proportion of the inhabitants are engaged in fishing. There is nothing inherently improbable in the theory that fish may carry the infection, or that a fish diet may represent a common additional factor in the development of the disease. As against this theory, Hansen maintains that it is necessary to demonstrate the bacilli in fish. He states that the people of Norway are using more fish than ever at present, but nevertheless leprosy is constantly decreasing.

Morbid Anatomy.—In tuberculous leprosy the lesions consist in granulomatous growths or diffuse infiltration of the skin and mucous membrane. The granulomatous growths are built up of small round and fusiform cells and large vacuolated cells, called by Virchow leprosy cells. These cells are probably of endothelial origin and are packed full of bacilli. They frequently develop into giant cells. The bloodvessels are increased. In diffuse leprous infiltration the same histological elements are observed. The new-growth invades the bloodvessels, hair follicles, and sweat glands. The bacilli are found everywhere, but in greatest numbers in the giant and leprosy cells.

In the macular lesions there is a larger proportion of connective tissue, the bacilli are fewer, and none of the large types of cells are seen. In the anæsthetic type, diffuse or nodular infiltrations are found in the nerve trunks. The nerves are firmer than normal and darker in color. The interstitial connective tissue is markedly increased and the axis cylinders are atrophied.

Leprous nodules and infiltrations are found in the liver, spleen, testicle, intestines, and kidneys. In the bones osteomyelitis, necrosis, and atrophy are observed, the bone being replaced in many instances of leprous mutilations by connective tissue. In the larger joints changes occasionally are observed that are very similar to the trophic joint changes of tabes.

Symptoms and Clinical Forms.—Leprosy shows itself under an extreme variety of forms. Its *beginning* is very *insidious* and at first the progress is very slow. Even after a number of years the lesions may be very insignificant and not at all conclusive to the casual observer. The *incubation* period is uncertain, and is usually accepted as very long. The average incubation is assumed to be between two and three years, although cases are reported in which it is supposed to have lasted from ten to twenty-seven years; these prolonged instances must be viewed with suspicion as to their accuracy. In many other cases periods as brief as from three or four weeks to three or four months are given. It will be recalled that the incubation of the Hawaiian inoculation case was four weeks.

Another disputed point in the symptomatology of leprosy is the existence of the primary sore or the *leprous chancre*, as it has been called. It has been shown that all evidence points to direct inoculation as the source of leprosy, and it is not unreasonable to assume a primary sore at the point of inoculation of the germ. Many observers hold that such a sore does occur, and that in a large majority of cases it is located on the *nasal septum*. Thus, Stickler was able to demonstrate ulceration of the septum in 128 out of 153 early cases.

Leprosy begins with marked prodromata, of which *fever* is the most common. It comes in crises of several days' duration and is usually mistaken for malaria. It is entirely analogous to the pre-eruptive fever of syphilis. *Head* and *joint pains* are common as well as general malaise, with frequent *drenching* and *exhausting sweats* unassociated with fever. *Epistaxis* is a common and early symptom and corroborates Stickler's observation of the existence of a primary ulcer in the nose. After lasting from a few months to two years, an unusually severe attack of fever ushers in the *primary eruption* or *macular stage* of leprosy.

Macular eruptions consist in erythematous patches, smooth, shiny, and slightly elevated. They occur all over the body, but are more profuse on the face, the backs of the hands, and forearm. The supraorbital ridges and malar prominences are commonly affected. The hairy scalp is not invaded. The patches come and go, although the later crops show a tendency to persist and for the skin to become slightly thickened. When the macules invade the hairy portion of the body, the hair is lost or becomes white and downy. The macules are anæsthetic, particularly in the centre of the patches.

Next follows the stage of development of the *leprous nodules* or the deposit of specific leprous infiltration. Here the disease may be broadly divided into three clinical types: first, tuberculous or nodular leprosy, in which the skin and mucous membrane are invaded by the specific new-growth; second, the anæsthetic or nerve leprosy, where the leprous deposits take place in the nerve trunk; third, the mixed type of leprosy, which combines both of the foregoing and to which class all cases ultimately tend.

TUBERCULOUS OR NODULAR LEPROSY may begin with or without the macular stage. The leprous nodules appear under the skin, particularly *about the face and ears;* the nodules are palpable as distinct, tough, flattened masses under the skin. They have a peculiar, rubbery consistence, are painless, and freely movable. *Diffuse infiltration of the skin* takes place, and large, flat, leprous patches are formed. The leprous lesions grow steadily larger, until on the face large folds and masses of tissue are formed, producing the condition known as Leontiasis, or the *lion-like face of leprosy*. New nodules appear and other portions of the body are invaded until practically the leprous lesions cover the entire skin surface. In the early stages the nodules occasionally diminish in size or may be entirely absorbed. Later they break down and extensive ulceration occurs. At this stage the mucous membrane becomes extensively involved. The cartilaginous structures of the nose are completely lost, and leprous ulcerations of the larynx occur, with loss of voice and ultimately cicatricial stenosis, which may cause intense dyspnœa. Extension of the leprous ulceration into the corneal structures may cause blindness. The ulcerations produce horrible and characteristic deformity.

Death takes place from exhaustion or intercurrent infection.

ANÆSTHETIC LEPROSY.—Anæsthetic leprosy shows a very different picture from the foregoing. The macular stage continues and is marked by increased *pigmentation* and *complete anæsthesia* of the macule. Gradually extensive *neuritis* is developed in the trunks of various nerves, causing *pain*, severe and neuralgic in character, and later large areas of anæsthesia or *numbness*. The superficial nerve trunks, the posterior auricular, and the ulnar where it winds about the internal condyle, are palpably thickened. *Bullous eruptions* occur on the hands, feet, and elbows, and along the course of the nerves breaking down and leaving extensive spreading and destructive trophic ulcers. As a further result of these trophic disturbances *extensive contractures* develop, and fingers and toes slough away. In the older cases the areas in the distribution of the affected nerves, which at the beginning of the disease were painful or numb, become completely anæsthetic and the muscles become extensively atrophied. When this condition occurs in the hand, a typical *claw-like hand of leprosy* is produced. The atrophied skin is particularly prone to injuries and to extensive ulceration. The course of this type of leprosy is excessively chronic.

The mixed leprosy presents a combination of the features of tuberculous and anæsthetic leprosy. In all cases of extensive tuberculous leprosy the nerve trunks eventually become involved, and the evidences of neuritis and trophic disturbances are added to the clinical picture.

Diagnosis.—In advanced cases leprosy could hardly be mistaken for any other disease. The lesions are too striking and distinctive. Difficulties in diagnosis arise in early undeveloped cases, particularly in the macular and anæsthetic types. Here, as Manson says, the touchstone of diagnosis is the anæsthesia. It should be sought for in the centre of macular areas, and in the centre of recent nodules, if any exist. Advantage may also be taken of the fact that leprous areas do not perspire. Baelz uses an ingenious plan not only for diagnosis, but also for mapping out the involved areas. Aniline is rubbed on the skin and pilocarpine is administered hypodermically. The leprous areas do not sweat and consequently remain unstained.

The most satisfactory diagnosis consists in identification of the bacilli. For this purpose a leprous nodule may be clamped, punctured with a needle, and the exuding drop of fluid properly stained and examined. Even in the very earliest cases, long before other symptoms appear, leprous thickening can be demonstrated in the ulnar or posterior auricular nerves. In doubtful cases the minute fragment of one of these nerves should be excised and stained for the bacilli.

Morrow calls attention to the fact that leprosy and nasal catarrh go hand in hand, and that in the large proportion of cases bacilli can be demonstrated in the nasal secretion. It has also been shown that where nasal secretion is scanty a few doses of potassium iodide will cause a sharp catarrhal flow, in which the bacilli may be demonstrated.

Prognosis.—In the vast majority of cases leprosy slowly tends to a fatal ending from exhaustion or intercurrent infection. Isolated cases, however, occur in which the disease is arrested or cured. These instances of arrest are more common in anæsthetic leprosy than in the tuberculous type. Many of these cases survive twenty or thirty years, and in a large proportion of these the specific process is probably ended, although the extensive damage by nerve involvement and trophic lesions remain. Even tuberculous leprosy has been known to disappear.

Many cases are arrested, and, after a long period of years, death occurs from some other disease not associated with the leprosy. These reported cures must, however, be accepted with extreme caution. It is probably more fair to speak of them as arrests. Mention must also be made of the marked amelioration that occasionally follows the removal of a leper from the country in which he has developed the disease.

Treatment.—The treatment of leprosy comprises careful attention to cleanliness, provision of good hygienic surroundings, abundance of nourishing food, proper clothing, hygienic dwellings, and light occupation. As special remedies gurgun oil and chaulmoogra oil have been extensively used. The former has been abandoned. Chaulmoogra oil (oleum gynocardii) may be administered by the mouth in doses of 2 drachms, or by the rectum, in an emulsion with hot milk, when it is badly borne by the stomach. In some cases this drug seems to have almost a specific action. In a few cases apparent cures and in a great many cases very marked improvement is observed. The oil may also be administered hypodermically.

Unna advises the internal use of massive doses of ichthyol combined with inunction of pyrogallic and chrysophanic acid externally. The treatment is supplemented by hot baths. Several cures have been reported following this plan. Crocker reports improvement from subcutaneous injection of the bichloride of mercury, and De Luca from intravenous injections of mercury according to Bacelli's method. Raynaud reports marked improvement following the administration of sodium cacodylate. Roussel reports an apparent cure following the administration of potassium chlorate, and Manson a case of nerve leprosy apparently cured by thyroidin. Tuberculin, antivenene, and the iodides have been used and do more harm than good. Danielsen recommends salicylate of soda in ascending doses combined with tonics. He believes that it cures leprosy if administered early. Baelz uses salicylic acid locally. He treats about one square foot of skin at a time by rubbing the diseased area with pummice stone until blood appears. Salicylic acid is then applied in a 20 per cent. ointment with lanolin and vaselin. This treatment is combined with the oil of gynocardium internally and hot baths.

Nerve stretching has been recommended and practised for the relief of the painful complications and trophic disturbances of nerve leprosy.

Prophylaxis.—The only means of limiting leprosy is by isolation. While absolute segregation is the ideal and proper measure, it meets with so much opposition, and so many cases are concealed, that in the long run a better purpose is served by adopting a reasonable compromise similar to that followed in Norway. This includes caring for indigent lepers in an asylum and allowing those whose people are able to take care of them to do so at their homes under proper restrictions. The Russian laws only isolate the tuberculous and mixed cases. Although it is true that the nerve cases are much less contagious, this regulation must be regarded as a mistake.

FEBRICULA.

Definition.—Febricula, sometimes called ephemeral fever, is a condition usually met with in children, and is undoubtedly a disturbance of the heat mechanism of the body produced by the action not of one but of several agents; that is to say, many different causes are responsible for it rather than one specific cause.

Etiology.—The causes of febricula are very numerous. In some instances it is probably the result of some infection which is overcome by the protective process of the body before it can develop into a full-fledged disease, such, for example, as an aborted influenza, or even one of the specific eruptive fevers, or some infection entering by the tonsils. Some years ago physicians believed that infectious diseases could be aborted in their early stages by proper measures designed to aid nature. This view fell into disrepute, but our knowledge of protective processes and of antitoxic bodies makes it probably true. In children an ephemeral fever is often due to gastrointestinal catarrh. Sometimes it is due to gastrointestinal intoxication.

Symptoms.—The patient after a feeling of wretchedness, rarely lasting more than a few hours, is found to be mildly *febrile*, the temperature being about 102° to 103° at the most. There may be *flushing of the face* and even *delirium* in young neurotic children. The *pulse* and *respirations* are *quickened*. The fever usually, but not always, ends by lysis in about three days to a week.

Diagnosis.—The diagnosis of febricula is made in most instances after the patient is well, for until then no one can tell that the symptoms are not the early signs of one of the acute infectious fevers. The important point is, not to be content with a diagnosis of febricula, which is but another way of saying that the condition is uncertain, but to search carefully for the real cause.

Treatment.—This consists in rest in bed, the use of a little calomel followed by a saline, and the employment of a mixture of citrate of potassium and sweet spirit of nitre to keep the kidneys active.

MILK SICKNESS.

Definition.—Milk sickness is a disease which is usually communicated to man by the milk, or by the butter or cheese made from the milk of cows ill of a malady called, when it affects cattle, "trembles," or "slows." When man is infected it is given this name and the additional one of "puking fever." The malady is one which exists in the Southwestern United States, and apparently does not affect animals on the Atlantic seaboard. It is said that the flesh of affected animals, if not cooked, may convey the infection. It is important to know that the infection may be transmitted from a seemingly healthy cow some time before it develops symptoms of the disease.

Symptoms.—The symptoms in the cow consist in refusal to eat, redness of the conjunctiva, staggering gait, and muscular tremors, whence the name "trembles." In man, after a day or two of ill-health, the patient is seized with *epigastric distress,* followed by *vomiting* and *constipation, fever,* and *thirst. Muscular tremors* also appear. The *breath* is peculiarly *foul* and *offensive* and the *tongue swollen.* If the disease is severe the patient may develop *typhoid symptoms,* and even become *delirious, comatose,* or *convulsed.* In fatal cases death may come as early as the fourth day, or be deferred for two or three weeks. The more severe the cerebral symptoms, the more grave the prognosis.

Treatment.—The treatment is purely symptomatic and consists in the use of stimulants or sedatives as they may be needed.

WEIL'S DISEASE.

Weil's disease is a very rare infectious malady, first described by Weil in 1886, which for some unknown reason seems particularly prone to attack butchers. It is characterized by the development of *fever* and *acute jaundice,* and appears usually in the warm months of the year. The victims of its onset are usually young and middle-aged adults, and the symptoms are *severe headache, lumbar pain,* and *cramp-like* sensations in the legs and arms. The masseter muscles also suffer from severe pains. Jaundice develops as an early symptom, and the liver and spleen are found swollen and tender on deep palpation. The stools may be putty-colored, as if from obstruction of the gall-ducts. The fever may last two weeks, often rises to 103° or 104°, and is characterized by sharp remissions, as in sepsis. Albuminuria may occur, and even coma may develop. Recovery usually takes place, but convalescence is slow.

Weil's disease must be separated from bilious remittent fever, catarrhal jaundice, and phosphorus poisoning. This separation is readily accomplished if the characteristic symptoms just described are compared to those presented in the course of these conditions. Remittent fever, which is the malady most closely resembling Weil's disease, is differentiated by finding the æstivo-autumnal parasite in the blood, and phosphorus poisoning by the history of the case and the phosphorescent character of the vomit, which can be noted if it is examined in the dark. Treatment is entirely symptomatic.

GLANDULAR FEVER.

Definition.—Glandular fever is an acute infectious disease characterized by a moderate febrile movement and a painful enlargement of the cervical lymphatic glands.

History.—The first accurate account of this disease was published in 1889 by Pfeiffer, although it is probable that the condition described some years before by Filatow, of Moscow, under the name of idiopathic inflammation of the cervical glands, was in reality glandular fever.

Cases have been reported from different countries on the Continent, and from England and the United States. J. Park West, of Bellaire, Ohio, has reported an epidemic in which ninety-six children were attacked, this being the most extensive epidemic on record.

Etiology.—The specific micro-organism of this disease, if there be one, has not been discovered, but that the disease is infectious in nature is shown by the fact of its occurrence, as a rule, in small epidemics involving several members of a family. The disease generally occurs before puberty, although Galvagni and A. E. Roussel have observed it in adults.

Symptoms.—The onset is *sudden* and is characterized by moderately high *fever, restlessness, headache, pain in the limbs,* and soreness and pain in the neck, which is increased by turning the head or by swallowing. The temperature ranges from 101° to 103° and may even go as high as 104°. The *bowels* are usually *constipated,* although in some of the severe cases observed by West *copious discharges* of thin, green feces mixed with mucus took place shortly before the beginning of convalescence. *Abdominal pain* is a common symptom, and pressure over the lower part of the abdomen, particularly in the midline between the umbilicus and the symphysis pubis, often elicits pronounced tenderness. The *swelling* of the *anterior cervical glands,* which usually begins on the left side and then extends to the right, attains its maximum between the second and fourth days. The glands are hard, easily distinguishable from one another by palpation, and are very sensitive to pressure. Suppuration rarely occurs. Examination of the pharynx reveals either a normal condition or a slight hyperæmia. The liver is always enlarged, and not uncommonly there is considerable swelling of the spleen. Acute nephritis is the most frequent and most serious complication. The duration of glandular fever is variable. European physicians have described a mild or abortive form in which the temperature falls to normal on the second or third day, although the cervical glands remain swollen several days longer. The average febrile period, however, is from seven to ten days. In West's ninety-six cases the average duration of the disease from its onset until the complete disappearance of glandular swelling was sixteen days.

Diagnosis.—The occurrence of a sudden febrile attack accompanied by an early, painful enlargement of the anterior cervical lymph glands, without any inflammatory involvement of the pharynx, makes recognition of the disease easy.

Prognosis.—Prognosis is always favorable. Convalescence is rapid, as a rule, although in some instances it is retarded by a considerable degree of depression and anæmia.

Treatment.—Experience has demonstrated that there is no drug which will influence the duration or course of this disease. The patient should be confined to bed and a mild aperient given to overcome constipation

MOUNTAIN FEVER.

So-called mountain fever really does not exist as a separate entity. It has been proved to be an aberrant form of typhoid fever infection in a number of instances, particularly by the United States Army surgeons or those attached to the United States Health and Marine Hospital Service. In some cases the infection may be paratyphoid. (See Paratyphoid Fever.) In still other instances the fever may be due to an anæmia depending upon intestinal parasites such as the *Anchylostomum duodenale.* Some cases may be "tick fever." (See below.)

TICK FEVER.

Definition.—Under the name of "tick" or "spotted fever," a febrile malady has been carefully described within the last few years. It is prevalent in Western Montana, particularly among the eastern foothills of the Bitter Root Mountains. Curiously enough, it is chiefly limited to the western side of the Bitter Root Valley, in an area from four to ten miles wide and fifty miles long. Fewer and milder cases occur in Idaho, Wyoming, Nevada, and Oregon, at a level of from 3000 to 4000 feet. Its occurrence is limited to the period of from the middle of March until the middle of July. Herders and ranchmen are usually the persons affected.

This form of so-called spotted fever is not to be confused with cerebrospinal meningitis.

Etiology.—According to the studies of Wilson and Chowning, and of Anderson, this disease is due to a parasite which is conveyed to man by the bite of the tick. To this parasite they gave the name *Pyroplasma hominis,* and therefore suggested that tick fever be called *pyroplasmosis hominis.* According to these observers this parasite in its smallest form slightly resembles the small hyaline bodies of malarial fever, but it is at no time pigmented. Stained with Laveran's or Nocard's stain chromatin masses are seen, as a rule, near one end of the parasite. It appears free in the blood or in the body of a red corpuscle. Often two parasites are present in one blood cell, but rarely more than this. They may be joined together by their lesser extremities, either when in the corpuscle or when floating free in the blood. Sometimes the so-called parasite seems to change its position in a blood cell, but no amœboid movement has been seen.

In the second phase of its development the organism in the red blood corpuscle appears to be solitary and distinctly larger than in the first phase just described. During this stage of its development they claim that it also presents active amœboid movements.

The red cells of the general circulation are not infected in great numbers, but if the blood in the capillaries of congested tissues is examined, it is found that a large number of the red cells have become infected. The greatest degree of corpuscular infection is said to be met with in the spleen, liver, lungs, and kidneys.

Since these observations of Wilson and Chowning, Stiles and Craig claim to have proved the non-existence of these so-called parasites, the former asserting his inability to discover any structures which lead him to a belief in the presence of a protozoön as a cause of the malady. Craig believes that the changes seen by Anderson in the red blood cells are not due to a parasite, but dependent upon certain alterations chiefly taking place in the hæmoglobin, such as are often seen during the course of epidemic influenza, typhoid fever, measles, variola, and other acute infections. The cause of this disease, if indeed it ultimately proves to be a separate entity, is, therefore, at present of uncertain character, the more so as there is reason to believe that the *Pyroplasma bigeminum*, which is the organism of Texas cattle fever, is incapable of infecting man.

Symptoms.—The disease makes its appearance in from three to ten days after the bite, with *chilly sensations*, *malaise*, *nausea*, *headache*, and *muscular soreness*. The *bowels are constipated*, the *conjunctiva congested*, the *urine scanty* and *albuminous*, and *slight bronchitis* occurs. *Epistaxis* is a constant symptom. The *fever* rises sharply after the chill, but has morning remissions like typhoid fever. The *rapidity of the pulse* is out of proportion to the fever, often amounting to 110 or 140 per minute, and there is a very moderate leukocytosis. *Respiration is rapid.*

Its acme is reached by the twelfth day, when it gradually falls by lysis for four days more, and so convalescence begins. In fatal cases the fever remains high, about 104° or 105°, or even 106°. The pulse is usually very rapid and thready, and the *blood rapidly becomes anæmic.*

The *rash* usually develops about the third day on the wrists, arms, legs, and forehead, and later on the back, chest, and abdomen. This rash, except on the abdomen, is often exceedingly profuse.

The skin presents an eruption of bright-red macules, varying from a pinhead to a split pea in size, and in severe cases they become petechial in character. They fade as the fever falls, and the skin may desquamate. Sometimes in the later stage of the eruption they appear like the marks on a turkey's egg. Anderson states that slight *gangrene of the fingers*, *toes*, and *scrotum* may occur. *Albuminuria* is a constant symptom.

Diagnosis.—The section of the country in which the disease occurs, the history of tick bites, and the finding of the changes in the blood are the factors which make the diagnosis positive. From ordinary purpura the disease is separated by the lack of sore throat and the absence of arthritis. From typhoid fever the diagnosis may be quite difficult. In that disease, however, the rose rash appears first on the belly on the ninth to the twelfth day, whereas in tick fever it appears as early as the third day, and on the wrists. The discovery of a positive Widal test in the blood will settle the diagnosis. Typhus fever is conveyed from man to man; tick fever is not directly transferred. Typhus fever breaks out in groups of persons. Tick fever always appears sporadically.

Prognosis.—The disease is a very grave one, for out of 126 cases 88 died, a mortality of about 70 per cent. Sometimes the mortality is as high as 90 per cent. Death usually ensues about the sixth to the twelfth day of the illness, but it may occur as early as the fourth day. Experience with cases which have occurred in Idaho, Wyoming, and Nevada shows that the mortality is far less in those States than in Montana.

Treatment.—So far as is known quinine seems to act as a specific in this disease. Anderson thinks it should be given in the dose of 15 grains every six hours, hypodermically, and its use continued into convalescence. The heart must be supported by stimulants if it is feeble, and the kidneys flushed by copious draughts of water. The tick, if found, should be removed from the skin and the part cauterized with 95 per cent. carbolic acid to prevent infection, if possible.

FOOT-AND-MOUTH DISEASE.

Definition.—This is an acute infectious disease of herbivorous animals, which sometimes attacks omnivora and which spreads in epidemic form over large territories, causing great mortality in the animals affected. When the disease attacks the cow the animal becomes feverish, suffers from swelling of the mucous membranes of the mouth, and develops blisters on the edges of the tongue and on the lips. These blisters become discolored and rupture, leaving ulcers. At times similar lesions appear on the teats. The milk of such animals is discolored and seems to be thickened as if by mucus.

The disease is rarely met with in England and America, and only possesses interest to us, because it is capable of being conveyed to man. This conveyance occurs in the case of children by the use of the milk of the diseased cows, and in adults, as a rule, by this means or by cheese or butter.

The *symptoms* in man are like those of *severe stomatitis*, associated with *fever*. Recovery in man usually occurs, but a mortality of about 10 per cent. is recorded.

The cause of the disease has not been isolated. Even a porcelain filter does not arrest the organism, if organism it be, that causes the malady.

MILIARY FEVER.

Definition.—Miliary fever, sometimes called "the sweating sickness," is an acute epidemic disease characterized by fever, profuse sweating, an eruption, and a peculiar sense of constriction in the epigastrium.

History.—The disease was far more prevalent in the seventeenth century than it has been since that time, but it still appears in certain parts of the world. Almost every country in Europe, including England, has suffered from its presence, but it has not, so far as I have been able to discover, ever appeared in the United States. The most recent epidemics have been in Austria in 1892 and in Styria in 1893.

Etiology.—The cause of the disease is unknown, but it is an acute infection, apparently resembling influenza in the manner of its spread, although it does not, as a rule, attack large numbers of people throughout a wide area, as does that disease. On the contrary, it is very often limited to the population of a single town or district.

Symptoms.—The symptoms of miliary fever are ushered in, as they are in all the infectious diseases, by *lassitude, headache,* and *anorexia.* This prodromal stage may last a day or two or be so brief as not to be recognized. The patients go to bed well, and wake in the morning to find themselves ill and suffering from a *drenching sweat,* which persists throughout the illness. The bowels are usually confined, the *tongue coated,* and the *pulse* but little altered in character for the first few days of the illness. A symptom complained of by the patient is one of *oppression,* as if the air of the room were hot and vitiated. The *fever* is usually high, rising to 104° to 105°, and in fatal cases to 107°. On the *third day* there appears on the skin an outbreak of *red miliary papules,* which often develop a white tip before they disappear, and between these are scattered large numbers of pearly vesicles, like sudamina, which seem filled with clear fluid. Prior to the appearance of this eruption a peculiar pricking or tingling sensation is felt in the skin. When the eruption has faded, *desquamation* sometimes occurs. The entire progress of the malady is usually completed in nine or ten days.

The following facts are also noteworthy, viz.: The *sweating* is constant, but is *characterized by paroxysms,* in which it becomes still more profuse. The rash appears on the mucous membrane of the palate and cheeks. The *sudamina,* or pearly miliary vesicles, although they give the name to the disease, are *not a constant symptom* in all cases.

Abortion nearly always occurs if a pregnant woman is attacked.

Miliary fever causes rapid emaciation.

It is very prone to be followed by a relapse, but the relapse is rarely fatal.

Prognosis.—Recovery usually occurs. In severe cases, in which the onset is fulminating, death may occur as early as the eighth hour after the attack begins. These cases have marked nervous symptoms, consisting of convulsions, delirium, and coma. Evidently the patient is overwhelmed by toxæmia. The mortality rate in various epidemics has varied from 5 to 25 per cent. The outlook in children is usually good.

Treatment.—This consists of cold sponging to control excessive fever, the use of copious draughts of water to compensate for the loss of water by the skin, and for the purpose of flushing the kidneys, and in the administration of stimulants, if they are needed, to support the heart.

JAPANESE RIVER FEVER.

Definition.—Japanese river, or flood fever, is an acute, infectious disease, occurring in a very limited area in the island of Nippon, beginning with a necrotic ulcer, attended by an exanthem and continued fever.

The disease occurs in laborers who cultivate the inundated bottom lands of several rivers on the west coast of the island of Nippon.

Etiology.—The etiology is obscure. The infection is evidently carried in the corn and hemp grown in these sections, and the primary eschar probably marks its point of entrance. The Japanese ascribe the disease to the bite of a small acarus. Baelz denies this. Given equal exposure, neither age nor sex shows any difference in susceptibility to the disease.

Symptoms.—After several days of *malaise* and *repeated rigors*, the disease begins with the breaking out of a *small, round eschar* in the groin, axilla, or neck. The neighboring lymphatic trunks and glands become swollen, hard, and painful. A continued *fever* ranging between 103° and 104° develops, with bronchial cough and marked conjunctivitis. About the sixth day a coarse, *red, papular eruption* appears on the face, forearms, legs, and trunk. This eruption fades away in from one to seven days. The fever lasts a week longer, when it falls rather rapidly, and convalescence begins with a separation of the eschar from the primary sore. The mortality varies between 15 and 70 per cent. in different epidemics. No definite postmortem changes are found, beyond congestion of the bronchi, occasional hypostatic pneumonia, marked enlargement of the spleen, and swelling of the mesenteric glands.

Treatment.—Treatment is symptomatic. Baelz advises quinine and salicylates as antipyretics to be used with caution.

FRAMBESIA (FRAMBŒSIA TROPICA, YAWS).

Definition.—Frambesia, or yaws, is a chronic contagious and infectious disease, characterized by the appearance of a diffuse granulomatous eruption on the skin.

History.—The history of yaws begins with the historians of the Spanish conquest of America. It is a disease very closely confined to tropical countries and very widely distributed in Africa, in the coast countries of tropical Asia, and in many of the Pacific islands. It also occurs in Central and tropical South America and the Antilles. The disease was exceedingly common in Cuba and the southern United States during the first half of the nineteenth century, having been brought there during the slave-trading days. At one time it caused such a degree of disability among the negroes that the planters were forced to adopt stringent rules for its limitation. Most of the large plantations maintained isolation barracks, or "yaw houses," for these cases. The disease still lingers in Cuba and the rest of the Antilles. It has all but disappeared from the United States.

Etiology.—Many bacterial forms have been isolated from yaw lesions, but as yet the specific cause has not been determined. The disease can be, and frequently is, conveyed by direct inoculation, intentional or accidental. Such inoculation may take place in wounds, abrasions, and other injuries of the skin. In some yaw countries, notably in Fiji, it is a common practice for mothers to inoculate their children, under the same idea which prevails among our lower classes, who frequently expose their children to pertussis and eruptive diseases, on the theory that the illness must be gone through with some time, and the earlier the better. Heredity has no bearing on the etiology of yaws. Neither does a pregnant or nursing woman with yaws necessarily infect her child. Outside of direct inoculation the disease is conveyed by food, particularly by cooking utensils. In persons particularly susceptible, infection may take place by sleeping in a yaw house. All ages are attacked, but the majority of cases are seen in children. The black, yellow, and white races are susceptible in the order named. As a rule, one attack confers complete immunity. Frambesia is also seen in domestic fowls.

Symptoms.—The incubation period of yaws is very variable. Generally speaking, in inoculation cases, it varies between fifteen and twenty days. In cases ordinarily acquired, the incubation is longer, ranging from fourteen to sixty days. In a small proportion of cases prodromal symptoms, *languor, malaise, headache,* and *rheumatic pains* are observed. This condition is followed by what is known as the primary eruption or the *primary sore,* concerning which there is some dispute among tropical practitioners. In experimental inoculation cases the primary sore is constant and occurs at the point of inoculation. It begins as a *small papule,* which, in the course of a week, is converted into a *shallow ulcer.* In another week the ulcer heals, leaving a slight, thickened scar. In ordinary infection by yaws it is sometimes present and sometimes absent.

The generalized eruption, the so-called *secondary* eruption, begins with the primary sore in exceptional cases, but, as a rule, is delayed for several weeks. Occasionally, in the period between the eruption of the primary and secondary lesions, a dry, scaly affection of the skin is seen.

The secondary eruption begins as *small papules,* which itch intensely. They are scattered all over the body, but are most commonly seen, in order, on the face, neck, limbs, genitals, and trunk. The hairy scalp is not commonly invaded; the axilla very rarely. The lesions are particularly numerous at the mucocutaneous borders, the mouth, nose, anus, and vulva. The eruption is roughly symmetrical. The papules, at first the size of a pinhead and slightly prominent under the skin, gradually increase in size till they are as large as a pea or a hazelnut. Small, *yellow spots of pustulation* appear on the summit of the lesions; the skin cracks; a sticky, yellow, seropurulent fluid exudes, which hardens and forms rupia-like crusts or caps over the summit of the growths. The cap is tough and adherent. When it is pulled off it reveals a shiny, red *papilloma* underneath. This warty growth, the true yaw, resembles a berry in appearance, hence the name yaw, *i. e.*, a strawberry; frambesia from *framboise*, a raspberry. Indeed, most of the local native names for the disease are words which mean berry in their dialect.

The growths resemble syphilitic condylomata in their appearance. They spring from the papillary layer of the skin, and the warty-like lobulations represent the greatly hypertrophied papillæ. The uncovered yaw freely exudes the sticky, yellow pus, already mentioned, and in a little while the cap is reproduced. As a rule, the lesions are painless, excepting where they occur under thick, dense skin, as in the palms and the soles, where tension may cause great pain. Itching is, however, very persistent and annoying. After persisting weeks and months, sometimes passing through recrudescences and successive crops, the lesions gradually grow smaller, the papillomata disappear, and a dry eschar is left, which falls off, leaving a patch of thickened skin, bleached in the negro and pigmented in the light-skinned races.

In old, long-standing, and neglected cases, *severe bone* and *joint pains* develop, and occasionally extensive periostitis and caries occur. These are the so-called tertiary lesions of frambesia. They are not constant; indeed, they never appear in properly treated cases.

Diagnosis.—There are only two diseases with which typical yaws can be confused, syphilis and verruga. Hutchinson believes yaws and syphilis, if not the same disease, are descendents of the same parent stock; that originally they were identical and have become differentiated by thriving for long periods on different soils. Yaws undoubtedly suggests syphilis very strongly, but there can be no question of the duality of the diseases. Syphilis and yaws have frequently been observed in the same individual; syphilitics have been successfully inoculated with yaws, and *vice versa*. Finally, the histological differences are marked. No giant cells are seen in yaws and no thickened bloodvessels.

Scheube believes yaws and verruga to be identical, but Glogner has recently drawn a careful distinction between the histology of the two diseases, and has clearly shown that they are not identical.

Prognosis.—The prognosis is uniformly good.

In patients reduced by disease and in infants the prognosis of yaws is not so favorable.

Treatment.—Iodide of potash is the remedy for frambesia. Mercury not only does not do these patients good, but actually seems to do them harm. Stomatitis occurs with the greatest facility and is very severe. When the general condition is low, arsenic, iron, and the bitter tonics are indicated. Most tropical practitioners advise local treatment of the lesions. This includes antiseptic and stimulating applications and removal of old lesions with the curette. The prophylaxis of yaws consists in cleanliness and isolation of the infected. Great care must be taken of abrasions and cuts, and infected dwellings should be avoided.

VERRUGA (VERRUGA PERUVIANA).

Definition. Verruga (a wart) is a chronic, infectious, and inoculable disease, characterized by initial fever, rheumatic pains, anæmia, and the development of granulomatous lesions (warts) on the skin, mucous membranes, and internal organs.

Distribution.—Verruga is limited to certain high valleys of Peru, on the Pacific slopes of the Andes. At present it is principally observed in the valleys of Huarochiri, Tanyos, Rimac, and Canta, at elevations varying from 3000 to 8000 feet above the sea. It is not observed at lower levels. Cases are also reported from the mountain districts of Ecuador, Bolivia, and Chile. The disease has existed since remote times in Peru, possibly in wider extension than at present. It occurred among the soldiers of Pizzaro's expedition, and is first mentioned by Zarate in his *History of Peru* (1543).

Etiology.—Verruga occurs in small epidemics, but is not contagious. Carrion, a medical student, in 1885, proved its inoculability on himself and died of the infection. As a rule, one attack of the disease confers immunity. The specific cause of verruga has not been established. The belief is prevalent among the population that the waters of certain springs are the cause of the disease. Moisture, heat, and elevation above the sea seem to be necessary factors. Malaria is apparently closely associated with the development of verruga: a particularly pernicious type, locally known as "Oroya Fever," being commonly observed with it. All ages and both sexes are equally liable. Natives of the verruga zone seem to suffer less severely than strangers coming to the valley. For a time this disease was believed to be a form of yaws, or frambesia, and, like it, was interpreted as a form of syphilis. Yaws, however, is not observed in the internal organs. Furthermore, verruga is observed in the domestic animals, including fowls, an observation contrary to any known manifestation of syphilis.

Symptoms.—The incubation period is given as ten days to a year. Fifteen to forty days (according to Odriozola) seems a more reasonable figure. In the inoculation case of Carrion the incubation was twenty-three days. Clinically, two stages present themselves, the stage of invasion and the stage of eruption. The stage of invasion begins with prodromal symptoms. *Lassitude, restlessness,* and *weariness* of the legs, lasting for a few days, are followed by an *evening fever.*

The fever gradually increases in severity, with *marked rigors,* and may be remittent or intermittent. In a few days *joint pains* develop. The joints invaded are the smaller articulations of the hands and feet, the knees, and the spine. The pain is severe, is worse at night, and is fugitive, passing rapidly from one joint to another. *Painful contractions* of particular muscle groups occur, most frequently in the calf-muscles and sternomastoids. Sometimes large muscle groups are affected, so that in extreme cases opisthotonos may develop. As the disease progresses, *anæmia* and *emaciation* occur. The *skin* becomes *pale* and *icteric;* the liver and spleen become enlarged. Soft bruits are heard over the præcordium. The fever persists from three to five weeks, when it gradually declines, and, with its disappearance, begins the stage of eruption. The *eruption* usually develops after twenty days, or it may be delayed as long as six or eight weeks. In rare instances it is observed at the very beginning of the disease.

With the breaking out of the eruption, all the general symptoms are remarkably ameliorated. Beginning first as *small, pinkish papules,* the lesions become *dark blue* in color, and finally develop into *warty excrescences.* They appear on the face, particularly around the eyelids and nose, on the limbs, about the joints, and rarely on the trunk. The palms, soles, and hairy parts of the body are also attacked. In size the lesions vary from a millet-seed to growths as large as an apple. They may be few or many hundreds in number. These warty growths are exceedingly vascular and bleed freely, thus increasing the anæmia of the patient. When they develop on the mucous membranes and internal organs, dysphagia becomes a very common symptom, and hemorrhages occur from the various organs that are the seat of the lesions; hæmatemesis, hæmoptysis, hæmaturia, metrorrhagia, etc. After persisting from four to six months, perhaps passing through various recrudescences, the lesions subside by involution and desiccation or desquamation, or they may ulcerate, or the larger lesions may suppurate.

Prognosis.—The prognosis is always grave, particularly so in white people, in whom 60 to 70 per cent. of all cases die. In natives the mortality is about 10 to 15 per cent. The early and complete establishment of the eruption is a very favorable sign. In delayed or partial eruptions the prognosis is grave. Excessive anæmia is also an unfavorable sign.

Treatment.—Treatment is symptomatic. On account of the very general association of this disease with malaria, quinine should always be freely administered. Sudorifics and hot drinks are usually employed with the idea of hastening or completing the eruption. Descent to lower altitudes not only diminishes the pain and abbreviates the disease, but also lessens the tendency to hemorrhages from the lesions. Odriozola recommends the removal of all ulcerated verrugas.

KUBISAGARI.

This disease occurs endemically in certain districts of northern Japan. It is very closely related to the endemic paralytic vertigo of Switzerland (Gerlier's disease). Kubisagari manifests itself by attacks characterized by *dimness of vision*, *diplopia*, and *ptosis*, associated with marked *weakness* of certain muscle groups.

With respect to the etiology of the disease, very little is known. Like Gerlier's disease, it is observed among people who live under the same roof with their cattle. Miura attributes the disease to the effluvium from the cattle and notes that the endemic section is also particularly badly infected with cattle plague.

Kubisagari is a disease of all ages and both sexes, and occurs principally in the warm months.

The course of the disease is very chronic. The attacks come on at intervals of a few hours to several days and last from a few minutes to two to three hours. They are brought on by hunger, indigestion, muscular fatigue, and eye-strain. Ptosis and diplopia are constant symptoms. Paresis occurs in various muscle groups, most commonly in the posterior muscles of the neck, the head dropping forward in consequence (hence the name "kubisagari," *i. e.*, one who hangs his head). Paresis of the muscles of mastication, deglutition, and locomotion may render chewing, swallowing, or walking difficult or impossible. Between attacks these symptoms all disappear, excepting the ptosis and head-hanging, which may be permanent. The superficial and deep reflexes are increased. The eye-grounds show congestion of the optic disk.

The prognosis is favorable. Kubisagari never tends of itself to a fatal issue.

Treatment.—Bromide of potassium has some effect in controlling the frequency of the attack. Miura reports the favorable action of the iodides and arsenic in some cases.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

ACUTE CORYZA.

Definition.—Acute coryza is an inflammation of the nasal mucous membrane, characterized in its early stages by hyperæmia, redness, and swelling, and followed by free secretion of mucus and serum.

Etiology.—Without any doubt acute coryza is an infectious malady, although it usually follows exposure to cold or wet. The exposure produces a condition favorable to the growth of the micro-organisms which cause the disease. No single organism has been isolated, and in some cases several are probably active at once. Coryza is a conspicuous symptom of certain forms of influenza and may be produced by a number of micro-organisms, among which the pneumococcus should be mentioned. Hajek claims to have isolated an organism called the *Diplococcus coryzæ,* which he believes is responsible for the malady.

The disease can be transmitted from one person to another, probably by droplets of infected discharge, the susceptibility of an individual depending upon both a local and general lowering of vital resistance. The breathing of vitiated air, as in badly ventilated theatres and steam cars, and of dust-laden atmospheres, as in certain industries, is a frequent predisposing cause. The possibility of a diphtheritic origin in certain cases should not be overlooked. Damp cold, even if of moderate degree, is more provocative of the disease than dry cold.

Pathology and Morbid Anatomy.—The pathology of acute coryza is that of an ordinary catarrhal inflammatory process affecting a mucous membrane. The bloodvessels of the submucosa become hyperæmic, congested, and engorged, and from them an extravasation of white blood cells and red corpuscles takes place, accompanied by a transudation of serum, which increases the swelling, and finally escapes upon the surface of the mucous membrane, to be thrown off with the desquamated epithelium. The mucous glands secrete an excess of mucus laden with dead epithelial cells and leukocytes or pus corpuscles. As recovery takes place, the inflammatory exudate in the submucosa is absorbed, the dead epithelial cells are replaced by young cells, and in this manner the process of repair is completed.

Symptoms.—The symptoms of acute coryza consist in primary *chilliness* and some restlessness and in a sensation of *dryness of the nasal mucous mem-*

brane of the part affected. This is accompanied by a loss of the sense of smell and by a *dull frontal headache*, probably due to congestion in the frontal sinus. There is frequently severe sneezing, due to the irritation of the nasal nerves by the inflammation and to the trickling of the serum over the angry mucous membrane. The voice sounds as if the nose was "stopped up." As the disease progresses, large amounts of mucopurulent material are discharged from the nostrils and find their way back into the nasopharynx. The constitutional symptoms are often quite severe, and consist in *chilliness* and flushes of heat, followed by relaxation of the capillaries of the skin and more or less perspiration. *Aching in the head*, in the *muscles*, and the *small of the back* are prominent symptoms, indicating that the local nasal process is not the only part disordered, but that other parts are indirectly affected. Without doubt, the two chief causes of these symptoms are the loss of large amounts of liquids by the nose, something like 1½ to 2 pints a day in some cases, and the absorption of toxic materials due to the infection.

Diagnosis.—The acute rhinitis due to an oncoming attack of measles, or that due to an attack of hay fever, are the two states that most closely resemble true coryza. In infants the possibility of the attack being due to syphilis, "syphilitic snuffles," must be considered.

Treatment.—The treatment of acute coryza consists, if the patient is seen in the stage of onset, of the use of a saline purgative to deplete the system and unload the bowels, and in the internal use of full doses, 20 to 30 grains every hour for five doses, of bicarbonate of sodium in water. At the same time the well-known combination called "rhinitis tablets" may be given. These consist of—

℞—Quininæ sulph.	gr j.
Camphoræ	gr. $\frac{1}{8}$.
Ext. belladonnæ	gr $\frac{1}{16}$.—M

Sig.—One or two every thirty minutes till six are taken.

The nasal mucous membrane should be washed by a gentle spray of normal salt solution, followed by a spray of—

℞—Cocainæ hydrochloratis	gr v
Chloretone	gr. ij.
Aquæ destillat.	q. s. ad f℥j.—M

Sig.—Apply as a spray.

Followed by a spray of—

℞—Antipyrini	gr. xv.
Cocainæ hydrochloratis	gr. j.
Aquæ camphoræ	fʒiij.
Aquæ destillat.	q. s. ad f℥j.—M.

This, in turn, should be followed by a spray of menthol in the proportion of 6 grains to the ounce of liquid albolene.

In many instances a hot foot-bath and a dose of 5 to 10 grains of Dover's powder may be used to abort an attack.

After the disease is well on its way, it is bound to run its course. We can only give relief by using a type of nasal treatment like that just suggested and in cleansing the nasal chambers of mucus.

When the attack has run its course the consequent debility is best controlled by the use of fresh air, arsenic, ammonium benzoate, and bitter tonics.

CHRONIC NASAL CATARRH.

Definition.—Chronic nasal catarrh, as its name implies, is a chronic inflammatory state of the nasal mucous membranes, frequently due to repeated attacks of the acute variety, or occasionally coming on more insidiously. When it is well developed the tissues of the nasal chambers are relaxed and somewhat œdematous, the secretion is abnormal in character and in quantity, and this pathological condition is often subject to acute exacerbations due to exposure to the usual causes of coryza.

Etiology.—The causes of chronic nasal catarrh are, as just stated, repeated attacks of acute coryza and continued exposure to the action of irritating dust or of cold, moist air, laden with infectious materials. The condition may be of syphilitic origin or arise from depleted vitality from constitutional disorders, such as Bright's disease. Foreign bodies should be searched for, particularly if the patient is a child, and nasal growths may be found as a cause, although, as a rule, the catarrh causes the formation of growths.

Pathology.—An examination of the nasal mucous membrane in cases of this disease shows that the bloodvessels are distended and have lost their normal elasticity. They are unable to drive out any additional blood which may be sent to them because of the other parts being chilled. Exudation into the connective tissues takes place,and so this structure becomes thickened and enlarged. At this stage the condition is sometimes called hypertrophic rhinitis.

Repeated or protracted irritation induces hyperplasia of the connective tissue of the submucosa, continued epithelial exfoliation, glandular atrophy, and sclerotic changes in all the layers of the mucous membrane. In the earlier stages these changes are those already mentioned when discussing acute coryza; later cell proliferation and leukocytic accumulation in the nasopharyngeal submucosa greatly thicken the membrane, particularly over the turbinates and the septum (*hypertrophic rhinitis*), while organization (*fibroid change*) increases the fibrous tissue in the areas involved, followed by contraction with atrophy of erectile, glandular, and even nerve tissues (*atrophic rhinitis*). This lessens secretion, which tends to inspissate, form scabs, and decompose, causing the fetid emanations to which the name "ozæna" has been given.

Extension to one or more of the facial sinuses, necrosis of bone, or involvement of the Eustachian orifice or tube are possibilities constantly to be remembered.

Symptoms.—The symptoms of this stage of the disease consist in a constant secretion in *excess of nasal mucus*, which passes in large part into the postnasal and nasopharyngeal spaces. This secretion may be thin and liquid or thick and mucopurulent, and is apt to vary in quantity with exposure to cold or dust. The secretion is so thick that it readily becomes inspissated and partly blocks the nasal passages, and, furthermore, becomes loaded with bacteria, so that it may be somewhat fetid.

Treatment.—The treatment consists in maintaining nasal cleanliness by an ordinary nasal douche-cup, to be used night and morning, employing in it normal salt solution or Dobell's solution warmed to the temperature of the body. The physician should also cleanse the parts, when the patient visits him, by a mild alkaline wash, and when no acute exacerbation is present the hypertrophied mucous membrane over the middle turbinate should be cocainized and then lightly touched with a small electrocautery, a piece of oiled cotton being placed between the spot cauterized and the nasal septum, to prevent adhesions from forming during the period of acute swelling which follows the operation. Care should be taken to keep the parts clean for several days to prevent infection, if the patient is primarily anæmic or debilitated. This treatment should not be resorted to before the general health is improved by tonics.

ATROPHIC NASAL CATARRH.

Definition.—In this condition the nasal mucous membrane and the underlying tissue undergo atrophy and contraction, with the result that the blood-vessels of the part are occluded or destroyed.

Etiology.—Atrophic nasal catarrh follows the chronic type of ordinary nasal catarrh as a late condition. At times it seems to be due to some congenital defect in the shape of the nasal chambers, and in some cases it begins to develop as the result of one of the acute infectious diseases.

Pathology.—The chief change is an atrophy of the cells of the nasal mucous membrane and an overgrowth of the submucous connective tissue, which is prone to undergo contractile changes. This cuts off blood supply and increases the atrophic process.

Symptoms.—These consist in the formation of scabs, or crusts of thickened, tenacious mucus, which are usually infected by many pathogenic germs. Some ulceration of the nasal septum may appear, and the patient may complain of a constant feeling of *dryness* and *irritation*, or *occlusion* of the nasal passages.

The nasopharyngeal mucous membrane is often dry and shiny in appearance. When the condition is far advanced a state of *fetid ozæna* develops, in which the breath of the patient becomes fetid beyond the power of words to describe it. Nothing equals it, except the breath in a case of pulmonary gangrene.

The *sense of smell* is practically *destroyed* by the process, and the patient is often ignorant of how disagreeable his breath has become.

Prognosis.—The outlook for a cure of this condition is unfavorable. The bad odor can usually be relieved.

Treatment.—The patient should be told to use a nasal douche-cup with warm Dobell's solution twice a day. Kyle recommends 1 drop of ordinary coal oil dropped into each nostril after this. The physician should see the patient every few days, cleanse the nasal chambers, and apply a 1:2000 solution of trichloracetic acid on an applicator to the nasal passages, or 1:500 solution of formaldehyde may be used.

HAY FEVER.

Definition.—Hay fever is an inflammation of the nasal mucous membrane which occurs periodically and usually at a time of the year when certain plants are in a certain stage of growth. Associated with the localized inflammation, there is often present an asthmatic condition, in which a sense of oppression is well developed. In other instances true asthmatic attacks ensue. Hay fever is often called " autumnal catarrh," " rose cold," " ragweed fever," or " periodic rhinitis."

Distribution.—The prevalence of hay fever depends largely upon the presence in the air of the pollen of certain plants, and in those parts of the country where these plants do not grow the disease is unknown. It is more rare in England than in America, but much more common in these countries than elsewhere, being comparatively rare in France and Germany. Negroes and Indians are apparently immune, and the lower classes very frequently escape, the disease being chiefly a malady of the so-called upper classes. The disease is rare after the fortieth year and affects males oftener than it affects females.

Etiology.—There are two chief factors in the development of hay fever, namely, an idiosyncratic state of the patient and the presence of an exciting cause in the atmosphere. Much discussion has taken place as to what the condition is that renders the patient peculiarly liable to this affection. In some cases it seems to be a neurosis of the nasal cavities or a local disease; in others it is said to depend upon a "lithæmic state," whatever that may be. In every case, however, there is a condition of nasal hyperæsthesia which renders the nasal mucous membrane extremely sensitive to irritants. Many sufferers from hay fever present irregularities in the nasal chambers which may aid in predisposing them to attacks of the malady.

The second cause of hay fever in the great majority of cases is the presence in the air of pollen from some plant, chiefly "ragweed." Pollen is not the only cause, however, for typical attacks occur in certain persons at seasons of the year when no pollen is present, the condition being induced by some irritating dust or vapor which in no way influences the ordinary individual.

Pathology and Morbid Anatomy.—In the state of the nasal mucous membrane there is nothing peculiar to hay fever, which presents on examination the evidences of an acute catarrhal inflammation with swelling and hyperæmia of the parts involved.

Symptoms.—The symptoms of "hay cold" are usually *sudden in onset* and often appear on a certain day which the patient can foretell, and nearly always at a definite period in the year, for the reason already given. An acute rhinitis develops with *irritation* of the *nasal mucous membrane*, and the running of salty fluid from the nostrils irritates the nares and the upper lip. The conjunctival mucous membrane is irritated and inflamed, and the eyes are tearful, partly because of this condition and partly because the tear ducts to the nose are stopped by the swelling of the mucous membrane. *Photophobia* and *neuralgic pains* in the head are often present and add greatly to the patient's misery. *Frontal headache* is constant and severe, and *tinnitus* and *fulness of the head* are also annoying symptoms. Some *deafness* may be present. Associated with these symptoms the patient often has marked *systemic depression* and *wretchedness* with *great mental depression.*

On examining the mucous membrane of the nose there is found undue pallor in long-standing cases which is not to be expected when inflammation is present. If a probe is touched to the mucous membrane patches of hyperæsthesia are discovered, as evidenced by sudden severe sneezing and other signs of acute irritation.

The attack is prone to persist as long as the patient remains exposed to the cause, and upon his removal from exposure may cease almost as speedily as it came on. In cases in which a reflex asthma due to the nasal irritation ensues the patient may become a chronic asthmatic even if the hay cold disappears.

Prognosis.—The outlook for cure in the affection is not good unless the patient can go away to a resort where the cause does not exist for a certain length of time each year. So far as life is concerned it never endangers it, but if the disease produces vital depression it undoubtedly increases the susceptibility to other diseases. The patient can be comforted by the statement that the attacks stop or diminish in many cases after forty years of age.

Treatment.—A competent rhinologist should always be asked to correct all nasal irregularities during the period of quiescence, and the physician should correct any gouty or lithæmic state by the use of ordinary exercise, a good diet, and the use of the salicylates, or bitter tonics with arsenic to improve the state of the mucous membranes in general. Not infrequently good results follow the use of 30 grains of phosphate of sodium in a cup of hot water before breakfast, given to stimulate the gastroduodenohepatic glands. In other cases salicylate of sodium in 10 grain doses or salol may be used for this purpose. These measures and the resort to a region free of the exciting cause in the autumn months are the prophylactic measures. A sea voyage usually confers complete immunity if taken at the proper time of year, and sometimes residence at some mountain resort does likewise, particularly if the altitude is very great.

In the way of local treatment for the attack the swollen mucous membranes may be constricted by the application of a solution of adrenalin chloride 1:5000, and after this is done the parts may be washed with a mild alkaline spray like Dobell's solution or normal salt solution which has been warmed. After this is done the cocaine solution and the antipyrin solution recommended for acute coryza may be employed and finally the parts coated by the use of the spray of menthol and camphor named in that article.

Within the last few years several attempts to produce immunity to hay fever by the use of preparations of golden rod and ragweed have been attempted, the patients taking them for some time before the time of an attack in the hope that they would not suffer from the disease. This plan has not so far proved very successful.

Still more recently Dunbar, of Hamburg, claims to have isolated a toxin from the pollen of certain plants, and by giving it to horses produced an antitoxic serum which, he states, will protect a susceptible person. This is not used hypodermically, but is dried, mixed with sugar of milk, and then finely triturated. A small part of this powder is to be snuffed up the nose. When it is desired to use the remedy in the eyes the fluid serum is employed. The value of this method is still undecided.

EPISTAXIS.

Etiology.—Nose-bleed is due to many different causes, chiefly traumatic. The condition only concerns us, from the medical standpoint, when it develops as a result of lesions in the nasal cavities or in the course of the infectious diseases, or in cases of heart disease in which there is cephalic congestion. Occasionally it occurs in very plethoric persons after severe exercise, and in them it may be a beneficial condition.

Severe nasal hemorrhage usually arises from an ulcer on the nasal septum, from cardiac disease, the commonest lesion being mitral disease, and in typhoid fever as one of the prodromes. Occasionally it is a desperately persistent state in hæmophilia, and even more rarely it seems to be of the nature of vicarious menstruation. Sometimes it is a manifestation of blood dyscrasia, as in leukæmia.

Treatment.—The treatment consists in plugging the nostrils with cotton, if necessary saturating the cotton with adrenalin chloride 1:2000, and in compressing the artery on the upper lip near the nose by pushing it against the jaw-bone. Internal measures are usually unnecessary and useless.

DISEASES OF THE LARYNX.

ACUTE CATARRHAL LARYNGITIS.

Definition.—Acute laryngitis, or acute catarrh of the larynx, is an inflammation of the mucous membrane lining the larynx, as a result of which there is more or less loss of voice, or aphonia, and perhaps a sense of constriction or respiratory oppression.

Etiology.—This condition arises as a result of any factor which directly causes irritation of the laryngeal mucous membrane, such as the inhalation of irritant vapors or dust. In some cases the inhalation of cool and damp air produces like effects, particularly if the voice has been used much before the exposure. Indirectly it arises as the result of getting the body chilled in a cold wind after exercise, and in still other cases it seems to be, at least in part, due to some disorder of metabolism, whereby gouty conditions ensue, and these in turn cause laryngeal inflammation by some indirect effect when the voice is much used. Another cause, particularly in the case of children, is "mouth-breathing," which permits the air unmoistened by the nasal chambers to pass over the laryngeal surface. In still other instances it arises as a complication of one of the acute infectious diseases, as influenza and of hay fever. The possible diphtheritic origin of acute laryngitis, especially in children, should never be overlooked.

Pathology.—The inflammation of the laryngeal mucous membrane is precisely like that of mucous membranes elsewhere, except for the fact that glandular tissue is quite scarce in these parts, and so there is but little mucus secreted even if a considerable amount of inflammatory exudate takes place in the tissues beneath the mucous membrane. The desquamation of epithelial cells and the presence of dead leukocytes cause the secretion to be white and tenacious, and the congestion of the bloodvessels gives rise to a sense of tightness in the laryngeal box which is distressing. As the congestion decreases the process of regeneration in the epithelial cells and submucous tissues takes place, secretion becomes more profuse, and perfect recovery ensues.

Symptoms.—The patient finds it difficult to develop the full resonance of his voice and often single words in a sentence, or all the words, are spoken somewhat huskily owing to failure to move the vocal bands as readily as in health. There is a sense of tightness in the larynx and even aching pain may be present. In some cases hoarseness is the only symptom, but in others the loss of voice is complete. Speech at this time is often so painful that the patient endeavors to avoid conversation.

An examination of the laryngeal mucous membrane at this time will reveal marked redness and hyperæmia and even small punctiform hemorrhages may be seen, particularly if the patient has repeatedly and violently endeavored to clear his throat by hawking or coughing. The ventricular bands are swollen, and this may be the chief cause of the loss of voice, for it often happens that the vocal cords escape the inflammatory process. In other cases the edges of the glottic opening, the epiglottis, and the mucous membrane over the arytenoids are inflamed.

As the process proceeds secretion is begun and small particles of mucus are occasionally coughed up. This is particularly apt to occur after severe coughing in the morning in order to dislodge inspissated mucus. The masses expectorated are often distinctly purulent, and discolored with soot if the patient lives in a city. Secretion gradually becomes more profuse. Pain disappears and the voice, which has been whispering, becomes hoarse and coarse in character, certain words which require effort being sounded with difficulty. Finally the voice recovers its normal tone and the attack is over.

Diagnosis.—Care should be taken that sudden attacks of hoarseness are not considered as due to simple catarrhal laryngitis until the possibility of diphtheria is excluded by a thorough examination of the throat and larynx. This is particularly important in children. In adults the possibility of aneurysm, tuberculosis, papilloma, and syphilis should not be forgotten.

Prognosis.—The prognosis is always good as to recovery if the exciting cause is removed.

Treatment.—The treatment of acute laryngitis consists primarily in removing the cause. If irritant dusts are present the patient must not be exposed to them, and if the outside atmosphere is raw and cold he must be kept in-doors until recovery takes place. When a gouty diathesis underlies the condition the salicylates should be freely used in the form of salicin, 5 grains three or four times a day, and the vegetable salts of potassium, such as the citrate, be given freely. About the neck may be fastened a capsicum draft, or, if this is not to be had, a folded handkerchief should be wrung in cold water and laid upon the larynx, being immediately covered by a cloth or piece of flannel, which is bound around the neck. The cold compress is promptly changed to a warm compress by the heat of the body, and this acts favorably upon the local inflammation beneath. The air of the room in which the patient is to sleep or rest during the day should be kept well moistened by steam disengaged by a bronchitis kettle or by adding pieces of unslaked lime to a tub of water. Into the water in the bronchitis kettle may be placed a few grains of menthol if it is desirable to exercise a very sedative effect, and the patient should be forbidden to go into any cold rooms or hallways. A hot mustard foot-bath and a hot lemonade with a drachm of sweet spirit of nitre at bedtime to produce sweating is also useful, or a dose of Dover's powder may be ordered if the patient is an adult. Kyle recommends the use of tablets of $\frac{1}{100}$ grain of pilocarpine every hour for four doses, or if it is important to attempt to abort the disease he suggests the use of 5 to 10 drops of dilute nitric acid in water every hour for three doses, and then every two hours for two doses. This often gives temporary relief if it does no permanent good.

If by any chance the inflammation involves the glottis and seems to endanger life, intubation or tracheotomy is necessary.

For the hoarseness and thick secretion of the stage of convalescence benzoate of soda or of ammonia, in 10 grain doses three times a day, are useful, or 10 grains of ammonium chloride may be given thrice a day in licorice and water. In still other cases terpin hydrate in the dose of a teaspoonful of the elixir every three hours may be used.

CHRONIC CATARRHAL LARYNGITIS.

Symptoms.—Chronic catarrh of the larynx is characterized by chronic hoarseness, by constant clearing of the throat in an endeavor to speak clearly, and finally, in severe cases, by ulceration of the laryngeal mucous membrane. Not rarely after a period of rest the patient finds that after the first few words his voice forsakes him, or, instead, he may find that if speaking is difficult at the beginning it becomes more easy as exercise limbers up the infiltrated muscles and engorged mucous membrane.

Pathology.—Pathologically the condition is characterized by chronic engorgement of the minute bloodvessels, thickening of the mucous membrane, and even infiltration of the submucous tissues and the laryngeal muscles. If this process persists for any length of time sufficient infiltration may be present to become unabsorbable and thus cause permanent alteration in the character of the voice. Not rarely there is thickening and swelling of the pharyngeal tissues as well, and the tonsils are the seat of chronic lymphoid changes. Spots of ulceration may develop between the arytenoid cartilages.

Diagnosis.—As stated in the article on Tuberculosis, hoarseness which is persistent should always be carefully investigated, as it often is due to tuberculosis or syphilis, or even papilloma. When it is due to aneurysm the laryngoscope will usually reveal one cord paralyzed, and when due to syphilis the history of the patient and the benefit produced by specific treatment must be noted. In persons of advanced years the possibility of malignant growth must be considered.

Treatment.—The treatment consists in the maintenance of cleanliness and free secretion in the upper respiratory tract by the use of alkaline sprays and nasal douches. To the larynx itself a spray of alumnol (3 per cent. solution) may be applied every second day. The use of tobacco and alcohol should be forbidden and the liver and kidneys kept active by mild alkaline purges and diuretics. Tonics to the general system, such as phosphorus, arsenic, and sometimes iron, particularly the syrup of the iodide are useful.

ŒDEMATOUS LARYNGITIS.

Definition.—Œdema of the larynx occurs as an acute affection, occasionally of such a severe degree that it endangers life. It is essentially an acute cellulitis of the laryngeal tissues, and when it involves the upper part of the larynx in particular it is called œdema of the glottis.

Etiology.—Œdema of the larynx is far more frequently due to injury than to any other cause, and in many instances is produced by the inhalation of irritant vapors or fumes. The only cases I have seen have been due to the patient attempting to swallow ammonia water undiluted, which both by actual contact of the fluid with the pharynx and of the fumes with the larynx has caused serious respiratory distress. Another traumatic cause is fracture of the larynx, as by throttling or other injury. Of the non-traumatic causes we find that acute inflammations in other parts may be provocative of this state, as, for example, tonsillar abscess with inflammation of the adjacent tissue gradually extending to the larynx. Sometimes it ensues as a result of grave lesions in the cartilages of the larynx, as in the chondritis arising in typhoid fever, or even in scarlet fever in association with the development of the "collar of brawn." Infection by the staphylococcus or streptococcus of the perilaryngeal tissues, the floor of the mouth or pharynx (as in Ludwig's angina) may extend to the epiglottis and larynx. While often an infection, it may arise in the course of some affection characterized by widespread œdema, as Bright's disease or chronic heart disease, and so appear to have a dropsical origin.

Pathology.—The condition of œdema of the larynx, as its name implies, depends upon the extravasation of fluid into the submucous tissues, producing an œdema or hydrops of the part, which is practically always of an inflammatory origin. This swelling may involve all the laryngeal structures equally and even extend well along the trachea. In mild cases the parts affected are not, however, seriously disorganized, and the swelling may disappear as rapidly as it came on, leaving behind it little trace of its existence. In fatal cases the swelling is usually found at autopsy to have largely disappeared, although the parts may be red and inflamed and somewhat relaxed. In such cases the microscope commonly shows a serous, or serofibrinous, suffusion of the affected tissues, the exudate containing a varying number of leukocytes. In other cases the submucous and even the perilaryngeal tissues may be infiltrated by pus, diffuse suppurative interstitial laryngitis, a most fatal disease.

Symptoms.—With the onset of this condition several characteristic symptoms at once develop, namely, *impairment of the voice*, *stridulous* or *labored breathing*, manifestly due to laryngeal obstruction, and increasing *cyanosis*. The patient is uncomfortable if lying down, and is more easy when sitting up and leaning forward. The tissues adjacent to the larynx may be swollen and the patient, if unable to speak, points appealingly to his larynx.

In some instances a state of chronic œdema, due to heart or renal disease, may ensue, which is rarely so severe or as pressing for relief as is the acute malady.

Diagnosis.—Difficult laryngeal breathing may be due to a laryngeal crisis in locomotor ataxia or to the lodgement of a foreign body in the larynx. These conditions should be excluded before the physician decides that œdema is the cause of the illness. Aneurysm of the aorta may produce severe laryngeal symptoms by pressure on the laryngeal nerves, and in children retropharyngeal abscess may, by rupture or pressure, produce somewhat similar symptoms.

Prognosis.—In the absence of virulent symptoms the prognosis is usually favorable even if the symptoms are severe, provided that when they appear the physician is ready to give relief by intubation or tracheotomy. If the œdema is due to the inhalation of irritating vapors and the lower respiratory tract is involved in the inflammatory process, the prognosis is, of course, very grave.

Treatment.—The treatment consists in the administration of an active saline purge to deplete the vascular system, and the setting free of steam from a bronchitis kettle in the air of the room in which the patient lives. To the water in the kettle may be added a few grains of menthol for its soothing influence on the laryngeal mucous membrane. A 10 per cent. solution of alumnol may be sprayed into the larynx for its astringent effect. When the œdema arises as a complication of renal or cardiac disease and is part of a general tendency to anasarca, so active a purge as elaterium or colocynth is indicated to remove fluid from the body, and a hot pack may be used to cause sweating if the heart is strong enough to stand it. The use of pilocarpine for this purpose is unwise, because it is so prone to cause pulmonary œdema. When the laryngeal obstruction becomes marked it may be necessary for the physician to quickly perform intubation or tracheotomy, but while he should be prepared to do so at any moment it is scarcely necessary to add that tracheotomy should only be done as a last resort.

SPASMODIC LARYNGITIS.

Definition.—Spasmodic laryngitis, sometimes called "spasmodic croup," or "false croup" in distinction from diphtheria or true croup, is a condition of acute laryngeal catarrh involving the mucous membrane in the region of the glottis, and resulting in swelling of those parts, so that the ingress and egress of air is difficult. The spasm of the laryngeal muscles, while it aids in producing the symptoms, is really of secondary importance as compared to this swelling.

Etiology.—In the past it was customary to consider spasmodic croup a disease in itself. We now know that it is a symptom depending upon several causes, some of which are external, some internal. Appearing as it does almost always in children between one and six years, but sometimes persisting in its occurrence up to puberty, it depends chiefly upon rickets or malnutrition, the presence of postnasal adenoids, which make the child a mouth breather, or to some defect in the nose, which causes the same condition. In some instances errors in diet before retiring to bed seem to precipitate an attack. Of the external causes which, however, are only active in those who have a tendency to attacks of this affection, may be mentioned furnace-heated air, which is so dry and dusty that if the child is a mouth breather the larynx becomes rapidly dry and irritated. An acute coryza may also bring on an attack in that it causes mouth breathing.

Treatment.—The treatment is evident from what has just been said. It may be divided into two parts, that for the relief of the attack and that for the cure of the underlying causes.

When any sign of croup is manifested the nurse should place 1 or 2 grains of menthol in the bowl of a dry spoon and heat it over a gas jet or lamp. This sets free in the air of the room the menthol vapor and soothes the laryngeal mucous membrane. If the attack is well developed the nurse should, in addition, disengage from a bronchitis kettle steam laden with menthol, or with oil of pine and oil of eucalyptus, in the proportion of 15 drops of each in the water in the kettle or on the sponge usually placed in its neck or spout. Internally a dose of 5 to 10 grains of bromide of sodium may be given in syrup, and over the larynx should be placed a compress wrung in hot or cold water.

For the prevention of future attacks the child should be relieved of adenoids or enlarged tonsils, should receive proper diet and tonics if rachitic, and should sleep in a bronchitis tent if the larynx is irritable. A sponge loaded with cold water may be sopped upon the skin over the larynx every morning to improve its vascular tone.

TUBERCULOUS LARYNGITIS.

Definition.—As its name implies, tuberculous laryngitis is due to the presence of the *Bacillus tuberculosis* in the laryngeal tissues and the consequent development therein of miliary or larger tubercles. (See Tuberculosis.)

Etiology.—In the vast majority of cases of laryngeal tuberculosis the infection is secondary to pulmonary disease, and is due to infection of the larynx by the sputum which the patient coughs up, or by direct extension from below upward. In rare instances it is undoubtedly a primary affection. I have had such a case under my care while writing this article. As in tuberculous infection in other parts there must be, in addition to the presence of the bacillus, a susceptibility to infection, or one acquired by general or local lowering of vitality. This disease occurs most commonly in males between twenty and thirty years of age.

Pathology.—Here, as elsewhere in the body, the development of tubercles takes place by leukocytic migration and the proliferation of cells, and is accompanied by the closing of bloodvessels and the necrosis of the masses formed, followed by breaking down and escape of the cheesy material, and the production of ulcers in the laryngeal mucous membrane. As the process of infiltration proceeds the perichondrium is attacked and necrosis takes place in the laryngeal cartilages.

In many other parts of the body the system makes efforts at repair, so that, even if the disease ultimately wins the battle, evidences of an active defence can be recognized; but in the larynx it very commonly happens that no such reparative or protective process occurs, and this is one of the reasons why the malady is so rarely cured. In some cases the infectious process takes on some of the aspects of a tumor, a tuberculoma, manifesting a tendency to infiltration and induration, with but little inclination to ulceration.

Symptoms.—There are few maladies which present such a distressing picture of suffering as does laryngeal tuberculosis. The *loss of voice*, which permits speech only with great effort, cuts off the patient from pleasant intercourse with friends and from expressing any but his most urgent needs, and then only with great pain and effort. The thickening of adjacent tissues nearly always makes *swallowing* most difficult and painful, and for this reason urgent thirst must be satisfied with but one swallow of water. The same *dysphagia* makes the use of solid food impossible and the taking of liquid nourishment almost so, yet the patient cannot combat the malady unless well nourished. Because of these factors and the constant pain and loss of sleep, the loss of weight in laryngeal tuberculosis is often extraordinary, almost equalling that seen in some cases of malignant growth elsewhere.

In the earlier stages the loss of voice and constant discomfort in the larynx may be the only symptoms. *Cough* is present in all cases to some extent, and is often exceedingly painful. After all the muscles about these parts have had their action inco-ordinated by direct infiltration or disordered nerve supply, particles of food get into the larynx and cause spasm and pain which is insufferable.

A careful examination of the chest wall will reveal in most cases some tuberculous focus.

If the laryngoscope is used in the early stages, an acute hyperæmia may be found, but in the chronic type the appearance will be that of marked local anæmia. A tuberculous lesion may be found in the epiglottis, from whence it gradually passes downward, or the disease may begin below the cords and work its way upward. These tuberculous areas are composed of small nodules or swellings which are hyperæmic in the acute cases and anæmic in the chronic cases. As these nodules grow they may, by their mere mechanical presence, cause obstruction to free respiration. The infiltration of the epiglottis becomes marked, and the mucous membrane may become dotted by a multitude of small, yellow tubercles which are easily recognized. These break down and, having done so, form small ulcers which coalesce and form larger areas of ulceration. Comparatively rarely the vocal cords develop tiny vegetations.

Diagnosis.—The discovery of a pulmonary tuberculous process in the presence of the hoarseness, which is persistent and does not clear up under the ordinary treatment for acute or chronic laryngitis, raises a suspicion of the tuberculous character of this malady at once. In syphilitic laryngitis the history of the patient, the reddened areola about the ulcers, and the presence of signs of syphilis elsewhere will aid the diagnosis. From carcinoma of the larynx we can separate tuberculous laryngitis by the fact that the former disease occurs as a single new-growth in a person who is usually past the period of life in which tuberculosis is prevalent.

Prognosis.—The prognosis is most unfavorable even in cases seen in the early stages, because the ability to carry on repair in these tissues is so poor and because experience has taught us that these cases rarely recover.

Treatment.—The treatment of laryngeal tuberculosis can be carried out only by a skilled and dexterous laryngologist. Even in his hands it may cause much distress and pain. In the hands of the tyro clumsy handling is probably worse than useless. For the palliation of the condition and of the suffering the parts may be sprayed with peroxide of hydrogen followed by a mild alkaline solution, and these in turn by a spray containing menthol 5 grains, oil of sandal-wood 5 minims, and liquid albolene 1 ounce. General tonic treatment such as is used in all cases of tuberculosis, with careful feeding, is essential. As a rule, the patient should avoid high altitudes, particularly if they are windy, as the drying of the mucous membrane increases discomfort in the larynx.

SYPHILITIC LARYNGITIS.

Etiology.—Syphilis of the larynx appears during the secondary and tertiary stages of the disease. In the secondary stage it may amount to nothing more severe than hyperæmia or erythema, such as is usually met with in ordinary acute catarrhal laryngitis, but in other instances mucous patches develop, which are most numerous about the aryepiglottic folds, the region of the vocal cords, the arytenoid cartilages, and on the edges of the epiglottis.

If active treatment is instituted they usually readily yield, but they may become distinct ulcers. When these heal there may be sufficient thickening of the parts to cause permanent hoarseness.

Tertiary syphilis appears in the larynx as a diffuse or circumscribed gummatous growth, which usually attacks the epiglottis, the cords, and the posterior wall of the larynx, causing thickening and infiltration of the tissues, and finally ulceration of the surface in some instances. The cicatrization of the ulcers, or sclerosis of areas by infiltration, may cause stenosis and distortion of the laryngeal wall.

Symptoms.—The symptoms are *hoarseness* and *loss of voice*, but pain is rarely present.

Diagnosis.—The diagnosis is based on a history of syphilitic infection or by the finding of evidences of syphilis and by the relief which follows specific treatment. Tuberculous ulceration is not so rapid in its development and the patient reacts to tuberculin. Further than this, in syphilis the upper surface and in tuberculosis the lower surface of the epiglottis is usually affected. From malignant growth of the larynx syphilis is separated by the fact that the ulcer is solitary in epithelioma.

Prognosis.—The prognosis is good if treatment is used early, before the stage of ulceration is well developed. After ulcers have become deep and severe they may be healed by treatment, but cicatricial contractions necessarily appear as healing goes on.

Treatment.—The treatment consists of iodide of potassium and the mercurials, as in other cases of syphilis. (See Syphilis.)

DISEASES OF THE BRONCHI.

ACUTE CATARRHAL BRONCHITIS.

Definition.—Acute bronchitis is an inflammation of the bronchial tubes which is usually confined almost entirely to the mucous membrane lining them.

History.—Bronchitis has been recognized as a distinct condition for many centuries. It was not, however, until the early part of the nineteenth century that this term was used to describe the condition now under discussion, when Badham, in England, and Franck, in Germany, first employed this term. As with many other diseases involving the thoracic organs, a clear description of the pathological condition was first given to us by the French physician Laennec.

Distribution.—Acute bronchitis is a disease which occurs in all parts of the world, but it affects chiefly the inhabitants of those regions in which the climate is moderately cold and raw, and where the degree of humidity in the atmosphere is high. On the other hand, hot and dry portions of the earth's surface are usually free from this disease. Another important factor in its prevalence is sudden changes of temperature and the prevalence of cold winds laden with moisture. For these reasons the disease is most frequent at those times of the year when sudden changes of temperature are apt to occur, and therefore is commonly met with in the late winter and early spring months.

Etiology.—The etiology of acute bronchitis, so far as external influences are concerned, has just been described. In a goodly number of cases, practically in all, it is probable that micro-organisms have much to do with the development of the disease, and that the primary hyperæmia and congestion of the bronchial mucous membrane is due to the exposure of the surface of the body to external influences and, to a slight degree, to the passage over the bronchial mucous membrane of an atmosphere which, because of its physical condition, is irritating to these parts. It is a well-known clinical fact that exposure of the surface of the body to cold seems to be followed by congestion of the bronchial mucous membrane, and that this actually takes place has been proved, first, by experiments upon animals, and, second, by observations upon man. Thus, it is possible by the external application, alternately, of heat and cold to the upper portions of the thorax to produce great changes in the capillary circulation of the larynx and trachea and probably the bronchial mucous membrane as well.

Much depends, too, upon the general health of the patient who is exposed to the provoking causes which have just been named. Strong, hearty, or robust individuals who have a well-balanced circulation and elastic blood-vessels frequently suffer from no pulmonary inconvenience from exposure, but persons who have been enfeebled by disease, or by advancing years, or those who are very young frequently suffer from such a severe congestion, and it may produce fatal consequences. Bronchitis is also not rarely the result of an inflammatory process which begins higher up in the respiratory tract and extends to the tubes. Practically all acute infections of the lung also produce some bronchial inflammation.

So, too, any condition of cardiac or renal disease which impairs circulatory activity is exceedingly prone to render the patient susceptible to this form of inflammation. We find, therefore, that acute bronchitis is a disease which is most prevalent in infancy and old age, and it is entirely competent, at these two periods of life, to produce death if it is present in a severe form. Those who have been attached to the departments for children in large hospitals cannot fail to have been impressed with the very great frequency of this disease in the winter months, and also with its rarity among the adults who come to the same institution for various ailments.

Both sexes are equally prone to suffer from acute bronchitis, but it is more frequently met with in males because males are more exposed to the provoking causes than females.

Certain of the acute infectious diseases very strongly predispose to this malady. Thus, it is nearly always present in a well-developed form in a case of measles, even when that disease is present in a mild form. Again, there are but few cases of typhoid fever which do not have a certain amount of bronchitis.

The reason that bronchitis so frequently complicates cardiac disease depends upon the intimate relationship between the circulation in the lungs and the right side of the heart, for the bronchial veins open into the venæ azygos and the superior intercostals, and so are intimately connected with the right side of the heart. These bronchial veins also anastomose closely with the pulmonary veins, and so valvular disease which results in congestion of the right side of the heart naturally tends to produce a disturbance of the circulation in the bronchial mucous membrane.

When emphysema of the lungs is present the coincident bronchitis is really due to two causes: first, the congestion of the right side of the heart which is so apt to ensue in emphysema, and, second, the pathological changes in the heart result in impairment of the bronchial circulation. On the other hand, bronchitis sometimes leads to emphysema. These affections are therefore interactive.

There are three other important etiological factors in the production of bronchitis which must also be considered. The first of these is the inhalation of irritant gases or vapors, producing what is known as acute traumatic bronchitis. The second is the inhalation of dust. These dusts may be vegetable, animal, or mineral in their origin. Sometimes all three forms are combined. Finally there can be no doubt that the inhalation of various micro-organisms may result in bronchial infection.

Of the forms of dust which produce bronchitis we find that vegetable dust seems to be the most frequent cause.

That form of bronchitis which most frequently follows the inhalation of irritant vapors or gases is seen in persons who have been exposed to ammonia fumes, irritating smoke, or to chlorine gas. Another form of local irritation producing bronchitis is that which is seen in large cities which are heavily veiled with smoke and fog. Thus, in the city of London the particles of moisture in the air become coated with the sooty materials, and so evaporation, even in houses which are fairly well heated, is greatly impaired, and the fog penetrates in-doors. As a result the frequency of acute bronchitis is greatly increased in London in those seasons of the year in which these fogs are prevalent, and the mortality of chronic bronchitis is wonderfully increased at these times. So powerful an influence do these deleterious factors exercise that it is a well-known fact that the mortality in the city of London may be doubled in those weeks in which the fog is present. Thus, on some occasions the mortality is as high as 46 per cent. as against an ordinary death rate of about 18 per cent. The deaths from diseases of the respiratory organs rose on one occasion from 415 per week to 994 per week during the prevalence of a dense fog. Of these, 694 were due to bronchitis and 185 to pneumonia. That this increase was due to the fog and not to the other conditions of the climate is proved by the fact that a similar increase in mortality did not take place in surrounding provincial cities and towns. These facts are well emphasized by West in his well-known book upon *Diseases of the Organs of Respiration.*

The micro-organisms infecting the bronchial mucosa are the pneumococcus, which is most common; Friedländer's bacillus, the *Streptococcus pyogenes*, and the pyogenic staphylococci. The Klebs-Loeffler bacillus is usually present in the bronchitis which complicates diphtheria. In some cases of bronchitis additional micro-organisms have been found, such as the *Bacillus typhosus*, *Bacillus coli communis*, and various forms of fungi. In most instances, however, bronchitis is polymicrobic in origin, and it is often impossible to decide what organism is the primary infecting agent.

Prevention.—Acute bronchitis can only be prevented by proper care of the general health, by proper clothing, and by the avoidance of climatic influences which are known to be deleterious. Persons who have learned by experience that they are peculiarly susceptible to the various provoking causes named, should, by change in climate or occupation, avoid these various causes of irritation.

Pathology and Morbid Anatomy.—Acute bronchitis is characterized by hyperæmia and swelling of the mucous membrane lining the bronchial tubes, accompanied by some infiltration of the submucous tissues. At first there may be an almost total absence of secretion or undue dryness of the surface involved, but very soon the engorged mucous glands begin to pour out into the lumen of the tubes considerable quantities of mucus, which also soon contains epithelial cells coming from the lining of the glands themselves and from the surface of the mucous membrane as well. Leukocytes, which have undergone diapedesis, as they do in all acute inflammatory processes, are also present, and even red blood cells may be seen. A similar extravasation of red cells may also take place into the submucosa, and, escaping on the surface tinge the sputum. In the smaller bronchi the lining epithelium may be cast off in shreds, and if the inflammation is intense, we may find the tubes almost or completely closed, with resulting capillary bronchitis or suffocative catarrh. By the extension of the inflammation to the peribronchial tissues and the pulmonary alveoli there is developed a bronchopneumonia. (See Bronchopneumonia.) As recovery takes place the dead epithelium and extravasated cells are expelled from the tubes by coughing, and new epithelium is developed from deeper layers of cells. Sometimes, particularly when a large number of infecting micro-organisms are present, the sputum is distinctly purulent. When bronchial inflammation persists for any length of time, or the attacks are frequently recurrent, permanent thickening of the submucosa results. This fibrosis may extend to adjacent structures (peribronchitis) or be continuous with the increased fibrous tissue of chronic interstitial pneumonia.

Symptoms.—The symptoms of acute bronchitis can be divided into three stages, namely, that of onset, the stage of profuse secretion, and the stage of convalescence.

In the stage of onset there may be a *chill*, which usually is not severe; a *short, dry cough* which may, by its persistance, be annoying, and, owing to the dry and inflamed state of the mucous membrane of the bronchial tubes, be distressing because of the *soreness* or *pain* it produces under the sternum. If the degree of swelling of the mucous membrane is marked, there may be a *sense of oppression*, and the breathing may be a little quickened. The *temperature* of the body is usually not much above normal in the adult, but in children it is often as high as 102° or even 103°, and the *pulse* is apt to be rapid in direct proportion to the degree of fever.

Auscultation at this time reveals an increased roughness of the inspiratory or expiratory bronchial sounds, and perhaps a few dry rales between the shoulder-blades.

In the second stage there is a disappearance of the soreness in the chest, but the cough may be persistent, and is more or less productive of expectoration of mucopurulent material. Febrile movement continues if that symptom has been present earlier. The pulse is but slightly quickened and auscultation reveals, particularly over the bronchial tubes posteriorly, large, *moist rales* and *rhonchi.*

The duration of an attack of acute bronchitis rarely exceeds ten days to two weeks, and is often shorter than this.

Treatment.—The treatment of acute bronchitis divides itself into two parts: that part which is devoted to allaying the inflammation in its early stages, and that which is directed toward the dissipation of the results of the inflammation after it has peen present for some days. In the early stage no better remedy can be administered internally than a prescription which contains in each dose ½ a drachm of syrup of ipecac and 1 teaspoonful to 2 teaspoonfuls of the official *liquor potassii citratis* of the United States *Pharmacopœia.* This should be administered every three or four hours.

If cough is an annoying symptom, and there is much pain in the chest, Dover's powder may be given to an adult in the dose of 2 or 3 grains every three or four hours until 10 grains have been taken. A mustard plaster may be applied to the chest, back and front, or the thorax may be rubbed with ammonia liniment or with chloroform liniment. In children a very useful counterirritant application to the chest is 1 drachm of the oil of amber in 2 tablespoonfuls of sweet oil. If the patient be a child, and if the air of the bed-room is particularly dry and irritating, because it is furnace-heated, much relief can be obtained by disengaging a small quantity of steam. This may be given off from a tea-kettle which is kept boiling constantly, or may be obtained by dropping large pieces of unslaked lime into a tub of water. It is probable that when the latter procedure is resorted to the air of the room not only contains an extra amount of moisture, but fine particles of lime, which act advantageously upon the bronchial mucous membrane.

If the evidences of bronchial irritation are very marked, the patient, whether he be an adult or a child, should sleep in a bronchitis tent. A bronchitis tent, it will be remembered, consists in a tent-like arrangement of sheets spread over the bed, and resting upon four corner sticks, one of which is tied to each corner of the bed. Under this canopy the patient will have plenty of air, and the steam from a kettle can be disengaged within its confines. If necessary, 1 or 2 grains of menthol may be added to the hot water every two or three hours. In other instances the bronchial irritation will be greatly soothed by pouring into the boiling water a tablespoonful of a mixture composed of equal parts of compound tincture of benzoin, oil of eucalyptus, and oil of pine.

After secretion has begun to form in the secondary stage of acute bronchitis it then becomes necessary to administer not sedatives, but stimulant expectorants, and of these chloride of ammonium, without any doubt, is usually followed by the best results. From 5 to 10 grains of this drug may be given with equal parts of the fluid extract of licorice and water every four or five hours to an adult, and, if cough is excessive, $\frac{1}{4}$ of a grain of sulphate of codeine or 20 drops of paregoric may be added to each dose. Under the influence of this remedy the expectoration of yellow or mucopurulent sputum is at first increased, but at the end of twenty-four or forty-eight hours the quantity diminishes.

When there seems to be lack of secretion, the compound licorice mixture of the United States *Pharmacopœia*, which contains a small quantity of tartar-emetic, may be used in place of the plain extract of licorice just named. If for any reason the cough and expectoration persist and do not diminish under the use of the chloride of ammonium, we may give with advantage 5 to 10 minims of the oil of sandal-wood in capsules, three or four times a day. Other patients do well at this time if they receive 5 minims of the oil of eucalyptus in capsules three times a day. The latter remedy, however, is quite apt to disorder the stomach. Another very valuable remedy in the secondary stage of bronchitis, to promote expectoration, is terpin hydrate, which is best given in the form of an elixir, dose, a teaspoonful, and which may be much increased in its efficiency if to each dose is added $\frac{1}{12}$ of a grain of heroin, or $\frac{1}{4}$ of a grain of codeine sulphate. The dose of terpin hydrate is from 2 to 5 grains four times a day, but in obstinate cases larger doses may be administered. Terebene may also be given in capsules in the dose of 5 minims three or four times a day.

If the general nutrition of the patient is not good and he seems somewhat debilitated, the employment of cod-liver oil, or syrup of iodide of iron, in moderate doses, will often produce the most advantageous results at this stage of the illness.

CHRONIC CATARRHAL BRONCHITIS.

Definition and Symptoms.—By chronic bronchitis is meant a condition in which there exists a chronic inflammatory process in the bronchial mucous membrane, as a result of which the patient suffers from *cough* and the *expectoration* of *thick, mucopurulent sputum.* When uncomplicated there is no febrile movement in association with this condition, nor is there, as a rule, any loss of flesh or impairment of the general health. It may be regarded as a subacute continuation of an acute cold. The chief objection to the term " chronic bronchitis " is that it so often is applied by the careless or ignorant, or by those who wish to use an euphemism, to designate a far more serious condition, such as pulmonary tuberculosis or bronchiectasis.

Treatment.—The treatment of chronic bronchitis is practically identical with that of the later stages of the acute form, which has just been described, but the most important thing for the physician to do, to whom is presented a so-called case of chronic bronchitis, is to carefully exclude tuberculosis or renal disease as causative factors in the case. Many cases of so-called chronic bronchitis are treated for weeks with ordinary expectorants when tuberculosis is present or Bright's disease is the real cause of the disorder.

BRONCHIECTASIS.

Definition.—Bronchiectasis, as its name implies, is a condition in which the bronchial tubes are dilated. This dilatation may occur in three forms, namely, the *cylindrical* or *fusiform*, the *saccular*, and the *trabecular* or *moniliform*. The first of these is the only true type of bronchiectasis, but the other forms are those most commonly met with.

The disease is not common in its well-developed form. If we combine the statistics of the Brompton Hospital, of London, with those of Biermer and Willigk, we find that in 8144 autopsies bronchiectasis was found in about 4 per cent. These are, however, postmortem figures, and do not represent a certain proportion of cases which do not come to autopsy because of a respiratory ailment.

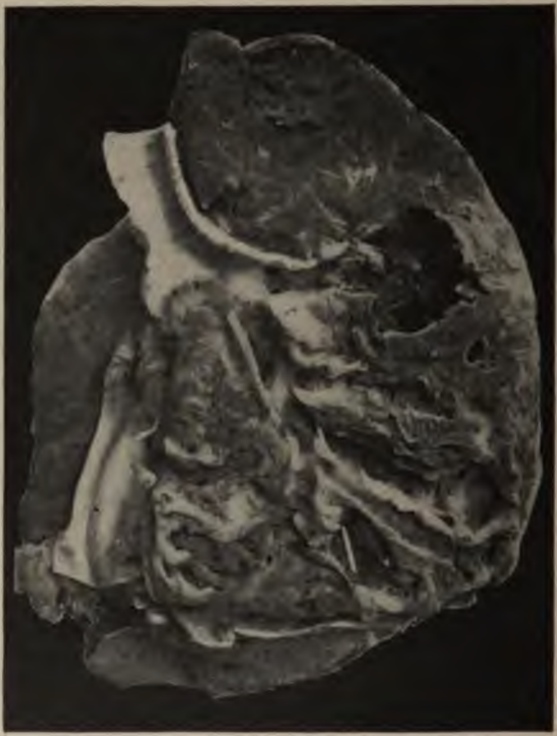
Etiology, Pathology, and Morbid Anatomy.—The *saccular form* is common in adults, and when it is present, the lung, at autopsy, contains one or more saccules, or globular cavities, which usually are not very large, but may be the size of a small lemon. These cavities or open spaces, when large, are due not only to simple dilatation of the bronchi, but to involvement of the surrounding tissues as well, and their walls are composed of parts of the bronchial tubes and thick connective tissue which has been formed in part as the result of chronic inflammatory changes, the lung tissue having undergone fibroid change. Sometimes these spaces are filled with thickened, inspissated secretion, and seem like closed cavities. It is readily seen, therefore, that the differentiation between this state and fibroid phthisis (see Tuberculosis of the Lung), so called, may be by no means easy, particularly as these pouches may become infected, ulcerate, and really form small abscesses.

The *trabecular* form is still less a true bronchiectasis, and yet it is the condition most commonly met with, and to which the term bronchiectasis is most frequently applied. It consists in irregular cavities with smooth linings, which cavities are surrounded by dense walls of overgrown connective tissue which do not contain any signs of remnants of the tissues of the bronchial tubes. On the contrary, the only relation borne by the bronchial tube to such a cavity is that it forms the trabeculæ with the atrophied bloodvessels. These cavities are often joined one to another by openings, so that the lung may be thoroughly riddled with spaces more or less well filled with secretion.

Cylindrical bronchiectasis (Figs. 51 and 52) is usually developed in children as the result of strain upon the bronchial tubes produced by the violent efforts in whooping-cough or measles. It probably depends primarily upon inherent weakness of the muscular and elastic coats of the tubes. The affected bronchus is uniformly dilated in its entire circumference for a considerable distance, and this dilatation may be so great that its calibre is increased to twice or thrice the normal. In other instances it is dilated in sections, with a normal or nearly normal calibre between.

When bronchiectasis occurs in the saccular and trabecular type it is a subacute or chronic disorder, and results from chronic inflammation of the bronchial tubes, with resulting atrophy of the elastic and muscular coats. Of the provoking causes influenza is an important factor. Another cause is the progress of cicatricial or cirrhotic change in the pulmonary parenchyma, which, as it proceeds, distorts the bronchial tubes, narrowing them in some places and widening them in others. In other instances it can be readily understood how chronic thickening of the visceral layer of the pleura may so result. In still others a localized bronchiectasis may be caused by the entrance of a foreign body.

Fig. 51

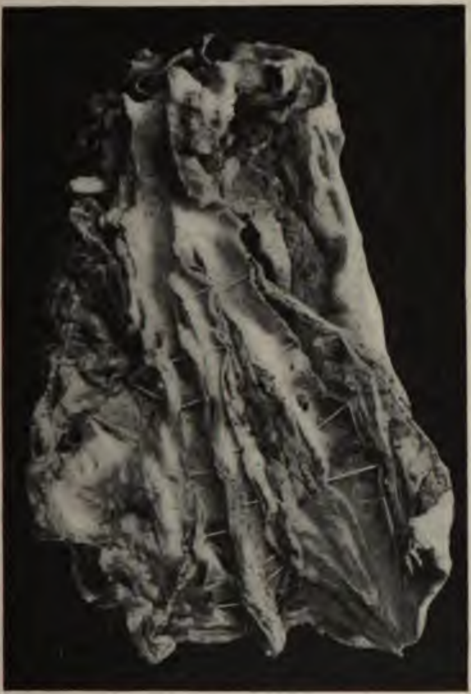


Bronchiectasis, originating in acute lobar pneumonia. Marked saccular and cylindrical dilatations with a large gangrenous cavity in the middle lobe. Duration eleven months. From a case under the care of Dr. Maguire. Dr. Barty King. (Brompton Hospital Museum. Scottish Medical and Surgical Journal.)

The influence of age in the development of the various types of bronchiectasis is quite noteworthy. Cylindrical dilatation is largely a condition limited to childhood, and the saccular and trabecular types are chiefly met with in adults. It has been thought by some physicians that the latter forms occur with increasing frequency as old age is approached, but the statistics of Barty King indicate that the age incidence of the pure type is from thirty to forty years. Thus, 53.1 per cent. of his cases occurred between twenty and forty years. The same observer places the proportions of the two sexes as 77 per cent. in males and 23 per cent. in females.

Symptoms.—The symptoms of bronchiectasis naturally vary greatly with the form of the disease which is present. *Cough* of severe degree may be considered the most constant of them all. This cough is peculiar in that in many cases it is particularly severe in the morning, persisting until the patient has rid his dilated and feeble bronchi of the secretions which have accumulated in them during the night. Not only is this cough peculiar in this respect, but it not infrequently happens that the patient, after a prolonged attack of coughing which is unproductive, is enabled to get rid of a large quantity of sputum, which may come away in a gush or which may not be dislodged until by some change in posture drainage from the bronchiectatic area can take place.

FIG. 52



Cylindrical bronchiectasis. A typical case. Dr. Barty King. (St. Thomas' Hospital Museum. Scottish Medical and Surgical Journal.)

The *sputum* is also somewhat *characteristic*, for it is often grayish-brown in appearance, somewhat *fetid in odor*, and *separates* when placed in a glass into *three layers*, the upper one brownish and thin, the second one mucoid, and the third granular and filled with dead epithelial cells and pus corpuscles. This lower layer also contains large crystals of the fatty acids and crystals of hæmatoidin. The sputum is so distinctly purulent that it may closely resemble that expectorated in cases of pulmonary abscess, a resemblance still further increased by the fact that it may be extremely fetid. It is not nummular, as in many cases of phthisis, and it rarely contains elastic-tissue fibres, which is of some importance in differential diagnosis. *Fever* is usually not present unless the purulent process in the bronchi is marked and septic absorption results. An additional symptom, sometimes met with, is *hæmoptysis* from ulceration of a bloodvessel.

The *physical signs* of bronchiectasis have little about them that is distinctly characteristic, and for this reason an absolute diagnosis may be difficult or impossible unless the lesions are so far advanced as to have affected the whole lung and caused alteration in the configuration of the chest. When the bronchiectatic spaces or cavities are large the physical signs are practically identical with those of pulmonary tuberculosis with cavity formation; but there is this important difference, namely, that the cavities in tuberculosis are commonly apical while the cavernous breathing of bronchiectasis is usually most marked near the base. The physical signs of cavity formation also vary with the condition of the cavity—that is, whether it is full of secretion or empty, and therefore change in the patient's position, and cough with expectoration, may cause very great differences both in examination by auscultation and percussion. Loud, moist rales and amphoric breathing may be present.

Diagnosis.—It is manifest from what has just been said that the differential diagnosis of bronchiectasis from pulmonary tuberculosis may be quite difficult and, indeed, impossible in certain cases, for it not uncommonly happens that a superficial dilated and sacculated area in the lung gives, on auscultation and percussion, physical signs which are identical with those which are produced in pulmonary tuberculosis with cavity. The presence of tubercle bacilli and of yellow elastic tissue in the sputum, of hectic fever, of rapid loss of flesh, and of night-sweats point to tuberculous infection. Another useful differential point is the fact that in tuberculosis the cavity is usually at the apex, whereas in bronchiectasis it is lower down in the lung. Still another point is that the patient is rarely as ill as in tuberculosis, and can get about year after year unless some acute intercurrent pulmonary malady intervenes.

Further than this, bronchiectasis and pulmonary tuberculosis may exist simultaneously. Thus, in 68 cases of bronchiectasis observed by Trajanowski, 21 occurred in individuals who were affected with phthisis, and in 75 autopsies on individuals who died from phthisis Wilson Fox found bronchiectasis in 27 cases. Twenty-one (21) were of the fusiform variety and six (6) were saccular.

Complications.—Hæmoptysis, as a complication of bronchiectasis, is rarely severe and occurred in Barthez's cases 16 times out of 39 cases. Grainger Stewart met with it 3 times in 8 cases, and Fowler met with it 14 times in 35 cases. Three of these cases were tuberculous.[1]

Another complication is rheumatoid arthritis. I have recently had a case under my care in which, after many years of chronic bronchiectasis, a maiden lady developed multiple arthritis, and in the course of a few weeks became completely disabled. Sometimes no more serious joint difficulties arise than swelling of the finger-joints and clubbing of the finger-tips, with incurvation of the nails, or the case develops true pulmonary osteoarthropathy. The joint complications are probably septic in origin. Pulmonary gangrene may also develop, and Duret has operated on such cases with success.

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[1] For some of the cases in French literature see Devic and Bertier, Lyon médical, January, 1904.

Brain abscess may arise from a septic focus in bronchiectasis.

Cyanosis and dyspnœa on exertion are such constant symptoms that they can scarcely be considered as complications.

When we consider the state of the tissues involved we can readily understand how readily a septic bronchopneumonia may be developed in these cases, either as a result of direct extension of the inflammatory process from the area primarily involved, or by the inspiration into other parts of the lung of septic material during paroxysms of coughing.

Prognosis.—This depends upon the state of the patient's health, the presence or absence of sepsis, and the presence or absence of tuberculous infection. In severe forms the health is greatly impaired. Recovery from the condition itself, if it be well developed, is manifestly impossible. Nevertheless, life may continue for many years.

Treatment.—The treatment of well-developed bronchiectasis can be only palliative. Once the condition of dilatation of the bronchial tubes has been established, it is evident that they cannot be brought back to their normal calibre. On the other hand, in the early stages of bronchiectasis, much can be done in the way of palliative treatment. It is a mistake, however, to give sedatives to control the cough unless the cough is so excessive that it materially interferes with sleeping and eating, for cough is a measure designed by nature to rid the dilated tubes of the secretions which certainly do harm if they are retained. Ordinarily, expectorant remedies cannot be expected to do as much good as they do in ordinary cases of bronchitis. The best to be employed are creosote in doses of 3 to 5 minims three or four times a day; guaiacol in the dose of 3 minims three times a day, or guaiacol carbonate in the dose of 3 grains three times a day. In some instances much good follows the administration of a mixture containing iodide of ammonium. Still other cases are benefited by the chloride of ammonium.

It must not be forgotten that many cases of bronchiectasis which have lasted for some years suffer as well from feebleness and dilatation of the right side of the heart, and the degree of cyanosis and dyspnœa on exertion can be much decreased by the administration of small and continuous doses of digitalis or strophanthus, and, in some instances, by the proper use of strychnine. A certain amount of rest in bed or on a couch every day is very advisable; and if the patient seems to have great difficulty in expectorating the contents of certain cavities, experiments should be made with different postures to determine that in which the cavity is most easily drained, and he should be instructed to take this posture in order to avoid prolonged and exhausting spells of coughing.

Within the last few years a number of clinicians have warmly advocated the employment of intratracheal injections of medicaments in cases of bronchiectasis. Various mixtures have been employed, of which perhaps the most popular have contained menthol, guaiacol, olive oil, or albolene. They do little good. In other instances, asserted good results, so far as elimination of the symptoms are concerned, have followed the inhalation of various drugs, such as the vapor of chloride of ammonium, creosote, and tar.

So far as climatic treatment is concerned, these patients should carefully avoid high, dry altitudes, and should resort to hill altitudes or the seaside resorts, unless the latter are too damp, in which case the drier places must be sought, as, for example, Thomasville, Georgia; Lakewood, New Jersey, or some similar spot not too near the sea, where there is a sandy soil and a heavy pine growth. Such patients, too, should be warned of the danger of complications which may follow exposure to sudden changes of temperature and to wet, and should wear flannels next to the skin all the year round, if possible, to avoid chilling the surface. If these precautions are taken, the greater amount of time spent in the fresh air the better, as in-door life for these patients is disadvantageous if the climate is at all suitable to their condition.

FIBRINOUS BRONCHITIS.

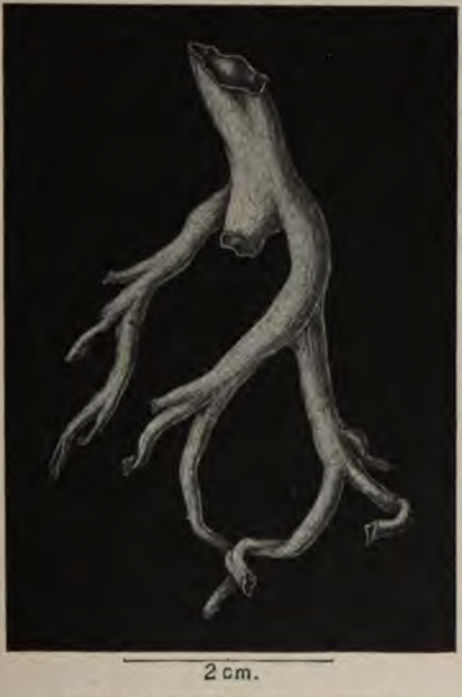
Definition.—Fibrinous bronchitis is an exceedingly rare affection, characterized by the formation of a fibrinous exudate which makes a cast of the bronchial tubes. As ordinarily observed it is in no way related to diphtheria, in which disease, however, casts of the larynx and trachea and even of the bronchial tubes sometimes form.

Etiology.—The cause of this strange affection is practically unknown. When it occurs as a complication or sequel of other diseases, it seems to bear no relation to them save that of coincidence. The condition is much more frequent in males than in females, and is not particularly prone to occur at any particular age. West states the youngest case recorded is four years of age, and the oldest seventy-two years. It has occurred more frequently after acute croupous pneumonia and during the progress of pulmonary tuberculosis and ordinary chronic bronchitis than in other maladies, but its occurrence in these affections is not sufficiently constant to justify us in considering that these relationships are direct. In some instances it is associated with the presence of mitral disease of the heart.

Pathology.—The casts when expelled are found to be composed of masses of gelatinous or pulpy-looking material which, when floated in water or carefully spread upon a glass surface, are found to be in the form of the bronchial tubes; sometimes even of the smaller tubes. The casts are tough and yellowish-white in appearance, and many are composed of fibrin in which may be found white blood cells and epithelium from the bronchial mucous membrane. Other casts contain no demonstrable fibrin, but are rich in mucin. Whether they are distinct forms or altered fibrinous casts is not known. The cast may be hollow or filled with gelatinous mucus. It is a curious fact that these casts may form without resulting in serious lesions of the lining membrane of the tubes, for even the epithelial lining of the bronchial tubes may not be found seriously impaired after a cast is thrown off (Fig. 53).

As the affection is very rare, and still more rarely causes death, we know comparatively little of its true morbid anatomy. Sometimes casts have been found at autopsy when the condition was not suspected to be present, and in other cases in which casts had been thrown off in life none have been found at the postmortem.

FIG. 53



Cast from a case of fibrinous bronchitis.

Symptoms.—The symptoms of fibrinous bronchitis chiefly consist in severe attacks of *cough* and *dyspnœa*, the cough being an effort to dislodge the membrane and the dyspnœa the result of the obstruction to the respiration. Sometimes the dyspnœa has been quite urgent, but it has usually been almost completely relieved after the cast is expelled. This expulsion of a cast may occur once in a lifetime, once in several weeks, once in several days, or several casts may be expelled in one day. Rarely the formation of a cast suggests periodicity. While the cough is usually severe in the effort to dislodge the exudate, the expulsion may be readily accomplished. Occasionally *hæmoptysis* complicates the case, usually amounting to nothing more than slight streaking of the expelled membrane, but in other instances the bleeding is quite profuse. The blood comes from the bronchial, not from the pulmonary, vessels. *Fever* may be present in the acute cases, but is usually absent in the more chronic ones.

Diagnosis.—This condition must be separated from diphtheria, which can be done by the absence of false membrane in the fauces and larynx; from croupous pneumonia, which is possible by reason of the absence of the fever and other signs of that disease; and from foreign bodies in the air-passages, which cause dyspnœa and violent attacks of cough, by the history of the patient.

Prognosis.—The prognosis as to return of the disorder is bad, as most cases suffer from recurrence, although acute cases in which complete recovery has occurred have been reported. In regard to the effect of the disease on life it may be said that this varies greatly with the general state of the patient's health and upon the gravity of the diseases which are associated with it.

In cases with no grave complications recovery may be expected in a majority, both as to complete temporary recovery and recurrence.

Treatment.—The only plan of treatment which has proved itself of value in a sufficient number of cases to be regarded with any confidence is the use of iodide of potassium in full doses. Some patients seem to be made more comfortable by the inhalation of steam. Climatic change is often essential, and Southern California, or Florida, or Madeira, may be resorted to.

BRONCHIAL ASTHMA.

Definition.—Strictly speaking, the word "asthma" may be applied to any condition in which the respiration is labored and difficult, but in medicine it is most commonly used to describe a condition of difficult breathing due to constriction of the bronchial tubes and further narrowing of their calibre by swelling of the mucous membrane lining them. This state of spasm of the bronchial muscle fibres and hyperæmia of the mucous membrane depends upon a neurosis. This neurosis may arise in turn from a large number of causes, all of which probably exercise their influence through the pneumogastric nerves.

It is unfortunate that the term "asthma" has also been applied to labored breathing due to various toxæmias, such as uræmia and the coma of diabetes. Renal disease may, it is true, indirectly produce true asthma, but this word ought not to be applied to that form of labored breathing in which there is no swelling or spasm of the sort described in the preceding paragraph. The term asthma is, therefore, used in this article to mean *bronchial asthma.*

Etiology.—The cause of asthma in many cases cannot be determined, and in some persons it is evidently due to some lack of stability in the nervous control of the bronchial tubes. In others the asthmatic attack arises because of the inhalation of bad air, which acts as an irritant to the respiratory tract, either because of the state of the atmosphere itself or because the air is laden with dust. The influence exercised by the atmosphere in producing asthmatic attacks is very great and varies in different cases to an extraordinary degree. Mere impurity of the air has little to do with this influence in some cases. Thus, I had under my care an old man, from a healthy country district in Pennsylvania, who came to Philadelphia to get relief from nightly attacks of asthma. Without any treatment the severity of the attacks diminished when he breathed city air on the level of the street, dust laden though it was, and his attacks ceased entirely so long as he remained in a private room of the fifth floor of the Jefferson Hospital, where there was less dust, but, perhaps, more smoke and gas from the neighboring chimneys. In other instances gases or fumes, as from coal or arsenic, produce an attack, and in still others the patient only suffers at that season of the year when the pollen of certain plants or flowers is set free. For this reason, sufferers from "hay fever" often suffer from asthma, since the exciting causes of both states are present, namely, a respiratory neurosis and the irritants in the air.

In still other cases the cause lies in the system of the patient and does not come from outside. Thus certain persons who are sufferers from gout will occasionally have attacks of asthma, just as they have pain in the toe or soreness in the voluntary muscles ; and, again, it not uncommonly happens that persons who have an unstable nerve supply to the bronchial tubes have an attack of asthma if exposed to great cold or if they have a slight bronchial congestion due to this cause. So, too, such persons may be seized with an attack as the result of great physical weariness or of nervous excitement, and it by no means rarely happens that feebleness of the heart, which results in poor circulation in the lungs, produces a seizure in susceptible persons. Such a case is called one of "*cardiac asthma.*" In other instances deficient activity of the kidneys produces indirectly a similar seizure or so-called "*renal asthma.*" In some cases great acidity of the stomach, and the various forms of indigestion, reflexly provoke an attack through the gastric fibres of the vagus nerves.

The nervous mechanism whereby an asthmatic seizure is produced is supposed to be as follows: The control of the circulation in the bronchial mucous membrane, and of the muscular fibres controlling the bronchial tubes, resides in the vagus nerves, which possess efferent and afferent fibres, not only connected with the lungs, but with the stomach and heart as well. There are also, in all probability, fibres which indirectly connect the nasal mucous membrane with the vagus. Certain causes of irritation acting upon the respiratory and gastric and cardiac fibres of the vagus give rise to an afferent impulse sent to the vagus centre, and this in turn results in the irradiation of an efferent impulse to the bloodvessels in the bronchial mucosa and to the bronchial muscular fibres, whereby the tubes are constricted, the mucous membrane becomes swollen, and, in addition, secretion takes place, which aids in obstructing still further the smaller tubes.

Pathology and Morbid Anatomy.—The pathology of this condition has been described in part in the preceding paragraph. The morbid change which is manifest is the engorgement of the mucous membrane, the thick, viscid, bronchial secretion, and the spasm of bronchial tubes. About this subject many earnest discussions have taken place and the profession are not in accord. Brodie and Dixon speak of four theories as to the cause of the attack, but believe that the spasm of the bronchial muscle is the essential factor. The other possible factors, in their opinion, are swelling of the bronchial mucosa, the so-called reactionary hyperæmia of Traube or vasomotor turgescence of Weber, *bronchiolitis exudativa* of Curschmann, and, lastly, a reflex spasm of the respiratory muscles.

The morbid anatomy, unless secondary conditions arise, is *nil*, for with the disappearance of the attack the lungs attain their normal state within a short time. It is only when repeated attacks of asthma occur that the patient as a consequence suffers from chronic bronchitis, emphysema, or bronchiectasis, although, if a single attack is very severe, he may develop bronchopneumonia, particularly if exposed to cold and dampness.

The chest of the asthmatic patient, who has suffered from this disease for many years, is usually like that of pulmonary emphysema in its configuration, and if the disease be present in early life, when the chest is very pliable, a "pigeon-breast" may be developed, or a well-marked Harrison's groove may be seen.[1]

In most instances in which asthma has been present for years the heart undergoes dilatation and hypertrophy, particularly on the right side, and its beat may be quite feeble if hypertrophy has not fully compensated for the dilatation. Secondarily, these cardiac changes may result in hepatic and renal congestion.

The scanty sputum which is expelled by asthmatic patients possesses in many instances peculiarities which are pathognomonic. This sputum, if examined, is found to contain little lumps or balls, which, if they are teased out on a plate of glass placed on a black background, are found to consist of minute curls or twisted fibres, in form not unlike the curls of hair on a child's head. These curls are called "*Curschmann's spirals*," and in their folds are found crystals of the fatty acids, the so-called "*Charcot-Leyden crystals*." That fatty acids are present, however, is denied by many. Thus, Goodhart and Taslett think these curls are related to the casts of fibrinous bronchitis, and they point out that such casts frequently show twists or spiral terminations. Hoffmann thinks the terminal bronchioles are spiral in form.

Before and during an attack of pure bronchial asthma there is an extraordinary increase in the number of eosinophiles in the blood. This eosinophilia is said not to occur in cases of renal and cardiac asthma.

Symptoms.—The symptoms of spasmodic or bronchial asthma, in well-developed cases, are very typical. The patient usually retires to bed perfectly well and wakes at midnight or in the early morning with a sense of *intense dyspnœa* and *oppression*, which may be so severe as to seem to threaten death from asphyxia, but death never occurs in an attack from this cause. The *attitude* of the patient suffering from asthma is most characteristic. If he is in bed he sits up and places his hands back of him on the mattress, so as to support himself in that posture which will enable him to use his auxiliary muscles of respiration to the greatest possible extent. His *respirations are labored*, his brow is covered with *sweat*, and his face is at first anxious and pale, and then *cyanosed* and *livid*. The efforts at inspiration and expiration are forcible, but the chest has the appearance of distention, since the intercostal spaces are often unduly full, and the anterior portion of the thorax is elevated. The difficulty under which the patient labors is that he retains in his chest an excess of air which has become vitiated, but which he cannot expel, and therefore he has no room for fresh air. The condition is rather one of difficult expiration than of difficult inspiration. Owing to the great shortness of breath the patient is often unable to speak except in a whisper, and speaks but a word or two with each breath. The superficial veins are engorged. The *urine* during an attack is often *scanty* and heavily loaded with urates, but after the attack it is often passed in large quantities, and is clear and limpid.

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[1] "Harrison's groove" is that depression which begins at the sternum at the attachment of the seventh or eighth rib, and extends backward in the line of the ribs toward the axilla.

The attack may last from half an hour to several hours, and leaves the patient quite exhausted. In some instances, with the passing of the seizure, almost total respiratory relief follows, but in most cases some dyspnœa persists for several hours, and cyanosis may be present till all respiratory difficulty is relieved. If the degree of relief is sufficient to permit sleep, the patient may be sufficiently rested to attend to business the following day, but in the majority of cases this is impossible, or at least inadvisable, because of the fatigue, the weak condition of the heart and lungs from the effects of the attack, and the presence of the bronchitis and bronchial secretion, which may result in fatal bronchopneumonia if the patient is not very prudent as to exposure.

Diagnosis.—The history of the attack, in its mode of onset and subsequent development, renders a diagnosis easy. The duty of the physician is to discover, if possible, the cause of the attack and remove it, resting confident that asthma is always a symptom and not a disease. The physical signs present in an attack are very characteristic. In the early stages auscultation reveals harsh bronchial breathing, with musical rales, which may be scattered here and there through the chest, and owing to the disturbed respiratory cycle it may seem as if one part of the lung does not expand simultaneously with the other parts. When the attack is well developed the difficult passage of air through the narrow tubes results in the still greater development of musical sounds, which may be described as resembling those made by a litter of mewing kittens or crying puppies. These to-and-fro, loud, musical rales, widely diffused through both lungs, are so characteristic of the asthmatic patient as to make the diagnosis certain in many cases.

There is one condition from which asthma in the later stages must be carefully separated, namely, that of pulmonary œdema. Aside from the fact that the underlying cause of pulmonary œdema is often serious renal disease, and therefore a dangerous state deserving recognition, the history is often given of previous attacks of shortness of breath, or even of wheezing respirations; and the physical signs in the later stage of bronchial asthma, when widely diffused and musical moist rales are heard, may not differ materially from those of pulmonary œdema, since musical moist rales and some impairment of resonance on percussion may be present in this condition as well. In spasmodic croup the obstruction to respiration is so clearly laryngeal and the chest is so free from widely diffused rales that the diagnosis is not difficult, and in laryngeal spasm due to locomotor ataxia the same freedom from musical rales in the chest again enables us to make a differentiation.

Sometimes labored respiration resembling that of spasmodic asthma occurs in acute pneumothorax, but the physical signs are so different that no difficulty is experienced in separating these two conditions.

Prognosis.—The prognosis as to recovery from an individual attack of asthma is very favorable, even if it be exceedingly severe, provided that no acute complication arises. The prognosis as to recovery from the tendency to asthma is very bad, for the history of the vast majority of cases is that they have recurrences. It is only in those cases in which there is a manifest exciting cause, external or internal, which can be removed, that a favorable prognosis as to the future can be advanced. The tendency of spasmodic asthma to produce bronchopneumonia, emphysema, dilatation of the right side of the heart, and secondary circulatory feebleness must never be forgotten; but, on the other hand, it is remarkable that very many asthmatics live to moderate old age without being invalided.

Treatment.—The treatment of spasmodic asthma may be divided into three parts: that devoted to the prevention of the attack, the relief of a paroxysm which is present, the removal of the underlying causes and of the sequelæ which are produced by the attack. It has already been pointed out that certain conditions of the atmosphere and the presence of certain kinds of dust in the air strongly predispose certain individuals to attacks of asthma. On the principle that an ounce of prevention is worth a pound of cure, it is evident that asthmatic patients should be exposed as little as possible to such provoking causes, and if they must of necessity sleep in a room the air of which has been heated by a furnace, steam should be disengaged in the air of this room, so that it will not be unduly dry. Such patients should be subjected to a careful examination of the nasal, pharyngeal, and tracheal mucous membranes, with the object of discovering whether they suffer from any localized spot of hyperæsthesia in these mucous membranes, for it not infrequently happens that foreign bodies or dry air may irritate these spots and so reflexly produce an attack. Indeed, in some cases of so-called "nasal asthma" it is possible by touching a hyperæsthetic spot on the nasal mucous membrane to precipitate an attack of spasmodic asthma. Such hyperæsthetic spots should be removed by the application of the cautery where it can be employed.

For the relief of the attack of asthma itself when it is threatened, the inhalation of nitrite of amyl and the drinking of strong, black coffee may be resorted to, or a hypodermic injection of morphine and atropine may be given.

When the attack is developed, an innumerable number of drugs have been recommended by various practitioners and by a still greater number of sufferers. Among the older remedies, without doubt, belladonna and its sister drugs possess the confidence of a large number of the profession; but none really exercise a powerful curative influence, unless they are given in doses which are so large as to be almost capable of producing moderate poisoning. These drugs probably act by their depressant influence upon the vagus nerve, and by altering the circulation in the capillaries supplying the bronchial mucous membrane. They are particularly useful in those cases of asthma in which, during the attack, there is formed a considerable quantity of bronchial secretion, and they are the chief ingredients, with nitrate of potassium, of most of the proprietary cigarettes and powders which are burned in the patient's room.

Of the so-called depressant remedies for asthma, we have lobelia, which is very highly thought of by many practitioners, particularly in England. On the other hand, some physicians are afraid of this drug, because of the depressant influence upon the heart. It is not to be employed when the heart is feeble. When the heart is strong, it should be given in full doses, if given at all. As much as ½ to 1 drachm of the tincture should be given in one dose, and repeated in the dose of 10 minims every half-hour or hour until the patient's circulation is markedly depressed and the skin is relaxed and perspiring. These doses may produce nausea and even vomiting, but the associated relaxation often will abort an attack, whereas smaller doses which do not produce vomiting may produce more profound circulatory symptoms, since all of the drug is absorbed and none lost by emesis. Pilocarpine may also be employed in those cases of spasmodic asthma in which there seems to be an excessive dryness of the bronchial mucous membranes. But the fact that this drug in some cases seems to depress the heart seriously, and in others cause an excessively profuse outpouring of bronchial secretion, has properly prevented its general employment. Many patients experience great relief in the early stages of an attack if they receive a hypodermic injection of ¼ grain of morphine with $\frac{1}{150}$ grain of atropine. In a disease which recurs frequently, as does asthma, this use of morphine is always dangerous, in view of the possibility of establishing the morphine habit. Furthermore, the after-depressant effects of the drug upon the following day often renders the remedy almost as bad as the disease.

For internal administration in the treatment of asthma, there is no drug which meets as many indications in as many cases as nitroglycerin. If the attack is threatened $\frac{1}{100}$ or even $\frac{1}{50}$ of a grain should be given hypodermically, and the same dose may be repeated every hour or two, particularly if the patient is one of advanced years and has a somewhat high arterial tension. In some cases the inhalation of a few minims of nitrite of amyl, poured upon a handkerchief, will serve to abort a threatened attack or to modify the severity of one which is already well developed.

In those cases where there is great irritability of the nervous system underlying the asthmatic attack, the occasional use of the bromides may be advantageous, but they are of little value for the prevention of an individual attack, and their continued use between attacks is obviously unwise. The same opinion may be expressed in regard to the employment of chloral, which has the additional disadvantage that the chloral habit may be instituted, or that the heart may be depressed to an undue degree.

For many years the author has employed a compound in tablet or elixir in the treatment of asthma, both as a preventive remedy and as a cure for individual attacks, and has gotten results from it which cause him to regard this formula with considerable favor. It is now placed on the market by all large manufacturing druggists.

℞.—Sodii iodidi	gr. ij.
Potas. bromidi	gr. ij.
Ext. euphorbiæ piluliferæ	♏iij.
Nitroglycerini	gr. $\frac{1}{200}$.
Tinct. lobeliæ	♏ij.—M.

Ft. in tabel. vel capsul. No. i.
S.—One every four to six hours.

It must never be forgotten, however, that asthma is a symptom rather than a disease, and that the remedies which prove useful in one case may in another prove entirely useless, because in each instance the underlying cause of the malady is quite different. It is this fact which has probably caused some physicians to speak in high praise of certain remedies in the treatment of spasmodic asthma, while others with equal experience assert that they have gotten no good results from the employment of such drugs.

Reference has already been made to the value of supplying asthmatic patients with moist air, particularly when they live in furnace-heated houses. It is the author's constant habit, when cases of asthma come under his care to place them in a bronchitis tent. To the air of this tent is supplied a small quantity of steam, with the result that the patient has a great diminution in the degree of dyspnœa, and frequently gets some hours of refreshing sleep. An additional advantage in putting these patients in a bronchitis tent is that it requires them to remain in bed, and so gives rest to the heart, which organ is often sadly in need of relief, since the difficulty of breathing and the lack of sleep throws upon it, day after day, in some patients, a very severe strain. In some instances the addition of a few grains of menthol to the boiling water seems to increase the efficiency of the bronchitis tent. In still others equal parts of oil of pine, oil of eucalyptus, and compound tincture of benzoin may be added, in the quantity of a tablespoonful or two to the boiling water, with benefit to the patient.

In all cases of asthma the physician should carefully examine the heart, and if there are any evidences of feebleness and dilatation of the right side of the heart, as manifested by venous engorgement, and the extension of cardiac dulness downward and to the right, small doses of strophanthus or digitalis should be given. In some cases in which the cardiac difficulty is marked during the attack, full doses of Hoffmann's anodyne are advisable. These drugs may be assisted in their stimulating influence by one or two hypodermic injections of strychnine.

It is also the duty of the physician in all these cases to carefully and repeatedly examine the urine, since renal disease sometimes produces true asthmatic seizures, and still more commonly produces attacks of dyspnœa, which the patient may call asthma, but which are really those of true uræmic poisoning. Then, too, if asthma is present with a moderate degree of albuminuria without casts, this albuminuria may aid the physician in determining that the heart is yielding under the strain, since this albuminuria is frequently due to renal congestion, resulting from a feeble circulation. The albuminuria disappears, the urinary flow becomes more profuse, and the heart's action gets better under the administration of digitalis or strophanthus, combined with rest, as already indicated.

DISEASES OF THE LUNGS.

BRONCHOPNEUMONIA.

Definition.—Catarrhal pneumonia, lobular pneumonia, or bronchopneumonia is an acute inflammation of the small bronchioles and of the tissues immediately surrounding them and their attached lobules, and primarily involves the lobules, rather than the lobes as does the croupous type of pulmonary consolidation. It is called bronchopneumonia because of this primary inflammation of the smaller bronchi, and it is called lobular pneumonia because it affects the lung by lobules rather than by lobes. More commonly still it is designated catarrhal pneumonia, since it usually follows inflammatory changes in the mucous membrane of the bronchial tubes. No single or specific micro-organism is the cause of bronchopneumonia, but it is due to infection of the bronchi and adjacent tissues by many pathogenic germs.

As with typhoid fever, so with bronchopneumonia: Gerhard, a Philadelphia student of Louis, in Paris, was the first person to clearly differentiate bronchopneumonia from croupous pneumonia (1834), although as early as 1823 Seger had separated the pneumonia of adults from this form which is that which commonly affects children.

Distribution.—Bronchopneumonia, because of its various causes, is found everywhere throughout the world.

Etiology.—Frequently bronchopneumonia is due to the micrococcus of croupous pneumonia, which for some unknown reason fails to produce a croupous exudate in a single lobe or in several lobes, as is usual when that organism enters the lung in an adult. In other instances pyogenic organisms such as the streptococcus or staphylococcus are responsible for the disease. Thus, in 103 cases of bronchopneumonia examined by Netter, Weichselbaum, and Pearce, the streptococcus was found in about 30 and the pneumococcus in 29. When associated with other organisms the number of instances in which these cocci were found was much greater. Primary bronchopneumonia is usually due to the pneumococcus, and secondary bronchopneumonia to the streptococcus.

When such a specific malady as diphtheria is the primary cause of the illness the Klebs-Loeffler bacillus is the most frequent cause of the pneumonic lesions. Thus, in 62 cases of bronchopneumonia following diphtheria examined by Pearce, this organism was found 52 times and the streptococcus 27 times. In still others the bacillus of Pfeiffer (that of epidemic influenza) brings on an attack. The tubercle bacillus not rarely is responsible for the inflammatory process, and almost any pathogenic organism entering the lower bronchial tubes may act as an exciting cause. Doubtless these organisms often gain access to the bronchioles in periods of good health without producing evil effects, but if by chance there is present a general or local impairment of vital resistance pathological changes ensue.

Bronchopneumonia is usually said to be capable of division into *two* *types*, namely, the *primary* and *secondary*. The primary form is met with in children and adults who are usually in poor health with diminished vitality, and seems to have its onset with the development of an acute "cold." It is most commonly met with in infants, and in them, as has just been stated, is usually a pneumococcus infection, although true croupous pneumonia at this age is not common. The secondary type is much the more frequent of the two; indeed, it may be considered the rule that bronchopneumonia occurs as a secondary affection in the vast majority of cases, for nearly always there is a history of a previous acute or subacute bronchitis, or of some disease which predisposes to such a condition, as whooping-cough, measles, or influenza.

In adults there is usually the history of a severe cold affecting the upper respiratory tract, or of the use of an anæsthetic drug by inhalation, which has at one and the same time irritated the air-passages and permitted the entrance of saliva or particles of mucus or food containing many micro-organisms which produce infection. A similar result may accrue in instances in which no such drug is employed. Thus, in some asthmatics during the progress of an attack there may be drawn into the air-passages micro-organisms from the mouth or tiny particles of food. In individuals suffering from the coma of alcoholism, cerebral congestion, uræmia, or apoplexy, with stertorous breathing, the same accident may ensue. Such a form of infection may occur in the coma following an epileptic seizure. In some instances the presence of an ulcerative laryngitis, due to syphilis, tuberculosis, or malignant disease produces an infection in this manner. Bulbar paralysis, or that due to diphtheria, may also provoke this type of pneumonia. In many cases of severe illness, as in typhoid fever, with foul secretions in the nose and mouth, this method of infection ensues because the ordinary sensitiveness of the glottic mucous membrane, and that of the trachea, is obtunded by the dryness of these parts or by the benumbing effects of the disease. This form of the disease is called aspiration pneumonia, or the "Schluck-pneumonie" of the Germans. Old age and debility are also predisposing causes.

Prevention.—From what has been said it is evident that the secondary forms of bronchopneumonia are capable of prevention, at least to some extent. Perfect cleanliness of the mouth is one of the methods of prophylaxis, in that it prevents the inhalation from the oral cavity of infecting micro-organisms. So, too, during the course of measles and whooping-cough careful avoidance of exposure and the use of a bronchitis tent to allay bronchial irritation is preventive in its influence. If local lesions in the upper respiratory tract exist, they should be modified or removed by proper treatment.

Frequency.—Bronchopneumonia is an exceedingly common disease probably outranking in frequency its sister malady, croupous pneumonia. As a terminal infection it causes death in many maladies otherwise almost never fatal in themselves, such, for example, as whooping-cough and measles, both of which have a high mortality in very young children from this very cause. Thus, out of 446 cases of bronchopneumonia in children cited by Holt, it followed or complicated whooping-cough 66 times and measles

89 times. It is a noteworthy fact that bronchopneumonia is the type which is particularly common in infancy, while croupous pneumonia is generally a disease of later life. In the child under five years it is very common, and fatal in direct proportion to the youth of the patient, whereas croupous pneumonia is rare in this period of life, and very rarely is fatal at this time. Out of Holt's 426 cases, 53 per cent. occurred in the first year of life and 33 per cent. in the second year.

Pathology and Morbid Anatomy.—This form of pneumonia, at least in its earlier stages, occurs in patches which cause the lung to present during life physical signs, and, after death, macroscopic appearances, ordinarily quite distinct from those of the solidified or hepatized lung of croupous pneumonia. Macroscopically the lung presents a mottled appearance because its surface represents three conditions of the pulmonary parenchyma, namely, (*a*) areas of consolidation, (*b*) areas of atelectasis or collapse, and (*c*) areas of emphysema, or enlargement, of groups of vesicles due to overdistention, resulting from collapse of adjacent lobules. The consolidated areas are pinkish, reddish, or grayish-yellow in hue, the emphysematous patches are paler and crepitate when touched, while the collapsed portions are bluish or mahogany in color and depressed below the rest of the cut surface of the lung.

The inflammatory process usually begins in the smaller bronchi and extends from them to the tissues immediately adjoining, forming patches of consolidation, which are deep red in hue, and which extend farther and farther from their original site, until perchance they coalesce and form fairly large airless consolidations. As the margin of the inflammatory zone extends, the primary area of inflammation undergoes necrotic degenerative changes, loses its red appearance, and may become grayish, through granular and fatty degeneration of the exudate. This inflammatory exudate not only invades the peribronchial tissues, but the vesicles as well, so that they are rendered airless. If the lung be cut across these patches of consolidation will project slightly, and in the centre of each can be seen the cross-section of the primarily involved bronchus, which looks whitish, and from which mucopus may exude. In some instances in which the infection is severe and the inflammatory process rapid, the mucopurulent character of the exudate into the bronchial tubes is very well developed, and this purulent process may extend into the peribronchial spaces and, in septic cases, cause small pyogenic foci. The exudate itself is composed, as would be expected from the character of the lesions, of serum, red cells, epithelial cells which have separated from the bronchial and vesicular walls, and a large number of leukocytes, and in varying numbers the associated bacteria.

The exudate contains much less fibrin than it does in croupous pneumonia, and a copious fibrinous deposit on the pleura is exceptional.

If the inflammation of the walls of the bronchial tubes is severe they become thickened and swollen, and therefore their lumen is greatly decreased or even occluded. This result is greatly aided by their becoming plugged with the mucus and dead cells, and so it not infrequently happens that a certain area, or several areas, of the vesicular portion of the lung is deprived of air and undergoes collapse or atelectasis.

It is worthy of note that catarrhal pneumonia is in the great majority of instances present in both lungs, and that it is not usually conspicuous in the bases posteriorly. The anterior portions of the lungs and particularly the apices, except in tuberculous cases, show little involvement unless the lesions are well developed elsewhere.

The exudate in bronchopneumonia undergoes resolution, as do most inflammatory exudates, by the degeneration of the extravasated and desquamated elements and their speedy absorption or expectoration. With this process the material plugging the bronchial tubes disappears and the collapsed vesicles, upon receiving their normal supply of air, expand so that complete recovery ensues.

When this does not take place we find the development of dense connective tissue about the air tubes and between the air spaces, which, as it increases in degree, causes thickening and induration. As this process increases the connective tissue distorts the lung so that the bronchi are twisted or bent, patches of vesicular tissue collapse, secretion is retained in the bronchial tubes, and as a result chronic bronchial inflammation, dilatation, or sacculation of these tubes occurs, and the patient becomes a sufferer from chronic bronchitis, with bronchiectasis or a chronic bronchopneumonia.

In other instances old tuberculous lesions are rendered active by the acute bronchopneumonia, or a new tuberculous infection is superadded, so that the case speedily passes into a well-developed pulmonary tuberculosis. In still other instances the whole process from the very beginning is really due to the *Bacillus tuberculosis*, and the patient rapidly develops unmistakable evidences of tuberculous infection, the microscope showing, sooner or later, the presence of these organisms in the sputum. In such cases the exudate in the air cells goes on to caseation, and may become encapsulated or disseminated, depending upon the virulence of the organism and the resistance of the patient.

Symptoms.—The symptomatology of bronchopneumonia *varies with the primary cause.* If it be primary it naturally presents symptoms which differ somewhat from those which it presents when it is secondary, and follows some more or less prolonged and exhausting malady. Then, too, the symptoms naturally vary with the age of the patient attacked, with the areas of the lungs which are involved, and with the severity of the illness which has preceded it.

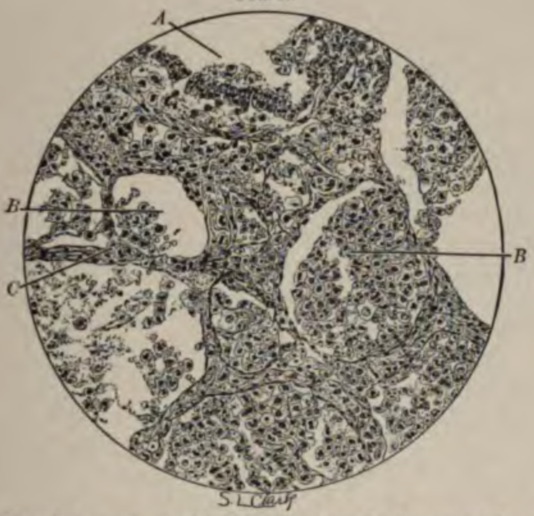
For the ready study of the symptoms of bronchopneumonia we may therefore form at least *three classes of cases:* those which are distinctly primary, those that are clearly secondary, and those which involve the small bronchioles very early in the attack, producing what is known as *acute, suffocative catarrh.*

When the child is attacked with the *primary form of bronchopneumonia* there is usually a *chill* at onset, which varies greatly in its severity, in some cases being so slight that it is scarcely noted, and in others amounting to a true rigor. Commonly there is a history that the child has been exposed to cold and wet. As in all inflammatory conditions in childhood, there is a sharp *rise in temperature,* almost from the very first an increase in the respiratory rate, and, it may be, very considerable evidence of respiratory difficulty.

It is curious to note how the severity of the symptoms of catarrhal pneumonia vary in different children. Some become *dyspnœic* very early, and others suffer very little respiratory embarrassment through the whole course of the malady.

So sudden may be the onset in this primary form of bronchopneumonia that it may be practically impossible for the physician to separate it from croupous pneumonia, particularly as children suffering from the latter disease rarely bring up rusty sputum, and also because they frequently have a pulse rate which is higher in proportion to the respirations than that ratio which is common in croupous pneumonia in adults. Cerebral symptoms may also be present, just as they are in croupous pneumonia; and this is not surprising in view of the fact already pointed out, that the pneumococcus is the micro-organism most frequently responsible for both forms of pneumonia in children. For this reason, probably, primary catarrhal pneumonia in children not infrequently ends by crisis, and recovery may speedily take place after a very few days of illness. Indeed, the outlook in a case of this kind in an otherwise healthy child, which is not very young, is usually favorable.

FIG. 54



Lung of a child. Catarrhal pneumonia following measles. In the upper left quadrant is part of the wall of a small bronchus, the epithelium of which is desquamating (*A*); several air vesicles containing catarrhal exudate are shown (*B*). The connective tissue of the bronchus and the intervesicular structure are slightly œdematous and the seat of considerable leukocytic infiltration (*C*).

The symptoms of the onset of *secondary bronchopneumonia* vary greatly from those just described. Instead of having a sharp onset the onset is insidious. A child having been ailing from some other malady for a number of days or weeks, is found to have an *accession of fever*, to be languid, to have a rapid pulse, to have a very marked increase in respiratory rate, and the skin is found to be hot and dry. The speed of the pulse is often excessive, reaching 150 to 200 a minute; but in this disease, as in all others, its quality is of as great importance as its speed. An irregular pulse is of evil import. The *cough* is fairly constant and sometimes produces *pain*, and if the area involved is at all large respiratory embarrassment is early manifested. This is shown by the increased number of respirations, by the fact that they are somewhat labored, and also by the fact that the intercostal spaces are frequently drawn in by the suddenness of the inspiratory movement. *Auscultation* will probably reveal in the smaller bronchial tubes some fine rales, with *exaggerated inspiratory* and *somewhat prolonged expiratory sounds*. The breathing is of the exaggerated, puerile type; the cough is unproductive. *Percussion* for the first twenty-four hours of the attack may reveal practically nothing. This is in part due in children to the resiliency of the entire chest, so that, unless very gentle percussion is exercised, the resonance of neighboring parts may cover the impaired resonance of the consolidated area. Again, those areas which have undergone compensatory dilatation possess a hyperresonance which may cover the impairment. If, however, the pathological lesion is well developed, at the end of twenty-four or forty-eight hours careful percussion will usually reveal distinct impairment of resonance, particularly if the lesions are, as is common, chiefly at the bases posteriorly.

If the disease is severe the symptoms of *dyspnœa* may become distressingly well marked, and *cyanosis* may become constant, the child being so short of breath that it ceases to cry, and, indeed, may have difficulty in taking liquids because of its dyspnœa; that is to say, it is so short of breath that it cannot take time to swallow. Usually at this time the *expression* is somewhat *anxious*. If the dyspnœa has been prolonged enough to exhaust the child, and the accumulation of carbon dioxide in the blood has been sufficient to benumb its sensibilities, it is *markedly apathetic*. These symptoms last a variable number of days, but usually a change for the better in a mild case begins to be noted by the end of the fifth, sixth, or seventh day, and with the beginning of improvement in the general symptoms auscultation will reveal that the rales in the chest are more moist, that on coughing they alter in quality more than before, and, further, air will be found passing through portions of the lung which heretofore have seemed devoid of it.

In other instances the disease runs a much longer course, and the child, after hovering between life and death for a number of days, slowly emerges from its illness, and the physical signs in the lungs equally slowly disappear.

In young children it is by no means an uncommon occurrence for the disease to spread in a violent form into the smaller bronchioles, and by the swelling of the mucous membrane, the copiousness of the exudate, and the wide area involved, produce what is known as *acute suffocative catarrh*, a condition which at one time was considered as a separate entity from bronchopneumonia, but which is now recognized as being simply a malignant form of the disease involving a large number of the smaller bronchioles, and so greatly interfering with respiration. Another term which has also been used to describe this condition is "capillary bronchitis;" in other words, this name is meant to bring out the fact that the finer bronchioles are involved. A very excellent term to describe this form of the disease is "acute disseminated bronchopneumonia."

Capillary bronchitis, or *acute suffocative catarrh*, is one of the most distressing acute maladies which affect young children. Its onset is usually very rapid, and within twelve or twenty-four hours the child may be suffering intensely from *dyspnœa*. At first, the dyspnœa and inability to get a sufficient quantity of oxygen render it fretful and restless; but very soon it becomes *weary*, and with the weariness of the general system there develops a weariness of the respiratory centre, which fails to send out sufficiently powerful influences to cause the remaining healthy portion of the lung to be completely filled at each inspiration. Very speedily, too, carbonic acid gas accumulates in the blood and benumbs the respiratory centre, so that within twenty-four or thirty-six hours after the beginning of the malady the child may lie in its mother's arms limp and motionless except for the rapid respirations which are required to maintain life. I have not infrequently seen a child in this condition as limp as it is when under the influence of ether or chloroform, and only semi-conscious. The finger-nails are livid, the lips much darker than normal. The mouth is apt to be excessively dry, owing to rapid evaporation of moisture from the high fever and rapid breathing.

During a portion of the attack the respiratory rate may become as high as 60 or even 70 a minute, and not infrequently death comes on as a combined result of the infection, of the dilatation of the right side of the heart, of the accumulation of carbon dioxide in the blood, and of general nervous exhaustion.

Probably the most characteristic symptom of capillary bronchitis is the *intense dyspnœa*, which is quite as acute in some cases as it is in diphtheria with laryngeal obstruction. Indeed, I have known intubation to be done in a case of capillary bronchitis, it being thought that the child had laryngeal trouble in addition to its pulmonary difficulties, although no laryngeal lesion was actually present.

The *temperature* in cases of capillary bronchitis varies very greatly. In some patients it is very high, rising, it may be, to 107°; in other instances it rarely passes above 101° or 102°. In the latter type, however, there may be a sharp rise a few hours before death. At the time of death the temperature may, superficially at least, be subnormal, although the rectal temperature may be high. The general run of the temperature pursues no definite course as it is wont to do in croupous pneumonia, but progresses very irregularly.

The degree of fever in bronchopneumonia of the secondary type is of little value for the purpose of determining the severity of the disease. Sometimes when the infection is quite severe the temperature may not rise above 101° or 102°; whereas in other cases which are really less ill, it may reach 105° or 106°.

Duration.—The duration of bronchopneumonia varies very greatly with the condition of the child at the onset of the disease. In primary bronchopneumonia, occurring in an otherwise healthy child, the malady may last for but a few days, and it is a noteworthy fact that it may be arrested in any stage of its development, so that recovery may speedily take place.

In secondary bronchopneumonia the duration is apt to be very much longer than in the primary form. Under these circumstances it commonly runs a course of from ten days to two weeks, and if the condition of the patient is seriously impaired at the time of its onset, it may last for three or four weeks. Usually, however, during the last ten days or two weeks of a prolonged attack of this character, the symptoms are much modified and the temperature is but a little above normal. Whooping-cough, which is a very frequent cause of bronchopneumonia, runs a course of from six to twelve weeks, and if bronchopneumonia develops early in the attack of whooping-cough the persistency of the spasmodic seizures, with their accompanying bronchitis, naturally prolongs the duration of the pulmonary disorder; whereas, in another disease like measles, which runs a much shorter course, the pulmonary disorder may disappear almost as soon as the eruptive disease, although very often it persists for a week or ten days, and convalescence from measles is well established. Much depends, too, as to the duration of the malady, upon the size of the areas of consolidation and the presence of more slowly liquidated exudates. Commonly, the greater the area infected, the greater the length of the disease.

Complications.—The complications of bronchopneumonia are not numerous. As already pointed out, it occasionally happens that tuberculosis develops in the area which is diseased. More rarely still the infection of the peribronchial tissues is so severe that pulmonary abscess results. Pleuritis is rare unless there happens to be a patch of consolidation close to the pleura, in which case a small area may be involved. This rarely spreads and still more rarely is accompanied by marked effusion, but it sometimes results in empyema.

Diagnosis.—Bronchopneumonia is to be differentiated from ordinary severe bronchitis by the presence of patches of impaired resonance on percussion, and by the fact that during the course of an acute bronchitis an exacerbation of temperature and of the general symptoms of severe illness ensue. Beyond these points it may be practically impossible to separate the two maladies. It is not to be forgotten that bronchitis is not associated with hyperresonance in any portion of the chest as a rule, but catarrhal pneumonia is frequently associated with this physical sign. Percussion in capillary bronchitis may therefore give exaggerated resonance owing to the emphysematous state of the vesicular parts of the lung, for, as in asthma, the difficulty, which often exists, is an inability to expire some of the air which has been taken in by forced inspiration. As has already been stated, hyperresonance may completely take the place of impaired resonance due to consolidation.

From croupous pneumonia bronchopneumonia is to be separated by the fact that its onset is more gradual or insidious, by the intermittent character of the fever, and by the irregular distribution of the physical signs in the chest. Another important differential point is the fact that catarrhal pneumonia is usually bilateral and joined at the bases, whereas croupous pneumonia is not; that bronchopneumonia is usually well diffused, and in both lungs, whereas the croupous type is usually but not always unilateral, and commonly limited to one lobe. Croupous pneumonia usually ends by crisis, whereas bronchopneumonia may end by lysis. The predominance of severe cerebral or meningeal symptoms is rather in favor of the croupous type of the disease.

In many cases it is impossible to determine whether the bronchopneumonia is due to the pneumococcus or some other coccus, or is the result of the infection by the tubercle bacilli. In the absence of enlargement of the mesenteric glands and of other signs of tuberculous infection, the differential diagnosis between bronchopneumonia of tuberculous origin and that due to ordinary causes is practically impossible, until the disease has advanced so far that other systemic manifestations of tuberculous infection are manifest.

Sometimes the intermittent and irregular temperature curves of bronchopneumonia suggest the possibility of malarial infection. The differential diagnosis in a case of malarial infection, with bronchial symptoms like those of bronchopneumonia, can of course only be made by careful examination of the blood, and by a more careful study of the temperature chart than is usual in the ordinary case. Then, too, in the malarial forms of the disease the symptoms will be modified or arrested by the use of quinine.

As already pointed out, the symptoms of bronchopneumonia, in children in particular, vary to an extraordinary degree with the primary illness from which the patient has been suffering. Thus, in whooping-cough the onset of the disease is often so insidious as to be easily overlooked because the rales are mistaken for those of the usual mild bronchitis. In other instances the symptoms may be those of tuberculous or acute meningitis, particularly in rachitic infants.

Not rarely an acute diarrhœa may be present, the stools being green and containing much mucus, some of which is due to a coincident gastrointestinal catarrh, and some of it being from the bronchial tubes, for a child rarely expectorates, and usually swallows what he coughs up from his chest. Because of the associated indigestion there may be vomiting and distention of the abdomen by gas, which factors all aid in increasing the adynamia, and in interfering with the cardiac and pulmonary activity. These facts emphasize a fact too frequently overlooked, namely, that the condition of the lungs should always be carefully investigated in all cases which present signs of illness elsewhere, since it may be found, to the physician's surprise, that the pulmonary condition is not rarely the primary underlying cause.

Prognosis.—The prognosis of bronchopneumonia varies very greatly with the underlying cause of the disease and with the age of the patient. In young infants it is an exceedingly fatal malady, whether it is primary or secondary, and during the first year of life, if the disease is well marked, the prognosis is always unfavorable. The favorableness of the prognosis increases with each year of age. Another important factor in the prognosis of these cases is the general vitality of the patient. Children who are naturally strong and healthy, and are provided with good air and sunshine, have a better opportunity than those who live in poorly constructed dwellings with bad ventilation, and whose primary vitality is necessarily limited. This question of the vitality of the patient is a most important factor from a prognostic standpoint, and therefore if the child has been much weakened and devitalized by prolonged illness, or has had its heart seriously weakened by the prolonged strain of severe whooping-cough, the outlook is much less favorable than if the disease attacks the child who is suffering from a mild attack of measles.

The mortality of bronchopneumonia in very young children in private practice is probably about 30 per cent.; whereas in asylum practice, where it is impossible to provide them with the same amount of fresh air and careful nursing, and where the health is often previously impaired, it is not infrequently as high as 65 or 70 per cent. While it is true that poorly nourished, rachitic children are very apt to fall victims to the disease, it is also a fact that well-nourished, stout, fat children sometimes have marked difficulty in surviving its attack; and this is particularly true if they are "condensed-milk babies," for such children usually have low vital resistance. The complicating maladies, as, for example, active diarrhœa and indigestion or vomiting, of course make the prognosis very uncertain, and if they resist treatment are still more cause for anxiety.

Treatment.—In the treatment of bronchopneumonia it is of the greatest importance that the child should be in a well-ventilated room which receives as much sunshine as possible, for bronchopneumonia is essentially a disease of bad ventilation. The temperature of the room should be kept constant, and every care should be exercised that it is not damp. If possible, it should be heated by a stove, or by an open fire, rather than by furnace-heated air, and if it is necessary to heat it by means of a furnace, care should be taken that the air of the room should not be allowed to become unduly dry. This may be prevented by having the air from the furnace flue pass over the surface of a pan of water, and if the air is very hot and thoroughly dried it is better to set free in the air of the room a certain amount of steam from a tea-kettle, a pan of boiling water, or by occasionally immersing a large piece of quicklime in a bucket of water.

There can be no doubt that the influence of dusty, impure, or dry air upon the bronchial mucous membrane in cases of this disease is most deleterious, and I believe that in many instances much better results can be obtained if it is possible to place the child in a bronchitis tent, or to provide the air of the room with a sufficient degree of moisture to make the apartment the equivalent of a bronchitis tent. This can readily be accomplished in the way just suggested, or by the use of what is known as a "croup kettle," which continually sets free a small quantity of steam. To the water which is placed in the croup kettle 1 or 2 grains of menthol may be added every two or three hours, and in some instances, for their soothing influence, a few drops of oil of eucalyptus and compound tincture of benzoin may be so employed. As far as possible the patient should be kept quiet in bed, or, in the case of little children, should be moved as little as is consistent with comfort; but if the child is very ill, it should not be allowed to lie in one posture hour after hour, but occasionally be changed, lest hypostatic congestion occur. Easily digested, nutritious food should be given in small quantities every two hours. No medicine which may disturb digestion should be given to a child in its food.

In the way of external applications to the chest, the child's back, sides, and front may be rubbed with a mixture of a teaspoonful of turpentine and three tablespoonfuls of sweet oil. In other instances a weak ammonia liniment may be used, or in still other cases oil of amber, in the strength of a teaspoonful to two tablespoonfuls of sweet oil. These methods of treatment provide sufficient counterirritation and do not maintain the febrile temperature as do the poultice or cotton jacket, both of which forms of application have now deservedly gone out of use, as it is inconceivable that they can favorably affect the lesion in the lung, and they certainly increase the discomfort, the fever, and the irritation of the child's nervous system.

Stimulants are not needed in all cases of bronchopneumonia, but are used wisely in a larger proportion of patients than in those who suffer from the croupous variety of the disease, because bronchopneumonia, as has already been pointed out, usually attacks the feeble and therefore those who commonly need stimulation. The quantity of stimulant which is given varies of course with the feebleness of the heart sounds, the condition of arterial tension, and the degree of general nervous prostration. One of the best stimulants which can be used is the carbonate of ammonium in the dose of 2 or 3 grains every three or four hours to a child of a year or two, usually giving it in the syrup of acacia and water. Carbonate of ammonium, however, acts best when it is given for comparatively short periods of time, and for a constant stimulant during the greater portion of the disease it is probable that brandy occupies the first place. Care should be exercised that the brandy is at least five years old, and that it is as bland as possible. It should be given very well diluted by water, and a child of a year may take as much as half an ounce to an ounce in twenty-four hours with advantage, 30 drops being given every two or three hours.

As a rapidly acting diffusible stimulant to meet critical periods of depression Hoffmann's anodyne in the dose of 5, 10, or 15 drops may be employed in young children. In other instances $\frac{1}{150}$ grain of strychnine may be used, or a larger dose than this, it being always borne in mind that the nervous system of a child is exceedingly susceptible to this drug. Like the carbonate of ammonium, strychnine is only to be used when it is necessary to bridge an exceedingly critical period. If the dose of strychnine is to be repeated, $\frac{1}{300}$ of a grain is a sufficiently large amount. This quantity may be given twice, thrice, or four times in twenty-four hours, but, as a rule, it is unwise to continue its use for a longer period than this.

Where the quantity of bronchial secretion is considerable, particularly in many cases of suffocative bronchitis, a critical period may be weathered by the use of small doses of atropine; from $\frac{1}{1000}$ to $\frac{1}{500}$ of a grain may be given every two or three hours by the mouth, or, if need be, $\frac{1}{500}$ to $\frac{1}{300}$ may be given hypodermically, if there seems to be danger of the child drowning in its own secretions. Oxygen may be taken by inhalation in some cases with advantage.

The use of antipyretic drugs is to be absolutely condemned. They are even more dangerous in this disease than in croupous pneumonia. If the temperature is so high as to be dangerous in itself, it may be controlled by cool or tepid spongings, with gentle friction; by the use of cool cloths to the forehead, or an ice-bag applied to the head if cerebral symptoms are marked. Sometimes, too, placing the child in a tepid bath for a few moments will act as an excellent antipyretic.

When the symptoms of respiratory oppression are marked and the fever is high, it is often advantageous at a critical period to dip the child alternately in cool and hot water, the water being hot enough to produce distinct counterirritation on the skin, and, reflexly, to arouse the dormant nervous system. Under these circumstances the child often rallies, takes deep inspirations, dislodges the mucus which is otherwise obstructing its breathing, and at the same time has a reduction in its temperature. Such an alternate hot and cold plunge bath should only be resorted to when conditions are desperate, and should not be repeated too frequently. A tepid bath, the patient being immersed or simply sponged, will also very frequently allay restlessness and permit quiet sleep.

In the protracted cases it is exceedingly important that pure air and good food should be provided. Not infrequently the child which fails to improve in the city may, when carried in its nurse's arms to the sea-shore or the mountains, change for the better to a remarkable degree within a very short period of time. This is particularly true if the weather is oppressively hot. Such patients also may be benefited in some instances, particularly during the winter months, by cod-liver oil inunctions, and, if the digestion will stand it, by the administration of small quantities of cod-liver oil or the syrup of iodide of iron by the mouth. Sometimes such patients are also greatly benefited by the administration of the hypophosphites.

During the acute stage of bronchopneumonia there is little use in employing the ordinary expectorants. During the stage of resolution, if the secretion is profuse, small doses, such as 1 or 2 grains of chloride of ammonium, with fluid extract of licorice and water, may be given twice or thrice a day. Rarely in young children is there much expectoration, either in the sense of expelling mucus from the mouth or coughing it up into the pharynx. The younger the child the less chance there is of freeing its bronchial tubes of secretion by coughing, and care should always be taken that the administration of an expectorant, which is not of very great importance, does not disorder the digestion, which is of far greater importance to the maintenance of the child's health and strength than any medicine can be.

The bronchopneumonia of adults usually follows asthma or the inspiration of irritant materials, and must be treated in much the same manner as that just described for bronchopneumonia in children, except that the doses should be larger in proportion to the age and size of the individual. In nearly all cases active stimulation is required, and digitalis and strychnine are particularly useful. Counterirritation, freely applied to the chest, seems to be of advantage in some instances, but here again the cotton jacket or the poultice ought not to be resorted to, as they simply oppress the patient and do little good.

METASTATIC PNEUMONIA.

Definition.—By the term metastatic pneumonia is meant a condition of consolidation of part of one lung, or more rarely parts of both lungs, as the result of the plugging of one or more of the pulmonary vessels by an embolus which is of septic origin.

Etiology and Pathology.—As elsewhere, emboli reaching the lung may be (1) simple or bland, (2) septic or infective; either of these may be massive or small. A large mass thrown into the pulmonary artery at once arrests the flow of blood, the patient gives a few gasps, possibly has a convulsion, or at least convulsive movements, and dies. Smaller emboli, if numerous (an embolic shower), may induce similar phenomena. The simple or bland embolus occludes one or more vessels, and leads to the formation of a hemorrhagic infarct. These irregularly shaped or conical areas vary in size, depending upon the magnitude of the occluded vessel and the efficiency of the collateral circulation. They may be central or peripheral, massive or small, single or multiple.

The question of autochthonous embolism is of pathological rather than clinical interest.

The affected area is airless, denser than the uninvolved pulmonary tissues, and near the centre it is dark purple or almost black in recent infarcts, black or brownish-black in older areas, and it may be surrounded by a zone of reactionary inflammation. If the lesion is peripheral the indurated area rises above the level of the pleura and is frequently covered by a delicate stratum of fibrin. Certain pleurisies, particularly those following operations, have been attributed to pulmonary infarction. Histologically such areas when recent show air vesicles occupied by blood cells and fibrin and more or less interstitial extravasation. Later the erythrocytes are fragmented, the leukocytes increased, phagocytes abundant, and evident reparative processes in progress.

The sputum is more or less blood-stained, and when the infarcts are large or numerous it may be intensely so. In fat embolism, such as may accompany fractures of the long bones, oil globules may be demonstrable in the sputum.

Whether the process arises to the dignity of a pneumonia depends, of course, upon the amount of accompanying inflammation. It is evident that a few small infarcts irregularly distributed may give rise to no symptoms because of the insignificant lesions induced; or, on the other hand, large, or multiple, areas may be accompanied by evident lung symptoms. Whether inflammation be marked or slight is so largely dependent upon the presence of infection that in the absence of bacteria the name metastatic pneumonia is scarcely applicable.

Embolism due to fragments of neoplasms entering the lungs usually escapes notice until the proliferating cells give rise to metastatic tumors.

The most important and gravest type of metastatic pneumonia is that seen in pyæmia.

During the course of a septic process in any part of the body, even though it may be so minute as to escape notice unless carefully sought, it is possible for a small clot (embolus) infected by micro-organisms to enter the circulation and, being carried to the lung, to plug one of the vessels. The difference between the infarct resulting from an ordinary embolus and the lesion ensuing from one of septic origin is very marked, for in the latter condition there speedily develops an acute local process due to the rapid extension of the infection from the embolus. In this way the immediate neighborhood of the closed vessel becomes engorged; polymorphonuclear leukocytes accumulate in the infected area, which rapidly undergoes liquefaction, necrosis, and an abscess is formed. As such emboli are rarely solitary, multiple foci are prone to develop; these may, by extension, become confluent or successive embolic showers may cause closely approximated lesions of different ages.

As the quantity of infective material and its distribution constantly varies, the anatomical result of such conditions can rarely be the same in any two cases. There may be a single area of infection, or the lung may be riddled by abscesses, the "pyæmic pneumonia" of old writers.

The area of solidification in the lung may resemble the patchy state seen in bronchopneumonia or the hepatized appearance of croupous pneumonia. There is, however, this important difference in the further progress of the local lesions between the two diseases named and that under discussion, for in metastatic pneumonia the inflammatory process usually goes on to suppuration, the entire infected area becoming crowded with pus cells and cocci, the walls of the vesicles and the connective tissue of the lung breaking down instead of remaining intact as in most cases of ordinary pneumonia. As a result we find one or more abscesses of the lung which may rupture into a bronchus, into the pleural cavity, or even through the diaphragm, and are practically always accompanied by marked septic fever.

The pleura rarely escapes, and empyema, in patients surviving sufficiently long, is not uncommon. As the abscesses open into the bronchi and eroded vessels give way, pulmonary hemorrhage is prone to occur. Large areas of pulmonary tissue may undergo necrosis and further complicate the case by the addition of pulmonary gangrene.

Symptoms.—The symptoms of metastatic or septic pneumonia present so little that is characteristic that they are often overlooked. This is because the lesion in the lung is secondary to some inflammatory process already present, which is responsible for much of the fever and other signs of an infection. In the midst of these symptoms, if they are severe, the slight exacerbation produced by the embolism is not recognized. It is only in those cases in which the area of the lung involved is very considerable that pulmonary signs are forced upon the physician, projecting themselves, as it were, above those already present. When the pulmonary symptoms are marked they so closely resemble those of an acute pneumonia that not infrequently the diagnosis of an intercurrent pneumonia is made, only to be modified when *repeated chills*, *sweats*, and a temperature chart indicative of sepsis show that the process in the lung is septic. (See Pyæmia.)

When the embolus is a large one and plugs the pulmonary artery at its bifurcation, death suddenly ensues.

The physical signs of metastatic pneumonia are practically identical in the early stage of the affection with those of bronchopneumonia or croupous pneumonia, for the consolidated portion of the lung produces dulness on percussion, bronchial breathing, and increased vocal fremitus and resonance. Later, when the consolidated area breaks down and begins to undergo suppuration, the physical signs may be those met with in beginning resolution in ordinary croupous pneumonia.

Prognosis.—The prognosis in metastatic pneumonia is bad because it is a septic process and also because it is a serious complication added to a process which is already more or less severe. It usually ends in abscess or gangrene, and these affections, particularly the latter, are fatal in the great majority of cases. In the rare instances in which recovery takes place the health of the patient is, as a rule, permanently impaired. I have, however, seen two instances followed by abscess end in complete restoration to health.

Treatment.—There is no specific treatment of this condition unless it be known that the streptococcus is the cause of the infection, in which case antistreptococcic serum may be employed. Even in these instances, however, it cannot do much good because, after the pulmonary vessel is mechanically closed by an embolus, no treatment can bring about its relief. The most that the serum can do is to limit the degree of general toxæmia.

Ordinarily the treatment must consist in the use of as much easily assimilated food as the patient can take without disordering his digestion, the administration of proper quantities of stimulants, and the careful control of such symptoms as may become excessive, as, for example, the reduction of high temperature, if it is persistent.

PNEUMONOCONIOSIS.

Definition.—This term is applied to a state of the lungs in which, by reason of exposure to, and inhalation of, various kinds of dust a deposit of the foreign body takes place in the pulmonary tissues and produces secondary changes. When the individual is exposed to coal-dust in sufficient amount and for a long-enough period to cause its accumulation in the lung, the state of the lung is called "*anthracosis;*" when the dust is derived from the grinding of iron or steel, it is called "*siderosis;*" when the dust arises from stones, the term "*lithiasis*" or "*chalicosis*" is employed. Still another type of foreign body capable of causing pneumonoconiosis affects those who work in large textile industries and shoddy mills. The minute particles of wool and cotton, and of the clay used for "sizing," often cause bronchitis and favor the occurrence of phthisis. The dust can often be found in the sputum of such patients. Reference to this type of the disease is made in the author's *Fiske Fund Prize Essay* for 1885. Still another form of exposure gives rise to "grain-shovellers' disease," and to "potters' rot."

Etiology.—Under ordinary circumstances the respiratory tract is able to get rid of minute foreign bodies which may enter it. This is accomplished by the arrest of the dust in the nasal and pharyngeal mucus, and by the action of ciliated epithelium lining the larynx, trachea, and bronchial tubes, which continually passes along toward the mouth for expectoration any dust particles which may enter. If these protective measures are insufficient because of the great quantity of dust inhaled, or where after a time this ciliated epithelium is destroyed, some of the particles are carried through the mucous membrane and are arrested in the nearby connective tissue;

but if the amount of dust is so large that even this third barrier is passed, then the dust particles are taken up by the lymphatics and carried to the bronchial lymph nodes, or to the interlobular pulmonary septa under the visceral layer of the pleura, or to the substernal lymph glands, where they are deposited and remain fixed. Very rarely the fine particles may enter the circulation and be deposited in the liver and spleen, as in a case reported by Welch, or they may be even excreted in the urine.

Pathology.—Up to this stage these results may possess no pathological significance, but in some instances the presence of large quantities of these foreign bodies produce a low-grade inflammatory process in the lung tissues which results in overgrowth of connective tissue; that is, a chronic productive interstitial pneumonia or pulmonary sclerosis. Occurring independently of the interstitial change, or associated with it, there is quite constantly a subacute or chronic bronchitis and emphysema, and finally areas of softening take place, in the fibroid portions of the lungs, which are small in size and filled with dust-stained fluid. Sometimes these communicate with a bronchial tube and may then become infected and ulcerate. These ulcerated patches or spots of softening may or may not be due to infection by the *Bacillus tuberculosis*. It is by this process that we have established "miners' phthisis" or "grindstone consumption" and "gold-dust complaint" of the lung. So common is this condition in Sheffield, England, that it has been called "knife-grinders' rot."

Symptoms.—As a rule symptoms of pulmonary trouble do not come on in serious form until the individual has been exposed for some months or years, when *chronic cough*, *dyspnœa*, and *loss of flesh* call attention to the insidious changes in the lungs. A macroscopic or, when this fails, a microscopic examination will usually reveal the dust in the sputum, and the history of the case renders the diagnosis easy.

Prognosis.—In an investigation carried on at Solingen, Germany, by Moritz, it was found that there were no fork-grinders above forty-five years of age and no sword-grinders above fifty. Of the total number of knife-grinders employed, only 5.5 per cent. were over forty years of age. Of the scissors-grinders there were 8.4 per cent. above forty. The fork- and sword-grinders work with dry grinding stones, while the knife- and scissors-grinders work with grinding stones which are constantly kept moist. The relatively greater number of scissors-grinders who live to be over forty is explained by the fact that the knife-grinders sit closer to their machines than the scissors-grinders, and thus inhale more dust.

Peabody, in some investigations made at Sheffield, England, found that the average period of knife-grinders who are able to continue their work is thirteen years. In South Africa, Fox states that the duration of life in gold mines where there is much dust from blasting is only four years. Out of 1377 rock-drill miners, 225, or 16.34 per cent., died in two and a half years.

Treatment.—The treatment is removal from exposure and the use of the medicinal measures advised in the articles on Chronic Bronchitis and Emphysema. It is the duty of all employers of labor in dusty places to provide free ventilation, both to dissipate the dust and to diminish the chance of tuberculous infection. In many industries the employees should use moist respirators to catch the dust in the respired air. Moist or wet grinding should be used instead of dry grinding to prevent dust, and workmen known to be tuberculous should be excluded from the workshop.

EMPHYSEMA OF THE LUNGS.

Definition.—The term emphysema, as applied to disease of the lung, signifies a condition in which the air content of the organ, in a large or small area, is in excess of the normal. Systematic writers ordinarily make it include (1) essential, hypertrophic or large-lunged emphysema; (2) atrophic or senile emphysema; (3) compensatory emphysema, a form of vesicular overdistention due to inexpansion, or absence, of pulmonary parenchyma in some juxtaposed or, less commonly, a distant area, and (4) a form of what occurring elsewhere is ordinarily termed "surgical emphysema," but in the lung is called "interstitial," "interlobular," or "intervesicular"—names that indicate the location of the air and differentiate the condition from the first named states or those forms in which the abnormal air content is intravesicular

Emphysema has also been divided into an acute and chronic form. In point of time the interstitial is always acute, the essential and atrophic always chronic, while the compensatory may be either. Some writers apply the term "acute" to that condition in which rapid overdistention of relatively large areas occurs as a result of violent inspiratory efforts, or obstructed expiration, such as occurs in cardiac asthma, bronchial obstruction, and allied conditions.

Briefly described, vesicular emphysema is a state in which there is atrophy of the septa between the air cells so that a number of vesicles coalesce. As a result we find in the lung many small, bladder-like spaces containing air. Associated with this minute change the entire lung increases in bulk and the thoracic cavity is usually much increased in all its diameters, especially the anteroposterior and the vertical, producing the so-called "barrel-shaped chest."

Etiology.—Much difference of opinion exists as to the primary cause of pulmonary emphysema. It is universally acknowledged that the condition develops as a result of inadequacy, either congenital or acquired, of the supporting elastic tissue between the vesicles; but one school of pathologists maintains that the giving way of the vesicular walls depends upon mechanical stress, while another school asserts that such a result ensues only when the normal support is removed through failure of nutrition in these parts so that atrophy results. The author is convinced of the correctness of the latter view, namely, that the coalescence of the vesicles takes place only after the elastic connective tissue has become wasted as the result of impaired circulatory supply. It is perfectly true that great pulmonary stress tends to produce emphysema of the lung, but it only produces this state when the connective tissue is unable to provide proper vesicular support.

Probably in a large proportion of cases the tendency is hereditary, the defect is congenital, and the tissues succumb as soon as any great stress is put upon them. If this primary nutritional feebleness be admitted as the fundamental cause of the condition, it is easy to understand how it is that persons so affected fall victims to emphysema when attacked by spasmodic asthma, or when following occupations which produce pulmonary stress, and it also makes manifest why it is that other persons exposed to equally severe exciting causes escape.

Frequency.—The frequency of true pulmonary emphysema is difficult to determine because many of the mild cases are overlooked, and patients do not present themselves for treatment until the disease is far advanced. To show how widely statistics may vary according to the method of their collection, it is interesting to note that Lebert states that pulmonary emphysema forms about 5 per cent. of all diseases, while Virchow found in nearly 200,000 cases, admitted to the Charité in Berlin, that the percentage of emphysema was only 0.3, a result confirmed by West at St. Bartholomew's in London. The disease is met with three times as frequently in men as in women, probably because they are exposed to its secondary causes more constantly, and it occurs chiefly between the ages of thirty and sixty years. It occurs in children, but rarely before they are ten years of age, although cases as young as two years of age have been recorded.

Pathology and Morbid Anatomy.—It has already been stated that the essential characteristic of pulmonary emphysema is the wasting of the interalveolar tissues so that coalescence of the vesicles takes place. As a result small, bladder-like spaces are formed, the lung loses its elasticity, and so fails to expel the air on expiration, with the result that the quantity of residual air is greatly increased. This results in dyspnœa in two ways: first, there is an impaired circulation of fresh air in the lung, and, second, there is a decrease in the area of the vesicular tissues, so that a much smaller surface is afforded for the absorption of oxygen.

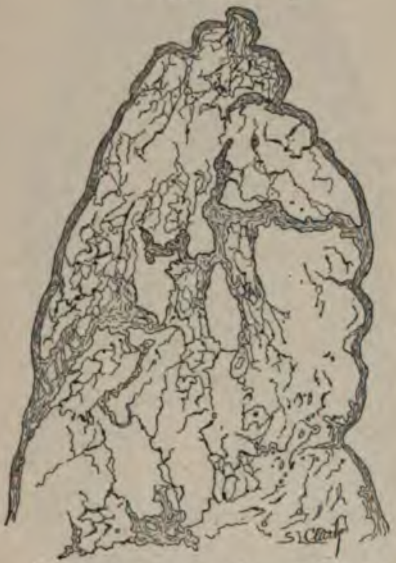
When the thorax of a case of essential emphysema is opened the lungs do not retract as do healthy lungs. Indeed, they may project into the opening which has been made. The left lung extends so far forward as to cover the heart, and the right lung may overlap the edge of the left. Often the epiclavicular spaces are distended by lung tissue, and if the disease be marked the convexity of the diaphragm is reduced, with consequent displacement downward of the adjacent abdominal viscera, particularly the liver.

The chest is changed in appearance because with the increase in the size of the lungs the ribs become more horizontal and the intercostal spaces more bulging, the sternum and costal cartilages are projected forward, and the normal dorsal curvature of the spine is exaggerated.

When the lungs are removed from the chest it is found that they possess four peculiarities aside from their great size: they are pale gray in color, unusually free from blood, dry, and when pressed between the fingers they lack the crepitation met with in normal lung-tissue. A noteworthy change is the presence of dilated pouches or bladder-like protuberances on the surface of the lung, and particularly at its margins. If the lung be cut, somewhat smaller spaces will be found scattered through it. It is noteworthy that in emphysema these open spaces are surrounded by thin walls which readily collapse, whereas the sacculations of saccular bronchiectasis are surrounded by areas of thickening and inflammatory change.

With the coalescence of the air spaces the capillaries which usually pass between the vesicular walls disappear, and this in turn diminishes the number of pathways by which the blood can pass from the right to the left side of the heart. As a result three chief circulatory changes ensue. Some of the blood finds its way by large anastomotic channels from one side to the other, and so is imperfectly oxidized. The increased obstruction to the flow results in distention and arteriosclerotic changes in the pulmonary artery, in dilatation, with more or less hypertrophy, of the right ventricle and finally in dilatation of the right auricle. Eventually, when the pathological process is far advanced we find that the liver is greatly engorged with blood, ascites may develop, the cardiac failure rapidly progresses, and death results from the various sequences of the primary lesions.

FIG. 55



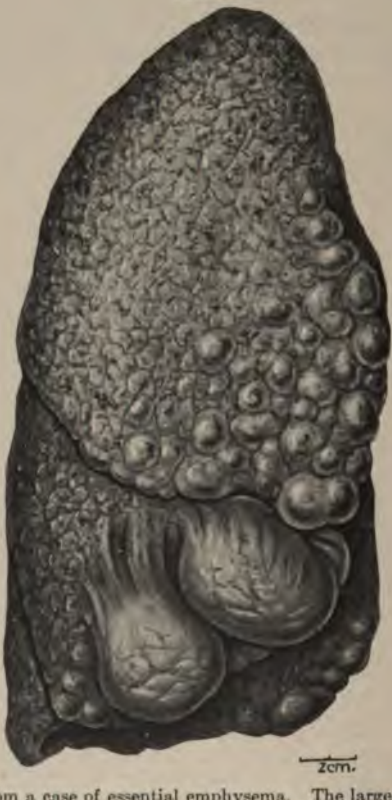
Section of anterior margin of the lung from a case of essential emphysema, showing the wasting and absorption of the vesicular walls.

Associated with emphysema there is usually more or less well-developed chronic bronchitis.

Symptoms.—The symptoms of emphysema may be best divided into the objective, or those that can be seen by the physician, and the subjective, or those described by the patient.

PHYSICAL SIGNS.—The most noteworthy objective signs are the *increase in the diameter of the chest*, so that the anteroposterior diameter equals the lateral; the *fulness or bulging of the intercostal spaces;* the *impaired respiratory movement of the thorax*, which may seem quite fixed; the well-filled or *distended cervical vessels,* and the presence in the epigastrium of the apex beat of the heart. If the case is severe we see in addition to these signs *pulsations of the jugular veins, labored breathing, cyanosis of the lips, fulness of the abdomen,* due to the displaced and engorged liver, and the accumulation of fluid in the peritoneum. Not infrequently inspection of the upper part of the epigastrium reveals a network of enlarged capillaries in the skin. These are the chief signs on *inspection.*

FIG. 56



Lung, anterior aspect, from a case of essential emphysema. The large bullæ on the anterior margin of the middle lobe are nearly two centimetres in diameter. Smaller vesicles are present on the anterior margin of the upper lobe and along the diaphragmatic border of the lower lobe. The apex is but slightly involved, but in some cases it is markedly affected.

On further careful physical examination we find on *palpation* that the apex beat cannot be felt at the normal area near the nipple because it is displaced and covered by the enlarged lung. The lower margin of the liver may be felt as low as the navel. Palpation of the chest while the patient speaks reveals a marked *decrease* in vocal fremitus. *Percussion* gives a high-pitched resonant note all over the chest, particularly over the upper lobes; reveals a decrease of the normal area of cardiac dulness; shows the liver to be as low as palpation indicated it to be, and gives flatness in the flanks and in the suprapubic area if ascites is present. *Auscultation* reveals a feeble vesicular murmur, marked prolongation of expiration because of the inelastic state of the lung, and sometimes there can be heard rales, which are due to the associated bronchitis. A curious crackling sound, the cause of which is not certain, is also heard sometimes. It is not due to pleurisy and is probably produced by the air in the bladder-like dilatations in the margins of the lungs. This sound is usually best heard at the apices.

SUBJECTIVE SIGNS.—The symptoms from which the patient complains are chiefly those connected with respiration. The *shortness of breath* varies greatly in different cases. In some it is constant. In others it is only developed when exercise is taken, and the difference in its degree on exertion varies widely in different individuals. Often dyspnœa is only felt on warm, oppressive, or humid days, while in other cases any exertion whatever produces such severe dyspnœa that the patient is forced to rest. This dyspnœa, as already stated, depends upon deficient oxygenation of the blood, upon the interference with the action of the right side of the heart, and upon the inability of patient to take fresh air into his lungs in large quantity because of the excess of residual air which is present.

The *cough* in some cases is so constant as to greatly annoy the patient. In other instances it is almost entirely absent. The development of this symptom largely depends upon the degree of bronchitis which is associated with the emphysematous change. If marked bronchial irritation is present, the cough is not only annoying because of its persistency, but also exhausts the patient, and aids in the dilatation and fatigue of the right side of the heart. The sputum which results from the cough varies in quantity with the severity of the bronchitis which is present, and is not peculiar in appearance unless by chance the patient is also a sufferer from asthma, when the characteristics of asthmatic sputum may be manifest. The digestive disorders sometimes complained of by the patient depend chiefly upon the impairment of the circulation in the liver, stomach, and intestines, produced by the secondary cardiac lesions. Sometimes, too, the urine is scanty, owing to congestion of the kidneys from the same cause.

Diagnosis—From what has just been said of the symptoms and typical signs of pulmonary emphysema it is evident that the diagnosis is not difficult. Indeed, in a well-developed case there is probably no pulmonary condition so easily recognized. The bilateral increase in the size of the chest, the narrowing of the intercostal spaces, the dyspnœa, the cyanosis, the prolongation of expiration, the hyper resonance on percussion are all to be noted in forming a positive conclusion as to the character of a case. It is not necessary for the diagnosis of emphysema that deformity of the chest be present Sometimes a marked degree of pulmonary change exists without any change in the shape of the thorax.

Emphysema of one lung is practically never seen, and therefore pneumothorax can be easily separated from emphysema.

Prognosis.—The prognosis of emphysema is always unfavorable; at least, so far as complete recovery is concerned. In many cases, however, the progress of the disease is so slow that the patient may live for years with a fair degree of comfort. Indeed, in some instances the pathological process becomes stationary. Patients with well-developed emphysema are, however, rarely fortunate enough to develop this arrest of the disease, and equally rarely live until advanced old age, usually because with advancing years the muscle fibre of the right side of the heart becomes less and less able to stand the strain which is thrown upon it.

It is vitally important, so far as prognosis is concerned, for patients suffering from pulmonary emphysema, to avoid exposure to sudden changes of temperature; for such changes may produce a severe bronchitis or pneumonia, conditions which the patient is ill able to withstand. The presence of a persistent chronic bronchitis renders the prognosis more grave than if this complication does not exist. Death rarely comes on suddenly in these patients, but slowly, as a result of constantly increasing circulatory failure. Lebert asserts that one-third of these cases die from cardiac dropsy, and the rest from pulmonary congestion and gradual feebleness, with slow suffocation, increasing cyanosis, and constantly developing bronchitis.

Treatment.—The treatment of pulmonary emphysema is, unfortunately, very limited. There is no curative treatment. The most that the physician can do is to improve the condition of the circulation and the nutrition of the patient, and to prevent him from throwing severe strain upon his pulmonary tissues and his circulatory apparatus. Where the patient follows an occupation which is manifestly injurious, he must be advised to give it up, and, for that matter, to avoid all violent muscular effort which will throw a strain upon his heart and lungs. Incipient attacks of acute bronchitis should be treated at the earliest possible moment, and, if chronic bronchitis is present, the remedies which are commonly given for that disorder should be employed, care being taken, however, that no drug is given which tends, on the one hand, to act as a circulatory depressant, and, on the other, to promote too free bronchial secretion, for it must always be borne in mind that drugs of this character may precipitate an attack of profuse bronchial secretion, in which the patient may drown in his own fluids.

Many of these patients will be benefited by the administration of 5 grains of carbonate of ammonium and 5 grains of chloride of ammonium given in a cachet, or capsule, or in fluid extract of licorice and water, three or four times a day. In other instances, if the bronchitis is chronic and well marked, creosote or guaiacol may be used; but care must be exercised that they do not disorder the stomach. If the secretion is thick and tenacious, iodide of ammonium, or iodide of sodium, in the dose of 5 grains three times a day, is useful, care being taken, however, that the administration of this remedy does not produce too free bronchial secretion. It must also be borne in mind that bronchitis complicating emphysema is not infrequently the result of impaired cardiac action, and, therefore, that the best treatment for the bronchitis is the administration of cardiac tonics, such as small doses of digitalis, 3 to 5 minims twice or thrice a day, or the tincture of strophanthus, or, in other cases, the administration, for a few days, of moderately large doses of strychnine or nux vomica.

When the patient's means permit him, it is important that he should avoid extreme climatic changes. High altitudes are, of course, not only disadvantageous, but even dangerous to patients suffering from pulmonary emphysema, because of the dyspnœa which such altitudes produce and because of the strain which is thrown upon the dilated right heart.

In cases of emphysema suffering from an unusually severe attack of dyspnœa, with great congestion and engorgement of the venous system, it is often advantageous to resort to venesection, removing as much as 20 to 30 ounces of blood; but it is manifest that this method of treatment can only be resorted to on a few occasions, and when the symptoms of dilatation and distention of the right side of the heart and of the liver are very well developed. Sometimes in these cases, if there is evidence of hypostatic congestion of the lungs, the application of wet or dry cups, posteriorly, near the bases is advantageous.

For many years various text-books have recommended the employment of the iodides in their various forms in the treatment of pulmonary emphysema, with the idea that they distinctly modify the pathological process going on in the lungs, and to a certain extent arrest the destruction of the elastic tissue which, by its failure, results in the coalescence of the vesicles. It must be manifest that even that wonderful drug, iodide of potassium, must be quite useless for this purpose in many instances. Any advantage which follows its employment probably depends upon its influence upon the associated bronchitis, or upon the effect which it produces upon the vascular system by diminishing the tendency to atheromatous change, and by reducing high arterial tension if it is present, and so relieving the heart of unnecessary burden. Still another advantage in the iodides may be that in some cases they act as a diuretic and so help to relieve the tissues of an undue quantity of fluid if dropsy be threatened.

Compensatory or Acute Emphysema.—This is an unfortunate use of the word emphysema, as the condition is not a true emphysema, but simply an abnormal distention of each individual air vesicle by active efforts at forced respiration, so that the entire lung may be increased in size and the areas of pulmonary resonance greatly increased. Usually in this state some high-pitched rales are audible in the chest. The condition may be seen in cases which have suffered from stridor due to laryngeal obstruction, or more commonly in those who are recovering from an acute asthmatic attack. It is also found in those parts of the lungs which have endeavored to compensate for other parts affected, as, for example, by pneumonia.

Interstitial Emphysema.—In interstitial emphysema the pathological condition is not like that of ordinary pulmonary emphysema, for the lung is riddled with tiny globules of air which find their way between the lobules and underneath the visceral layer of the pleura, where they may form quite large blebs. The condition arises whenever air escapes into the pulmonary tissues, as after tracheotomy, when it extends down along the trachea into the lung itself; fractures of the ribs with puncture of the lung; other wounds of the lung; rupture of air vesicles by great thoracic compression, as in sand crushes, even without injury to the skeleton, and occasionally results from violent abnormal respiratory action, as in whooping-cough, strangling, and sneezing. It has been observed after severe convulsions in epileptics and eclamptics.

Small-lunged Emphysema.—Small-lunged emphysema is sometimes called senile or atrophic emphysema, or senile atrophy of the lung. It resembles ordinary emphysema, as just described, in the fact that there is a wasting of the walls of the air vesicles, so that several vesicles form a larger cavity; but instead of the lung being larger and more voluminous than normal, it is shrunken and small, so that the heart is uncovered, the diaphragm raised in well-marked cases, and the whole thorax distinctly decreased in size. The expansion of the vesicles, as in large-lunged emphysema, is most marked at the apices and the edges of the lung. Inspection of the chest in such a case shows the intercostal spaces obliterated by the drawing together of the ribs, while the epiclavicular and episternal spaces are exaggerated and the respiratory movement is feeble and very shallow. On percussion the chest is found to be hyperresonant everywhere, but there is a great increase in the area of cardiac dulness, due to the retraction of the lung. On auscultation little that is abnormal is heard, save that expiration may be prolonged.

Except there be an associated bronchitis, the patient with this type of emphysema rarely suffers from much inconvenience as a result of the pulmonary disease, and life is not materially shortened.

Treatment.—There is no curative treatment for this type. The physician can only order rest, good food, proper clothing, and the avoidance of exposure.

GANGRENE OF THE LUNG.

Etiology.—This condition arises in individuals whose general vitality is greatly impaired by some primary disease, with the result that various micro-organisms, putrefactive and otherwise, produce death of part of the pulmonary parenchyma, and so a slough is formed. Manifestly, the causes of gangrene and abscess must be nearly related, and why gangrene rather than abscess should develop in any particular case is difficult to determine. Infarction of the lung, or pulmonary hemorrhage, may, by affording a nidus for the development of putrefactive germs, result in this state, and so may croupous pneumonia; yet it is a curious fact that bronchopneumonia, which is often due to profound debility and secondary infection, rarely so results. Equally curious it is to note that pulmonary tuberculosis in all its forms is rarely complicated in this manner.

The most common cause of pulmonary gangrene is embolism and thrombosis, after this croupous pneumonia, and, thirdly, injuries to the lung through the chest wall, as in gunshot injuries. It may also arise from foreign bodies in the bronchi. It may also be due to pressure produced by an aneurysm, or tumor, or by an extension of an infective process to the lung from the œsophagus, pleura, vertebræ, mediastinum, or ribs. It may also follow the inspiration of particles of food. Rarely it is due to pressure of an aneurysm or to perforation of the œsophagus when that tube is affected by cancer.

Frequency.—Pulmonary gangrene most frequently attacks males in middle life—that is, from twenty to forty years of age—and is undoubtedly a very rare affection. In a large hospital service only a single case may be met with in many years.

Pathology and Morbid Anatomy.—No description of pulmonary gangrene is better than that given by Laennec, who, nevertheless, in an experience of twenty-four years, saw only 2 cases. He divides the condition into three stages: (1) that of early mortification, in which the pulmonary tissue is œdematous and of dark-brown or greenish hue, the sloughing area looking shreddy and water-soaked; (2) that of deliquescence or liquefaction, the part of the lung affected becoming still more soft and flabby; and (3) that of excavation or abscess formation, in which the lung undergoes the separation of the slough and the formation of a line of demarcation to limit the pathological process. At this line of separation a consolidation takes place, the sphacelus breaks down, and suppuration rapidly results in the coughing up of the dead tissues. As a matter of fact, it is incorrect to speak of a single sphacelus, for the cavity usually contains separate masses of shred-like tissue.

Finally the limiting wall may undergo fibroid contraction, as it does in abscess, and the area be more or less closed, a focus usually remaining, from which more or less foul pus is constantly discharged. In the majority of cases this reparative process does not occur, and the patient dies. Over the seat of the process the pleural membrane is usually thickened and may be covered by a fibrinopurulent exudate, while if the pleura be perforated a putrid empyema may develop. In such cases pyopneumothorax may also manifest itself. Extensive suppuration of the bronchial glands may occur.

Pulmonary gangrene affects the lower lobes oftener than the upper.

Symptoms.—The symptoms of pulmonary gangrene in the early stages are not very definite. They depend, to some extent, upon the severity of the lesions and upon the micro-organisms which produce it. The patient is *markedly prostrated*, the *heart's action* is *feeble* and rapid, the *skin leaky*, the *face anxious* and thin, and the *tongue dry* and coated. The *temperature* runs the typical course of hectic fever, and it is a noteworthy fact that the exhaustion seems out of proportion in its severity to the febrile movement. Sometimes these symptoms are ushered in with *severe chills*, which recur at irregular intervals. The *respirations are quickened*, and there may be *cough* and *expectoration*, but until the break-down goes on so far as to result in suppuration there may be but little material expectorated. If a cavity forms, the ordinary signs of excavation, with those of surrounding consolidation, may be developed upon auscultation and percussion.

One of the most characteristic symptoms of pulmonary gangrene is the odor of the patient's breath and of the materials which he expectorates. There is probably no discharge from the human body the odor of which is so penetrating and disgusting as is that of pulmonary gangrene. Not only does it render the patient disgusting to everyone who comes near him, but it penetrates every part of the room in which he exists, and often can be smelled throughout the whole house. On some days it is worse than others, but the variation of the quantity of expectoration does not necessarily mean a variation in its fetid character. The quantity of material which is expectorated does not give any very definite conception of the size of the lesion of the lungs. West quotes a case of Godlee and Williams in which the patient expectorated a quart daily, and yet the autopsy revealed a gangrenous cavity which was not large enough to contain more than an ounce of fluid.

The sputum is peculiar, in that on standing it separates into three layers. The upper layer is apt to be yellowish-green and opaque; the middle layer is opalescent and turbid, and resembles saliva when a considerable quantity is gathered in a glass. The lowest layer consists in a mass of greenish or brown-looking material, which contains considerable quantities of pus, altered red blood cells, and fragments of connective tissue. A careful microscopic examination of this sputum will show that it is filled with an immense number of micro-organisms, and crystals of leucin and tyrosin can be seen in large numbers. Various fatty acid crystals are also present. The sputum, at first alkaline, becomes acid, and seems to exercise a peculiar digestant or disintegrating influence upon the shreds of connective tissue which it contains.

The cough in a case of pulmonary gangrene varies greatly according to the amount of material which is expectorated, and also with the degree of bronchial irritation which coexists. Sometimes, after a prolonged spell of coughing, a considerable amount of material from the gangrenous area comes away in a gush. Sometimes, too, the fluid which is expectorated is distinctly blood-tinged, due to the ulceration of small bloodvessels in the part surrounding the affected part. Not only may free hæmoptysis develop, but septic emboli may be carried elsewhere, as, for example, to the brain or liver, and so cause secondary abscesses.

If by chance the patient swallows any of the sputum, septic diarrhœa may be established and the stools may also become excessively offensive. The degree of exhaustion gradually increases, the heart becomes more and more feeble, the patient more and more emaciated, and, finally, dies of asthenia.

Diagnosis.—The diagnosis between a moderate degree of pulmonary gangrene, pulmonary abscess, and bronchiectasis may be almost impossible, since, if bronchiectasis exists, the fetor of the sputum may be very marked. If the sputum under the microscope shows a large amount of connective tissue, the diagnosis is largely in favor of gangrene. The absence of tubercle bacilli in the sputum and the presence of the various bodies already named as appearing in this fluid will also aid in differentiation of the case. When the gangrene cavity is small, a positive antemortem diagnosis may not be possible, the more so because of the presence of acid-resisting bacilli, which may be mistaken by the novice for tubercle bacilli.

Treatment.—The treatment of pulmonary gangrene is not promising. It is the duty of the physician to maintain the strength of the patient, as far as possible, by the administration of nutritious food given at frequent intervals, in small quantities, so that the digestion will not be overloaded; to give stimulants, as alcohol; and occasionally, if the circulation becomes feeble, to administer strychnine hypodermically, or by the mouth. Bitter tonics may also be prescribed for the purpose of maintaining digestive activity. The employment of antiseptic inhalations, as suggested in the treatment of pulmonary abscess, may also be resorted to, but at most only do good by soothing the irritation of the bronchial mucous membranes and cannot, of course, influence the pulmonary parenchyma where the disease exists.

If the evidences of sepsis are marked and anæmia is present, the tincture of chloride of iron is to be administered, and the heart supported by alcohol, digitalis, and occasionally by caffeine. The internal use of creosote, carbolic acid, and similar substances, with the idea that they exercise a beneficial influence upon the gangrenous portion of the lung, is futile.

A few cases of gangrene of the lung have been treated surgically, with success, by incision and drainage. For these methods the reader is referred to surgical treatises.

PULMONARY ABSCESS.

Etiology.—Abscess of the lung is always due to invasion of its tissues by one or more forms of pyogenic micro-organisms. Single large abscess occurs very rarely, but it is met with as a sequel of lobar pneumonia, bronchopneumonia, and as a result of injury to the lung by the entrance of foreign bodies through the chest wall or by the respiratory passages. Most commonly small abscesses are the result of septic emboli. Abscess may be due to the extension of a septic process from the mediastinal tissues or of the liver. So, too, a suppurative process in the deep tissues of the neck may result in secondary infection of the lung.

When pulmonary abscess ensues after an attack of croupous pneumonia or bronchopneumonia, it is usually not single, but multiple, the area of consolidation being the seat of several foci of purulent material. These formations are not by any means so rare as in the larger variety. Holt states that he found them in about 7 per cent. of the autopsies of young children dying of pneumonia. Such foci are really not true abscesses; that is to say, they have no true abscess wall. When these formations are numerous, as they usually are, and of considerable size, the patient may maintain a high temperature for a long time after the acute primary disease has passed away, and may, by causing septic absorption, ultimately produce the patient's death. It may be difficult, even at autopsy, to state positively whether the purulent infiltration of the later stages of both forms of pneumonia is present, or if there is a true suppurative abscess in the lung. In both cases the areas of softening are found to be infected by the streptococcus, staphylococcus, or other pyogenic organism.

When the foci are of large size, and are multiple, the prognosis is bad, for widespread suppuration in the lung is always fatal when the breaking-down process involves the exudation of pneumonia. If there be a single, large, localized abscess involving the area of pneumonic exudate, the prognosis is less grave, but it is exceedingly bad, nevertheless. To sum up, therefore, we find that suppuration takes place in the lung in three degrees or forms after pneumonia: (1) as a mild suppurative process, which is really nothing more than a rapid breaking down of the exudate of the disease; (2) as a more severe process, partaking more of the character of true suppuration, in which multiple and large foci of pus form; and (3) of a single large suppurative process; in other words, a single abscess of the lung. As already stated, these so-called "abscesses" rarely have a true abscess wall.

Abscess, multiple or single, when it arises from the entrance of a foreign body, only occurs if that body enables infecting micro-organisms to enter the surrounding tissues. Thus, a marble, or small stone, entering a bronchus may be there for a long time without causing abscess; whereas, the entrance of a piece of food, a straw, or a fragment of cork, or other organic matter may speedily cause a septic suppurative pneumonia and death. Such an abscess may follow a septic infection in a gunshot injury.

If by chance the patient recovers from the acute illness, there may be left a constantly discharging focus of pus.

Again, we find pulmonary abscess forming as the result of a septic embolus entering the lung. About the site of its lodgement an inflammatory exudate rapidly forms, and this speedily proceeds to suppuration. Pus and yellow elastic tissue are expectorated, and the patient dies of septic poisoning and exhausion, or if recovery takes place there is formed around the zone of necrotic tissue a wall of inflammatory exudate, which prevents further destruction of the parts, and, with recovery, proceeds to organization, finally developing into more or less well-formed fibrous tissue, which gradually contracts until the cavity disappears or is greatly decreased in size. We have in this type what may be called the true form of abscess as it occurs in other tissues; whereas, the ordinary suppurative foci hitherto described are hardly to be regarded as true abscesses. Occasionally the abscess cavity persists for months, and we have then a chronic pulmonary abscess.

When abscesses elsewhere than in the lung break into its tissues the result is not always a pulmonary abscess by any means. It is often extraordinary how much foul pus may pass from an empyema or hepatic abscess through the lung, and be expectorated, without causing any severe lesions in these organs.

Sometimes suppuration takes place in an echinococcus cyst in the lung.

Symptoms and Diagnosis.—The diagnosis of pulmonary abscess in its early stages may be practically impossible, for there may be present no other signs than *cough*, *fever*, and *scanty expectoration*, with patches of *impaired resonance on percussion*. In unresolved pneumonia the physical signs may be identical, but the leukocyte count is rarely above 15,000 or 20,000, whereas in abscess from 30,000 to 50,000 white cells may be present. As the pus is freely formed, much aid may be gained from the *temperature chart*, which may show the long sweeps of septic absorption. There may be *sweats*, *chills*, and some *hectic flushing*; but these do not necessarily point to abscess of the lung, for they may be due to empyema or an abscess elsewhere, or be a result of tuberculosis. If the sputum becomes distinctly purulent, and the microscope shows abundant pus and masses of connective tissue without tubercle bacilli, the diagnosis is readily made. About this time it may be possible, too, to discover the physical signs of cavity.

When a single large abscess is present the positive diagnosis may be made evident by the sudden rupture of its contents into a bronchus, and the expelling through the mouth of a considerable quantity of pus. I had under my care recently a young woman, aged twenty years, who, after an attack of typical croupous pneumonia, developed a more and more septic temperature, and, finally, expelled at one time nearly a pint of pus from the right lung. Constant expectoration of pus persisted for several days, and then an equally large amount was expelled, nearly causing death by strangulation. After a long convalescence she reached perfect health. In this case the fluoroscope revealed the site of the abscess very clearly. Care must be taken that the purulent expectoration and fetid breath of a case of bronchiectasis is not considered an indication of true pulmonary abscess.

Prognosis.—The prognosis in these cases is always very grave. Death may ensue, not only from septic absorption, but from the gradual exhaustion due to prolonged suppuration or from the ulceration of the wall of a blood-vessel with consequent severe hæmoptysis. Again, a secondary pneumonia may develop from the primary suppurative process.

Treatment.—The treatment of abscess of the lung divides itself into three parts: the support of the patient's strength by good food and the moderate use of stimulants; the resort to as much fresh air and sunshine as possible; the inhalation of gentle antiseptic balsams which do not really influence the abscess, but perhaps benefit the associated bronchitis; and, lastly, by the use of the knife, bone forceps, and the actual cautery, to open the abscess through the chest wall and lung.

As supporting drugs, iron and arsenic, whiskey and port wine, are particularly valuable. Easily digested semi-liquid foods, with digestants to aid their speedy absorption, are valuable, and in the way of an inhalation equal parts of oil of eucalyptus, oil of pine, and compound tincture of benzoin may be added to the water in a croup kettle, and so dissipated through the air of the room. Codeine and cannabis indica may be used to relieve excessive, painful cough; but large doses of these drugs should not be used, because they prevent the expectoration of the pus, and if the patient sleeps soundly while under their effects, rupture of the abscess may cause fatal asphyxia.

CONGESTION OF THE LUNGS.

Definition.—Strictly speaking, there is a congestion of the lungs whenever severe exercise is taken, but this, of course, is not referred to here; nor is it the intention to consider that form which precedes, or rather constitutes, the early stage of croupous pneumonia, and which ends in the formation of a croupous exudate. The form of congestion here referred to is that due to mechanical causes which interfere with the proper passage of blood through the pulmonary vessels (passive congestion), or that due to intense irritation caused by inhaling irritant vapors or fumes.

Etiology and Pathology.—The most common cause of pulmonary congestion is progressive valvular inadequacy at the left auriculoventricular orifice, or, in other words, mitral disease, either obstructive or regurgitant. These lesions dam the blood back into the lungs, and the right ventricle undergoes hypertrophy in an endeavor to drive it onward. As a result the pulmonary capillaries are placed under abnormal strain, increased hemolysis occurs, and when the condition becomes chronic there is produced what is known as brown induration of the lungs. At autopsy they appear of a dull reddish-brown hue, the incised surfaces becoming brighter red after exposure to the air. The supporting tissue of the lung is thickened and less elastic than normal, and the organ is heavy, as shown by the fact that when placed in water it does not float so high as normal lung-tissue. Microscopically the connective tissue and the alveolar epithelium, some of which is desquamated, contain granules of brownish pigment derived from the hæmoglobin content of the disintegrated red blood cells.

The bloodvessels are tortuous, and the capillaries which line the walls of the alveoli project in loops or tufts into the air spaces. Sometimes hæmoptysis of moderate degree arises from rupture of these vessels or those which are in the bronchial tubes. It can be readily understood why it is that a person with these lesions is a ready victim for pneumonia, hypostatic congestion, and infarction.

Acute pulmonary congestion resulting from sudden failure of the left ventricle is often the cause of sudden death in the course of an attack of sunstroke or after the inhalation of irritant gases.

Closely connected with this form of congestion from an etiological standpoint is *hypostatic congestion* of the lungs. In this state the lower portions of the lungs are commonly affected because the patient is usually in the dorsal decubitus and the blood accumulates in the most dependent part of the organs. Associated with this accumulation of blood in the vessels of the lung, an excess of serum collects in the intervesicular structures, producing œdema, or, passing into the vesicles, causes the affected part to become essentially airless.

The causes of hypostatic congestion are not very different from those of ordinary congestion as just described, save that the failure of the right side of the heart is more marked and the condition is more frequently met with as the result of profound asthenia occurring in the course of some malady like severe typhoid fever or advanced renal disease. That the dorsal decubitus is not the chief cause is proved by the fact that many persons suffering from certain maladies which require the maintenance of this posture do not suffer from hypostatic congestion. That posture exercises some influence, however, is shown by the fact that if the patient remains on one side the stasis is often unilateral.

Autopsy in cases of hypostatic congestion reveal the involved areas darkened in color, often black or purplish-black in hue. They may be airless, with frothy accumulations in the bronchial tubes, loss of crepitation on pressure, and a doughy condition when one finger is pressed upon the lung, resembling the sensation produced by œdema elsewhere. In some cases, not only a serous exudation takes place into the vesicles, but red and white blood cells are extruded, which may render the lung so red that it looks somewhat as if true croupous pneumonia were present. To this state has been applied the term "splenization," in distinction from the red solidification in true pneumonia called "hepatization" or "hypostatic pneumonia." Still less frequently actual hemorrhage into the lung occurs as the result of giving way of the walls of small vessels.

The causes being identical on both sides of the chest, it is natural that hypostatic congestion should usually be found to be bilateral. It begins at the bases and slowly creeps upward, until it may involve the lower lobes of each side, and even the middle lobe on the right side and part of the upper on the left.

Symptoms.—The symptoms of that form which is due to valvular disease at first are those of *shortness of breath*, with repeated attacks of bronchitis, which may become chronic. The mucus expectorated may contain tiny clots of blood arising from the dilated vessels just described. If the hemorrhage is free an infarct of the lung may develop into an area of consolidation and hæmoptysis may occur. Sometimes this accident follows an improvement in the condition of the heart, which is produced by rest and tonics, because the renewed strength of the right ventricle ruptures a weak and tortuous vessel.

The symptoms of hypostatic congestion differ greatly with the rapidity with which the condition develops, and the underlying cause. When the exudation rapidly takes place evidences of *respiratory embarrassment* develop and *dyspnœa* and *cyanosis* are often marked. If the condition is slow and gradual, as in most instances, when it complicates some state of adynamia, as in severe typhoid fever, the symptoms are so gradual in onset that not until the lungs are seriously involved is attention called to respiratory disorder. Cases of the acute type are seen chiefly as the result of renal disease and cardiac failure, whereas, as just stated, the gradual type comes on in the course of the infectious diseases.

The physical signs of hypostatic congestion are not well marked in the early stages. Careful light *percussion* may reveal *slight impairment of resonance*, and *auscultation* may discover a few *moist rales*, which are chiefly bronchial, forming small rhonchi or sibilant sounds. These are the signs which it is important to recognize, since it is at this time that the physician can do much, in many cases, to limit or even prevent the spread of the condition which is beginning to develop. Later on the condition is so well marked that the merest tyro can recognize it by reason of the bronchial breathing, the moist rales, and the absence of vesicular sounds which have been put aside by the exudation. The only thing to be done at such a late hour is to endeavor to support the circulation, so that the lesions will not spread and so that the patient may be kept going till absorption or resolution occurs.

The presence of hypostatic congestion is often not recognized, because the physician does not carefully examine the lungs. In many cases, too, it is agonal, particularly if death comes slowly.

Diagnosis.—Hypostatic congestion must be separated from *catarrhal* and *croupous pneumonia*, and from *pleural effusion*, serous or purulent. An important point in the differentiation is the fact that in both forms of pneumonia the temperature is usually febrile, and if they complicate some preexisting state the fever is usually exacerbated when the pulmonary condition develops, whereas distinct febrile movement is unusual in hypostatic congestion unless it is in turn associated with a true pneumonic process. The sputum, if any is raised, is frothy in cases of congestion, but is sticky and rusty in croupous pneumonia, and perhaps mucopurulent in the catarrhal form. The cough is loose and productive (juicy) and not hard and difficult as in pneumonia. Then, too, the onset of congestion is not characterized by a chill nor by pain in the chest. *Pleural effusion* may be separated from hypostatic congestion by a change in the level of dulness on percussion when the patient changes his posture, by the fact that the percussion note in congestion is rarely as flat as in effusion, by the fact that pleural effusion is rarely bilateral, and if at all profuse usually displaces the heart to the left if it be on the right side and downward if it be on the left.

Prognosis.—This depends largely on the promptness with which hypostasis is discovered and treated, the cause of the condition, and the vitality of the patient. When due to renal disease and associated with a general tendency to œdema, the prognosis is bad. So, too, if it ensues in a prolonged exhausting fever the prognosis is bad because it indicates great feebleness. In old persons and in young children it is very often the cause of death during the course of other diseases.

Treatment.—This consists in preventive measures, such as changing the posture of the patient every hour, in the use of cold sponging if fever is present, to readjust the circulation, and in the proper use of stimulants if the heart seems feeble. As soon as any signs of the malady appear, the patient should be made to lie on one side and then on the other and not upon the back. Two or three dry cups should be applied to the chest over the base of each lung posteriorly, or in their place a mustard plaster may be used. If the heart is feeble, strychnine, digitalis, and belladonna are useful. It may be wise in urgent cases to give strychnine and atropine hypodermically and to use Hoffmann's anodyne in the dose of a drachm every hour for several doses. Sometimes if the patient is strong enough to stand active purgation colocynth or elaterium are valuable cathartics, the latter being given in the dose of $\frac{1}{8}$ grain, but when the symptoms are urgent and the venous system is engorged free venesection should be practised.

Manifestly it is the physician's duty in all these cases of exhausting disease to carefully listen to the chest at every visit during an illness, to note the first sign of this insidious state.

TUMORS IN THE LUNGS.

Tumors in the lungs are rarely met with. They may be benign or malignant, but are usually the latter, and occur as primary or secondary growths; tumors secondary to growths elsewhere being much the more frequent. The benign tumors are chondroma, fibroma, osteoma, and dermoid cyst. The malignant tumors are sarcoma, carcinoma, and occasionally endothelioma. The sarcoma and carcinoma usually occur as nodular masses which as they grow push the lung-tissue aside, or more rarely they occur as infiltrating growths which extend along the bloodvessels or bronchial tubes. If the tumors are placed peripherally or are primary, it may be difficult to determine whether they are pleural or pulmonary. In cases of Hodgkin's disease and in leukæmia typical masses of lymphomatous tissue are quite frequently found infiltrating the lung, and they may cause consolidation throughout considerable areas.

When the malignant growths are secondary they are usually found in both lungs unless the tumor is the result of extensive infection, as in the case of tumor in the chest wall directly involving the lung tissue through the pleura. In such an instance the growth at first is single, whereas when it has spread by metastasis it is multiple. Secondary cancer of the lung is more frequent in women than in men because of the frequency with which women suffer from carcinoma of the breast.

Symptoms.—The symptoms are not characteristic. They depend largely upon the situation of the growth and upon the degree of pressure which they exercise upon surrounding tissues. If they press upon nerve trunks they cause *severe pain;* if upon a large bronchus they produce *cough* and *expectoration;* and if a considerable area of lung-tissue is involved they cause *dyspnœa,* particularly if the growth or growths press upon the bloodvessels and so cause pulmonary congestion or stasis, so that as the disease advances pulmonary œdema aids in decreasing the area for the oxygenation of blood. Great and manifest engorgement of the superficial veins of the neck and head is sometimes present as the result of pressure on the superior vena cava, and if the vagus or the recurrent laryngeal nerves are pressed upon cardiac neuroses and laryngeal spasm or paralysis may ensue.

Diagnosis.—The diagnosis of tumor of the lung when no primary growth exists elsewhere is extremely difficult. The presence of thoracic pain, in the absence of signs of aneurysm, and inability to discover cardiac disease, aortitis, or disease of vertebræ should arouse the suspicion of the presence of a growth, which may be confirmed by the presence of dulness on percussion in the area affected. When these symptoms develop in a patient who has a growth elsewhere, or has had a growth elsewhere which has been excised, as in carcinoma of the breast, they possess much more diagnostic value. Stokes considered that prune-juice sputum was a very typical sign of malignant growth in the lung. Emaciation may be a marked symptom, as it is so often in cases of malignant growth elsewhere in the body, but the maintenance of flesh by the patient does not negative malignant growth, as sometimes little weight is lost.

It is hardly necessary to add that the malignant tumors are more frequently met with in middle life or in advanced age than in youth.

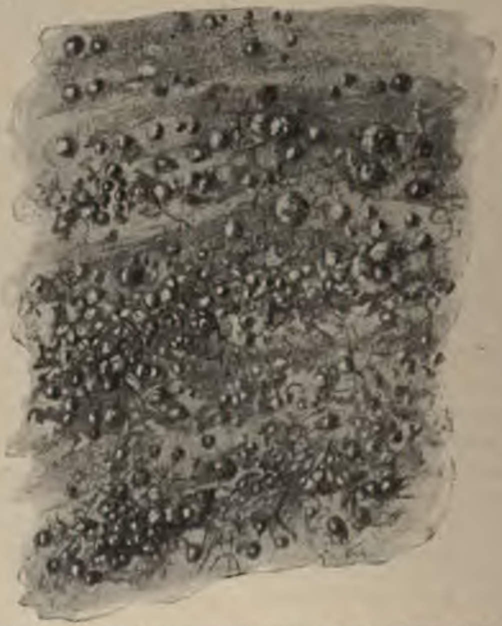
No treatment is of any avail so far as cure is concerned. The most that can be done is to support the system by good food and relieve pain by morphine.

DISEASES OF THE PLEURA.

PLEURITIS.

Definition.—The term pleuritis, or pleurisy, is applied to an inflammation, either acute or chronic, of the serous membrane which lines the thoracic cavity and in its reflections covers the lung; the so-called parietal and visceral layers of the pleura. This inflammation is always the result of an infection by some pathogenic micro-organism. It occurs in four forms, namely, as dry or fibrinous, serofibrinous, purulent, when it is called empyema, and that due to tuberculosis, or tuberculous pleurisy. Sometimes malignant disease affects this membrane, and this may be considered a fifth form of pleural inflammation (Figs. 57 and 58).

Fig. 57

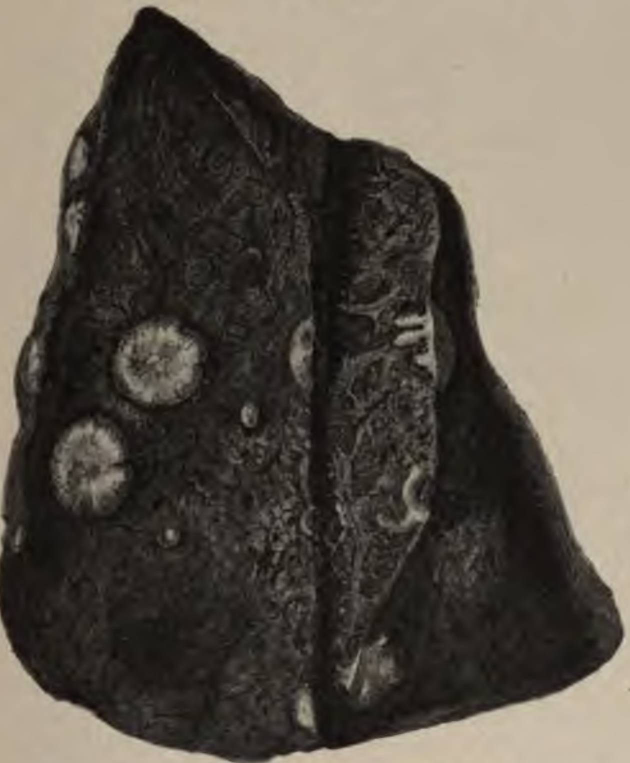


Carcinomatosis of the costal pleuræ. (Kast and Rumpler.)

Etiology.—As just stated, pleurisy is practically always due to an infection by some micro-organism. In a large number of cases it arises as the result of an invasion of the lung by the pneumococcus, with or without an associated pneumonia. In other instances it is due to the entrance of pyogenic organisms such as the staphylococcus and streptococcus, and in still other cases from invasion by tubercle bacilli. Infection of the pleura may also take place through the pericardium, the mediastinal tissues, the vertebræ, and the diaphragm. Sometimes, though rarely, it is from the chest wall itself, after injury to the thorax or by extension of infection from the mammary gland. Pulmonary abscess may, by the extension of the inflammatory process, produce pleuritis, or a bronchopneumonia may cause a secondary infection. In some cases, however, the inflammation of the pleura is a primary lesion without any pathological change in the lung except as a secondary condition.

The relative frequency with which acute pleurisy is produced by each specific micro-organism is unknown, since recovery takes place in mild cases and no opportunity of determining the provoking cause presents itself. The pneumococcus is, however, the cause in the majority of cases.

FIG. 58



Metastatic carcinoma of the visceral pleura. (Kast and Rumpler.)

When empyema follows pleurisy the necessity of setting free the pus enables us to determine the character of the infection in the great majority of cases, and the statistics derived from this source give us some conception of the relative frequency with which pleurisy follows infection by different organisms. (See Empyema.)

Frequency.—Pleurisy is most commonly met with between the ages of twenty and forty, but it is by no means confined to these decades of life. On the contrary, it is very frequent in young children—at least, as a complication of pneumonia in its various forms—and is also not rarely met with in persons of advanced years. In adults pleurisy occurs more than twice as often in males as in females, but in early childhood this predominance does not occur. As an illustration of these facts it is interesting to note that in 651 cases in St. Bartholomew's Hospital, London, 465 were in males and only 186 in females. The distribution of these cases as to age was as follows: five years and under, 25; ten years, 59; fifteen years, 50; twenty years, 54; thirty years, 179; forty years, 149; fifty years, 85; sixty years, 35; over sixty years, 15.

Pleurisy occurs most frequently in the early spring and late autumn, when great changes in temperature take place. This does not mean that exposure to cold produces pleurisy directly, but rather that the exposure reduces vital resistance to such an extent that infection takes place.

So, too, a number of acute and chronic diseases result in pleurisy, not because they have any direct effect on the pleural membrane, but because they lower vital resistance at the same time that they expose the pleura to infection by their specific germ. Thus, pleurisy may be indirectly produced by the acute specific fevers and by Bright's disease, the first of which provides a predisposing cause and a specific germ, while the latter lowers vital resistance in general. So, too, it is possible for damage to the chest wall to result in acute pleuritis.

It is to be constantly borne in mind that of all specific infections that by the tubercle bacillus is the most important, because of the prognosis, because it is often insidious, and because it is probably one of the most frequent causes of pleurisy.

The pathology, morbid anatomy, symptomatology and treatment of the various forms of pleurisy are best considered under the specific description of each type.

Dry Pleurisy.—Dry pleurisy, as its name indicates, is an inflammation of the pleural membrane with a minimum amount of serous exudate. It may be circumscribed or localized, as over a tuberculous cavity, or may be diffused over a large area, as in croupous pneumonia. The pathology and morbid anatomy of pleurisy of the dry type may be described as follows: As in all inflammations of serous membranes, there is an acute hyperæmia followed by infiltration and exudation of blood cells, fibrin, and, it may be, serum. The pleural membrane is lustreless in appearance, and roughened or granular, and is somewhat thickened, partly because of infiltration, but chiefly by reason of the fibrinous exudate on its surface. This exudate is a primary factor in the formation of adhesions between the visceral and parietal layers of the pleura. Sometimes the exudate is remarkably profuse or perhaps a number of layers are formed, so that the pleura may exceed a quarter of an inch in thickness, and is somewhat reticulated or uneven on the surface. Such an exudate is rarely completely absorbed after the attack has passed, and it often organizes and produces impaired resonance on percussion and other morbid physical signs during the lifetime of the patient.

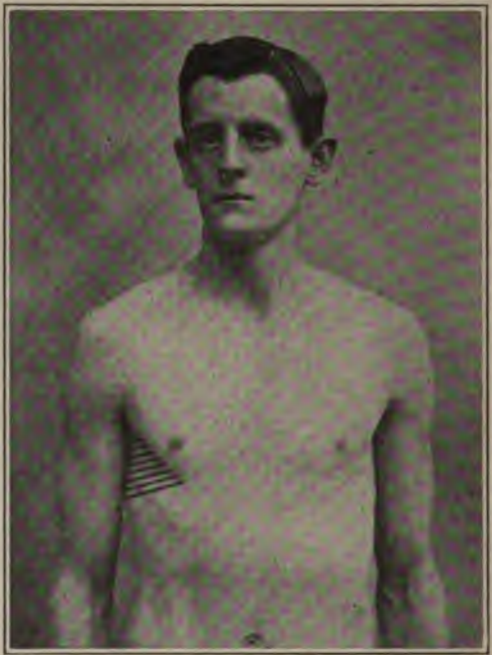
Symptoms.—The onset of acute dry pleurisy is characterized by a *severe pain*, or "stitch," in the side and by the development of some *fever*. The pain in the side is sharp and stabbing in character and the patient "catches his breath," to use a popular expression, when he endeavors to inspire. Speaking, coughing, or any movement which causes increase in the thoracic movement, greatly increases the pain, which can, however, be markedly relieved, as a rule, by strapping the side of the chest which is affected, and so diminishing its freedom of movement. The pain which is developed by pressure on the chest wall is sometimes of two types, namely, severe pain produced by deep pressure, and *exquisite tenderness of the skin* over that part of the pleura which is inflamed. In the great majority of cases the patient states that the greatest pain is between the mammary line and the posterior axillary line, but it may be complained of in many other parts of the chest, particularly if the disease be due to tuberculosis. Young children who have not been trained in the localization of pain often state that the suffering is in the epigastrium, or in the left or right hypochondrium, and even in adults I have more than once seen physicians misled into a diagnosis of appendicitis because of the pain referred by the patient to this region, when in reality the cause was acute pleuritis. In all cases of pain below the diaphragm it is a good rule for the physician to examine the condition of the thoracic viscera before asserting that abdominal disease is present. As severe stabbing pain in the thorax is sometimes due to aneurysm, muscular rheumatism, or intercostal neuralgia, these possibilities must be excluded before we can decide that the cause is pleuritis.

The most important physical sign which determines the diagnosis of this affection is the so-called "*friction sound*" produced by the rubbing of the visceral layer of the pleura upon the parietal layer, both layers being roughened and dried by the early stage of the inflammation. This friction sound is usually best heard just below and just back of the nipple on the side involved. (See Fig. 59.) In persons who have very thick chest walls and who breathe superficially, by habit or because of the pain, it is often necessary that they take a deep breath before a friction sound is produced. Sometimes the friction sound is so creaking and loud that it sounds like the noise made by a new leather saddle when it is first used; at other times it so soft that only the most careful auscultation will reveal it, and it may resemble the fine rales of croupous pneumonia. In other cases this creaking can be felt by the hand of the physician. If the pleurisy be situated near the heart the action of that organ may cause the pleural friction sound to occur as often as the heart beats, and so lead one to the diagnosis of pericarditis. This is called a *pleuropericardial friction sound*, and may also depend upon a simultaneous development of pericarditis and pleuritis.

A second important physical sign is the *diminished respiratory movement* on the side of the chest which is affected, as may be seen by the eye and recognized by the feeble respiratory sounds when auscultation is performed, the semi-fixation of the chest being an effort to decrease the thoracic movement, and so limit the degree of pain. My colleague, Coplin, has suggested that the fixation is in part due to changes in the intercostal muscles themselves. (See article on Croupous Pneumonia.) This fixation may extend to one side of the diaphragm, and so result in decreased abdominal movement on that side. The rate of *respiration may be increased* in order to compensate for the shallow breathing, but it is never the hurried or urgent respiration met with in cases of real dyspnœa.

There are two other signs of pleurisy which are of some diagnostic value, namely, the *suppressed cough*, which the patient attempts to stifle in order to prevent pain, and the *attitude of fixation of the body* so that inadvertent movement of the patient himself, or change in his position made by his attendant may not produce pain. Sometimes if the skin is not hyperæsthetic the patient lies on the affected side to render it fixed, or he may lie on the well side to avoid pressure on the involved pleura.

FIG. 59



Area in which a right-sided pleural friction sound is usually heard best.

The *fever* in acute pleurisy is rarely high in adults, although it may be in young persons. Often it never rises above 102°, and the pulse is usually only increased by reason of the fever; so that it bears no direct relationship to the disease.

Diagnosis.—Dry pleurisy is separated from muscular soreness due to strain by the facts just given and by the history of an injury; from muscular rheumatism by the fact that signs of this malady are to be found elsewhere; from intercostal neuralgia by the inconstancy of that affection, and by the fact that ordinary breathing does not increase the pain in the majority of cases, and, further, that all three of these conditions are not accompanied by any febrile movement or evidence of general systemic disturbance. Acute pleurisy of the dry type lasts from a few days to two weeks. A longer attack than this should arouse the suspicion of the presence of a more persistent disease, such as tuberculosis.

Prognosis.—Barring complications the prognosis is favorable. (See Empyema, and Pleurisy with Effusion.)

Treatment.—The treatment of dry pleurisy consists in applying adhesive strips two inches wide, and overlapping one another one inch, from the middle line of the vertebræ to the middle line of the sternum, not following the line of the ribs, but passing from behind forward horizontally. They should be applied from below upward, and with a sufficient degree of pressure to produce almost complete fixation of that side of the chest. The pain, if it is extensive, may be further controlled by the administration of 3 grain doses of Dover's powder every two or three hours. If necessary, a hypodermic injection of morphine may be given. If the fever is high an ice-bag may be applied to the head, and tepid or cold spongings over the entire body may be employed. An ice-bag may also be applied to the side of the chest which is inflamed, for the relief of pain.

In the earliest stages of an acute dry pleurisy, in a strong, healthy individual of a plethoric type with a bounding pulse, there can be no doubt that the administration of sufficiently large doses of the tincture of veratrum viride or the tincture of aconite is advantageous, as it may diminish the local hyperæmia in the pleura and decrease the action of the heart so that it pumps less blood into the inflamed area, thereby causing determination of blood to the peripheral capillaries. This vascular relaxation, associated with sweating, tends to still further relieve the local congestion, and altogether exercises a beneficial influence upon the local lesion. These depressant drugs, however, are distinctly contraindicated unless the patient is strong and hearty, and after the first twenty-four hours of the illness they are probably useless. Indeed, after this time they may do harm. If they are used at all, they should be given freely. Thus, 3 minims of the tincture of veratrum viride may be given every half-hour until the patient is very slightly nauseated or until his skin becomes moist, when the drug should be stopped. A similar method of employing aconite may also be practised.

The employment of a poultice, or cotton jacket, in the treatment of pleurisy is less and less resorted to at the present time. There is no reason to believe that its influence is advantageous, and it very greatly increases the discomfort of the patient because of the heat and consequent sweatings which are produced. Further than this, there is always danger of the patient taking cold by the poultice becoming chilled, or during the removal of the poultice or cotton jacket for cleansing purposes.

It is important to remember that the presence of a moderate pleural effusion does not require the physician to institute measures for its immediate relief, because in a very considerable proportion of cases absorption will take place by natural processes, and so nature will produce a cure.

Finally, all patients convalescing from an attack of dry pleurisy should be instructed to present themselves to the physician several times at intervals of a few days, in order that he may have the opportunity of determining whether the pathological condition has entirely disappeared. It happens, all too frequently, that such patients are discharged "cured" at the end of a few days, when they actually have an insidious tuberculosis, the primary pleurisy having been due to this cause.

Pleurisy with Effusion.—While a large proportion of cases of acute pleurisy are dry, in the sense that no excess of serum is poured out by the inflamed serous membrane, it is not to be forgotten, on the other hand, that a considerable number of cases of pleural inflammation terminates in more or less profuse outpouring of fluid into the pleural sac. This forms what is sometimes called "pleurisy with effusion," or "pleuritic exudation." While the dry type often only involves a patch, or small part, of the pleural membrane, that form which is accompanied by effusion, unless limited by adhesions, usually affects the entire pleura of one side, and, indeed, it may be bilateral, although this is, fortunately, a rare occurrence.

The exudate is composed of two parts: (1) a solid portion, consisting of fibrin and cells, which is attached to the surface of the pleura and which constitutes the basis by which adhesions binding the two layers of the pleura together may be formed, and (2) serum or fluid exudate, which may be so abundant that the pleural sac is completely filled. This fluid is always turbid or cloudy from the presence of degenerated and exfoliated endothelial cells, particles of fibrin and blood cells, particularly leukocytes. It is worthy of note that the pleura in cases of pleuritis, accompanied by serous effusion, is usually not so markedly infiltrated as in the dry type.

These effusions are usually the result of infection by the pneumococcus, the staphylococcus pyogenes, and the tubercle bacillus. The latter infection is always to be suspected in subacute cases with much fluid and little plastic exudate.

An examination of the literature on the bacteriology of this state shows that a large number of organisms have been found in pleural effusions and also that in many cases the effused fluid is sterile. (See Empyema.) For example, Lemoine made cultures from the fluid of 38 cases of serofibrinous pleurisy, and found it sterile in 28 instances.

Recovery, which takes place in the majority of cases, occurs by the absorption of the serum and the partial absorption and shrinkage of the fibrin, but the chief change in the plastic exudate is organization brought about by the formation of granulation tissue, which finally becomes dense and cicatricial in character.

There is probably no form of pleural effusion so prone to confuse the clinician as loculated or ensacculated effusions. These may form between lobes, between the base of the lung and the diaphragm, or on the mediastinal aspect of the organ. Their localization is maintained by marginal adhesions that prevent the diffusion of fluid throughout the pleural cavity. Empyema, similarly limited, offers identical difficulties in diagnosis.

The lung may be markedly distorted, displaced, or compressed by the adhesions, and even the heart may be forced from its normal position.

Symptoms.—The symptoms of pleurisy with effusion are not very characteristic, except in so far as the physical signs are concerned, but these are typical, and some of them pathognomonic. If the onset of the attack of pleurisy has been sharp the severe pain already described passes away as the effusion takes place and so separates the inflamed layers of the pleura, at the same time probably depleting them. The fever often diminishes or disappears when the stage of effusion is reached. *Dyspnœa* may or may not be present, according to the size of effusion, the spaciousness of the chest, and the ability of the healthy side to do enough work to compensate for the part which is impaired in function. Strong, hearty individuals often seem to be more *dyspnœic* than feeble ones, probably because in the former case the effusion is more rapid and the restricting adhesions are more firm. *Cough* in this stage of effusion is usually not severe, and may be absent, except on exertion. It is often due to an associated bronchitis.

The *posture* of the patient, if the effusion be large, is usually characteristic, in that he persists in lying on the affected side, in order to permit the healthy lung to have full play. Turning him on the affected side may cause urgent dyspnœa and a sudden change to the erect posture may do likewise, since the pressure of the fluid on the diaphragm interferes with its movements or with the action of the heart.

PHYSICAL SIGNS.—The physical signs of pleural effusion are as follows: *Inspection* shows decrease in respiratory movement on the affected side, with increased activity on the healthy side; bulging of the entire chest on the diseased side, with fulness of the interspaces and some fulness it may be in the hypochondrium. *Palpation* reveals an absence of vocal fremitus on the affected side, and if the effusion be on the left side the apex beat of the heart is displaced downward and to the right.

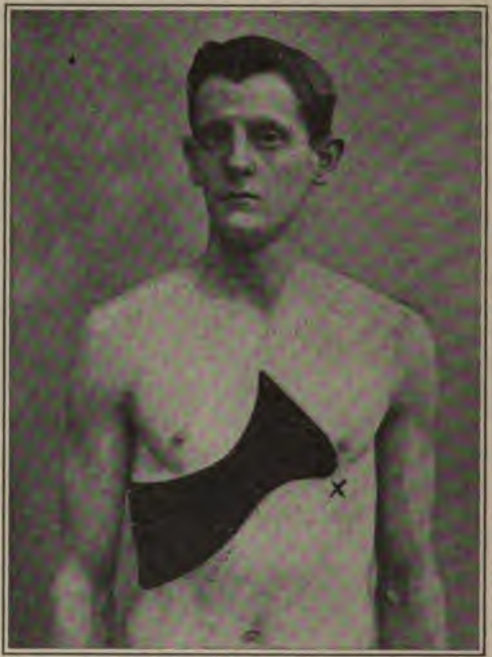
Percussion elicits flatness, or marked dulness, except at the apex above the fluid, where the percussion note is peculiarly high-pitched, and almost tympanitic—the so-called Skodaic resonance. Percussion of the liver, if the effusion be on the right side, may show that the lower margin of liver dulness is abnormally low. If the effusion is on the left side, percussion shows dulness in Traube's semilunar space. (See Fig. 60.)

Auscultation discovers that there is an absence of breath sounds in the area where percussion gives flatness, except it may be for distant and transmitted bronchial breathing. Along the vertebral column and near the inner edge of the scapula on the affected side ægophony, or the "bleating voice" sound, may be heard if the patient speaks, while vocal resonance in the apex of the lung, where Skodaic resonance is present, is greatly increased, even to the degree of pectoriloquy. At this place above the effusion bronchial or tubular breathing may be very marked. Sometimes the breath sounds are even amphoric in character.

Occasionally, as the result of the formation of adhesions, pleural effusion is circumscribed within narrow limits, and the presence of an inflammatory exudate produces an area of dulness which is much larger than that space occupied by the fluid. The introduction of an aspirating needle for diagnostic purposes may, therefore, readily mislead the physician, since a dry tap will often occur unless the needle happens to enter that portion of the area of dulness which actually contains the fluid. The mere introduction of the needle into the centre of the area of dulness is not necessarily followed by the withdrawal of fluid, since it not infrequently happens that a considerable mass of inflammatory exudate lies to one side of, or above or below, the fluid. These loculated effusions are more common in cases of empyema than in ordinary cases of pleurisy with effusion. (See Empyema.)

The rate at which effusion takes place varies very greatly. Rarely the chest may become filled in a few days; more commonly it takes a week or even three weeks. Rapid effusion is more dangerous than the delayed type, because the thoracic viscera in the former case do not have time to adjust themselves to the altered conditions. I have seen a case of rapidly forming pleural effusion in which sudden death followed the turning of the patient on his well side.

Fig. 60



Showing at x mark the so-called area called Traube's semilunar space, where, in health, percussion gives a tympanitic note, which becomes flat in left-sided pleural effusion. The solid block represents hepatic and cardiac dulness.

The duration of pleural effusion varies very greatly. Small effusions are often absorbed with surprising speed within a few days, but large ones are often very slowly absorbed and may not be absorbed at all until some of the pressure is removed by aspirating the chest.

Diagnosis.—It is a noteworthy fact that while the diagnosis of pleural effusion is very readily made in some cases, in other instances it is so difficult as to baffle the most experienced clinician.

Pleurisy with effusion is to be separated from pneumonia, from tuberculous consolidation, from pulmonary œdema and hypostatic congestion, from new-growths in the lung, pleura, and mediastinum, and from pleurisy with great fibrinous exudation and thickening.

If on examining one side of the chest it is found to present impaired movement, impaired percussion resonance, and absence of breath sounds, it is fair to suppose that the cause is effusion, if in addition we find, in disease of the right side, displacement of the apex beat to the left, or, if it be left-sided, obliteration of Traube's semilunar space. This opinion is still further confirmed if the area of dulness on percussion varies with a change in the posture of the patient, and if Skodaic resonance is present above the area in which resonance is impaired. On the other hand, it is not to be forgotten that high-pitched resonance is often met with in that part of the lung which is over an area consolidated by pneumonia. In pneumonia distinct bronchial or tubular breathing is usually heard throughout the consolidated area, and this, of course, is not the case in effusion; but if the bronchial tubes become plugged by secretion in pneumonia, this important differential point is destroyed. Again, it sometimes happens that if the physician auscults the chest with the unaided ear he can readily hear bronchial breathing even if an effusion be present, although if he uses a stethoscope bronchial breathing seems absent. In pneumonia, however, bronchial breathing is usually associated with rales which are absent in effusion.

Very useful in the differentiation of the two affections is the history of the patient, in whom the early symptoms of the two diseases are usually quite at variance, unless the case has been one of primary pleuropneumonia.

In cases of tuberculous consolidation the appearance of the patient and the history of onset may be valuable differential points, and if loss of flesh or fever is present these facts are still further emphasized.

When pulmonary œdema is present the presence of moist rales, the feeble heart action, and the discovery of some prolonged preceding illness, or of renal disease predisposing to pulmonary œdema, and bilateral dulness, are the points of value in making a diagnosis.

In cases of acute pleurisy with great thickening of the pleural membrane there may be marked impairment of resonance on light percussion, and a friction sound may be heard, but deep percussion may elicit normal pulmonary resonance.

Growths in the lung, or pulmonary abscess, usually are so peculiarly placed and surrounded by healthy tissue that careful examination of the chest and a study of the patient's history will be sufficient to make the differentiation.

Pneumothorax is separated from pleural effusion by its high-pitched resonance on percussion and the other physical signs of that condition which are only partly modified if the pleura is chronically thickened.

There still remain two important diagnostic points in these cases which have to be studied before diagnosis can be reached, viz.: Are the physical signs due to the possible presence of subphrenic abscess, which, pushing the diaphragm upward, encroaches upon the thoracic space, or are they due to abscess or hydatid cyst in the liver? These conditions become manifest if the patient is carefully examined for them. Further, their rarity is a point against their presence.

Lastly, it is important to determine the size of the effusion in order that the danger to the patient may be appreciated. It is not possible to even approximate the actual quantity, because the capacity of the chest varies greatly in different cases, but the extent of the effusion can be decided by the line at which percussion dulness first changes to impaired resonance, and higher to high-pitched resonance.

After a diagnosis of pleural effusion has been made, the question which arises is whether the effusion is serous or purulent, and if serous whether it is the result of inflammation or transudation. This is a most important question, since the treatment is quite different in each instance.

This may be determined by performing *paracentesis thoracis* and to a great extent by an examination of the fluid after it is withdrawn by aspiration. Its specific gravity, if the cause be of an inflammatory nature, varies from 1.010 to 1.018, and it contains large amounts of fibrin and albumin. On the other hand, the fluid due to transudation in dropsy shows a specific gravity of only about 1.008 and contains little fibrin and albumin. (See Hydrothorax.) When the effusion is due to tuberculosis the specific gravity is very high (1.012 to 1.024). The symptoms and diagnosis of empyema will be found discussed below.

CYTOSCOPY IN PLEURAL EFFUSION.—In 1900 Widal and Ravaut called attention to the cytological examination of the fluid of pleural effusion, asserting that the nature of the pleurisy can be determined by the organized elements held in suspension in the exudate. According to their observations, the fluid of tuberculous pleurisy is characterized by the presence of lymphocytes, that of the acute infective pleurisies by polymorphonuclear leukocytes, and that of the pleurisies dependent upon new-growths and the aseptic pleurisies accompanying renal and cardiac disease, by shreds of endothelium. Further investigations have not confirmed these results, for Naunyn found that the effusions complicating Bright's disease often contain lymphocytes instead of endothelium shreds, and Tarchetti and Rossi found lymphocytes in only a portion of the tuberculous effusions which they examined. Moreover, Patella's investigations convinced him that lymphocytes are not characteristic of primary tuberculous effusions. On the other hand, Barjone and Cade, and Gemelli, of Milan, have found lymphocytes in the fluid of all tuberculous pleurisies. From what has been said, it is evident that the subject is yet in its experimental stage, but the discovery of a marked lymphocytosis in the fluid is certainly of some value as indicating tuberculosis, particularly if it is associated with other signs. So too high a count (60 to 90 per cent.) of polymorphonuclear cells is indicative of an infection by the pneumococcus.

Prognosis.—The prognosis in cases of pleural effusion is favorable, except in two conditions. If the formation of the fluid is very rapid and very copious, pressing upon the heart and lungs and seriously impairing their action so that dyspnœa becomes urgent, the prognosis is, of course, grave, unless relief is given by thoracentesis. Again, if the effusion is primarily due to tuberculosis, or to nephritis, which, by decreasing vitality, has permitted infection to take place, the prognosis must be correspondingly grave as to ultimate recovery.

Treatment.—The early stages of pleurisy with effusion are, of course, treated in a manner identical with that already described for a dry pleurisy.

It is only when the effusion has formed and is in such large quantity that it produces pressure upon a vital organ, or, again, when it remains unabsorbed for a considerable period of time, that the physician should undertake measures for its removal.

The only measure of any value when the pressure is sufficiently great to be producing serious symptoms is "tapping" the chest by means of an aspirator. The skin over the affected side should be first thoroughly cleansed, as if for the performance of a minor surgical operation. A hollow needle having a moderately wide calibre, and attached to a rubber tube three feet long, which is filled with fluid, is then pushed into the pleural cavity in the sixth or seventh interspace in the midaxillary line. Care should be taken that the aspirating needle should be kept well down on the upper surface of the nether rib, in order to avoid injuring the intercostal artery, and the physician should grasp the needle with his thumb and forefinger not far from its point, so that after it pierces the skin it will not suddenly plunge into the chest for several inches, and so, perhaps, do damage to deep-lying tissues. No sooner does the needle enter the pleural cavity than the end of the rubber tube is lowered to a level with the floor and the contents of the pleura is in this way siphoned out of the chest. The advantage claimed for this method of treatment is that the degree of suction is at no time great, and, furthermore, it is constant. Again, there is no danger of the fluid being withdrawn with too great rapidity.

A very much more frequently resorted to measure of performing *paracentesis thoracis* is to attach a large needle, or trocar and cannula, to a piece of rubber tubing, which, in turn, is attached to a tube running through the cork of a bottle in which a vacuum has been produced by a small hand-pump. The entrance to the bottle is guarded by a small stopcock. After the needle has been placed in the chest, the trocar is withdrawn, the stopcock is turned, and the fluid is drawn by the vacuum from the chest into the bottle. It is rarely if ever proper to completely empty the chest by this means at one sitting, particularly if the effusion has been a large one. Too rapid withdrawal of the pressure in the thorax may cause serious disturbance of the action of the heart, or too rapid an expansion of that portion of the lung on the affected side which has been compressed by the fluid, with the result that damage is done to the pulmonary tissue, or that a peculiar form of gelatinous exudation into the lungs takes place, which is only relieved by constant and exhausting cough, and sometimes results fatally.

Should constant cough develop during paracentesis, it is best to discontinue the operation at once.

It is also important to remember that not infrequently the withdrawal of a small quantity of the effusion, by the relief of pressure and the establishment of normal lymphatic and blood circulation in the chest wall, may result in the natural absorption of the remaining fluid with a very considerable degree of rapidity, so that even if the chest is not emptied by the aspiration it may become so in a few days by a natural process. This holds true with particular force in those cases of large pleural effusion which do not require interference because of pressure symptoms, but which do not undergo absorption by natural means until after absorption has been stimulated by the performance of paracentesis.

It is necessary that the physician should exercise care in inspecting his needle before he employs it. Experienced clinicians have frequently been infected by a dry tap when they were skilful enough to diagnose an effusion, but careless enough not to notice that their needle was plugged.

A pleural effusion should not be permitted to remain too long in the chest, since its presence tends to increase the organization of the inflammatory process on the surface of the lung, or results in the formation of such firm adhesions that decortication of the lung by the surgeon is necessary if recovery is to ensue.

The employment of purges, diuretics, or diaphoretics in cases of pleurisy with effusion, with the object of causing an absorption of the fluid, is, for very good reasons, futile in almost every instance. It has already been pointed out that in this disease the pleura is almost invariably covered by a dense fibrinous exudate, which is plastic in character and mechanically interferes with the absorption of the exudate. Even if the physician is able, by the administration of powerful hydragogue cathartics, to cause a concentration in the blood, this concentration does not result in the absorption of the pleural effusion, because of the obstruction just spoken of, and also because absorption takes place from the pleura chiefly by the lymphatic vessels, and not by the bloodvessels. The only result of administering powerful diaphoretics and cathartics to patients suffering with effusion following pleurisy is to exhaust their vitality without materially influencing the local condition.

The application of blisters to the chest, with the hope that they will stimulate absorption, is probably quite as futile as the employment of purgatives, although they may indirectly result in the absorption of fluid by stimulating the removal of the film of plastic exudate which covers the pleural membrane.

The condition in pleural transudations following, or accompanying, cardiac or renal dropsy is quite a different one from that due to inflammation. In the latter condition there is not any fibrinous exudate, and the effusion takes place by a process of transudation from the vessels, the fluid being quite different in its character from that found after the acute inflammatory process just discussed. Purgatives may therefore do good.

Aside from the operative measures, which are necessary in about one-half the cases of pleurisy with effusion, the physician should administer mild tonics, with the object of aiding digestion, and he should support the system by the administration of proper quantities of nutritious food. If after tapping the fluid it recurs, it should be withdrawn a second time. Such a recurrence rarely takes place in the effusion following pleurisy, although it is not infrequently met with in cases of ordinary transudation into the pleural cavity in other pathological states.

PURULENT PLEURAL EFFUSION, OR EMPYEMA.

Definition and Etiology.—By empyema we mean a condition in which pus has accumulated in the pleural space or spaces. It was taught at one time that such an effusion might primarily be serous and by infection become purulent, but at present this view is not generally held. Empyema occurs as a sequel to infection from the lung in the great majority of cases, but it may arise from primary infection of the pleura. The condition is far more common in children than in adults (Fig. 61). In children it is generally the result of the presence of the *pneumococcus*, which commonly causes a

Fig. 61

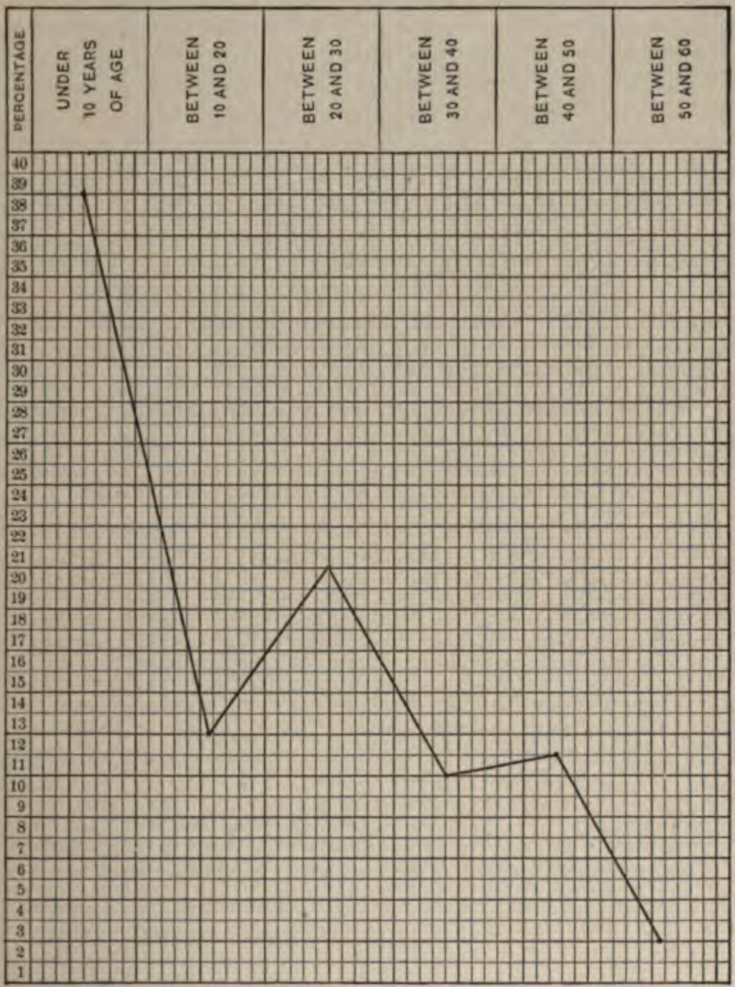


Chart showing morbidity percentage of empyema due to all causes at different ages, based on 403 cases occurring in five hospitals in the United States and England.

bronchopneumonia or a croupous pneumonia first and an empyema afterward, but in adults the streptococcus is usually the exciting cause. The condition occurs much more frequently in boys than in girls.

In 69 cases of empyema in children, P. S. Blaker found the pneumococcus in 62 cases; the streptococcus in 3; the pneumococcus and streptococcus in 3, and the staphylococcus in 1. In 40 cases in children reported by Bythell, 26 were due to the pneumococcus and 9 to the pneumococcus and some other organism (Fig. 62).

FIG. 62

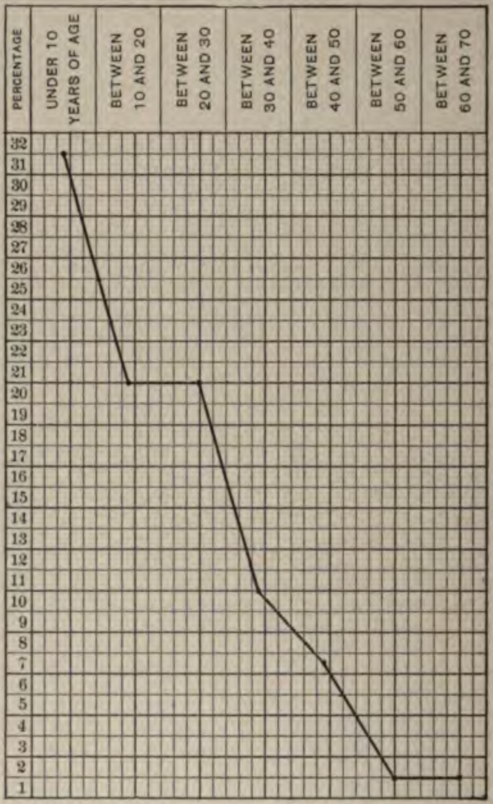


Chart showing morbidity percentage of empyema due to the pneumococcus at different ages, based on 286 cases collected by Netter. Large percentage in childhood.

Empyema is sometimes due to infection by the ***Bacillus tuberculosis***, and it is a fact worthy of note that the pus in such cases is usually sterile, only revealing the presence of tubercle bacilli, when by chance some of the exudate which lines the pleura is obtained through the aspirator. In other words, sterile pus from an empyema raises a suspicion of tuberculous infection. Bacteriological examination of pus from 311 cases of empyema, occurring in hospitals in the United States, Canada, England, France, Germany, Austria, and Italy, showed that the pneumococcus was the infecting organism in 92 cases; the streptococcus in 58; the tubercle bacillus in 30. In the remaining cases the pus was sterile, or more than one micro-organism was found. If children are excluded from these statistics, the streptococcus becomes the most common infectious agent.

When no pulmonary lesion can be discovered in a case of empyema, it must be recalled that a very small and insignificant lesion in the lung, and, therefore, one which is easily overlooked, may be the focus for a very severe pleural infection, and, therefore, the inability of the physician to find a primary pulmonary lesion does not prove that it has not existed.

The character of the pus found in cases of empyema varies considerably in different cases, the variation depending in part upon the micro-organism which has produced the condition, and upon the duration of the malady at the time the effusion is examined. Usually it is creamy and homogeneous; in other cases it is thin and separates on standing into a thick and thin layer. When the effusion is an old one, the pus may be quite thick and curdled in its appearance, containing clot-like masses or shreds of fibrin, which plug the aspirating needle and make aspiration impossible. While the color is commonly a creamy yellow, it is sometimes slightly pinkish in appearance, and may be greenish in hue, and in still other cases, when a considerable amount of blood has been extravasated into the effusion, it is a dirty, pale-cocoa color. In some instances, as in cases due to infection by the pneumococcus, the pus is almost odorless, while in others, particularly if the empyema has ruptured into a bronchus, it is fetid.

Purulent effusion in the pleural space is usually profuse. Indeed, it has been taught that as a class these collections are larger than are serous effusions, which is not, however, always true by any means. On the other hand, they are very much more likely than are serous effusions to be walled off and encysted by reason of adhesions, thereby forming a small pocket of pus.

Symptoms.—If after an attack of pneumonia the *temperature* does not fall, or if, after it has been normal or near normal, it begins to rise again, and the patient has *chills* or chilly sensations, empyema should be sought for.

The symptoms of empyema in general are those of impaired health. The patient is, as a rule, *pale* and ill-looking, suffers from *loss of weight*, and *sweats*, which are particularly prone to come on when he sleeps.

A *moderate fever* may be present, and suppressed or even well-developed *chills* may recur. It is important, however, to remember that in some cases none of these constitutional symptoms are manifest, the fever in particular, being so mild that they fail to attract attention, so that the condition of empyema is suspected only when some shortness of breath calls attention to the thorax.

The pus in cases of empyema sometimes becomes so completely walled off from the surrounding tissues that it remains for weeks without producing any signs of its presence, but in other cases, and these are the more numerous it causes such severe pressure symptoms or so much evidence of sepsis that relief is demanded by the patient. In other cases—and these are often those in which the empyema has not been recognized—the pus burrows its way out, rupturing into a bronchus or perforating the chest wall. Very much more rarely it empties into the pericardium, or even into the œsophagus. In other instances it has perforated the diaphragm, although this process is, curiously, much more rare than the rupture of a subdiaphragmatic abscess into the pleural cavity.

Statistics as to the relative frequency with which rupture into a bronchus takes place are not in accord. Thus, of 195 cases of empyema occurring in St. Thomas' Hospital, London, and in the Leeds General Infirmary, 11 ruptured into the lung, a percentage of 5.64; while an analysis of a large number of cases of empyema collected by Netter gave a percentage of 26.2 rupturing into the lung. These latter figures certainly must be far too high.

The physical signs of empyema have already been discussed when describing those of serous effusion, for in both states they are practically the same. Sometimes the presence of pus may be shown by an *œdema of the superficial tissues,* which is often met with over deep-seated suppurations. Empyema is also apt to produce more bulging of the intercostal spaces than is serous effusion, perhaps because there is more wasting, and so the bulging is more readily observed. In some instances of empyema, however, the contraction of the thickened pleura draws the edges of the ribs so closely to one another that bulging of the interspaces is obliterated.

When pulsation is transmitted to the purulent effusion, so that the impulse is manifest through the interspaces, it is called "*empyema necessitatis.*"

Complications.—The chief complications of empyema have already been named, viz., sepsis and perforation. The signs of sepsis are similar in this state to those produced by accumulations of pus elsewhere, and require no further discussion. The symptoms of perforation into a bronchus consist of an attack of violent coughing, during which the patient expels, in large, or sometimes in small amount, a quantity of almost pure pus. After the pus first appears, it is commonly brought up in mouthfuls several times a day, and more rarely in such large quantities as to threaten the patient with suffocation. This drainage of pus through the lung, curiously enough, rarely causes serious permanent damage to the lung, which may ultimately entirely recover if the physician will but provide an opening in the chest wall for proper drainage.

When the pus escapes externally by burrowing, it most frequently does so about the sixth intercostal space in the axillary area, but it sometimes burrows a great distance and escapes by way of numerous openings. In other instances it burrows far down the trunk and discharges as low down as the pelvis. Indeed, Barton has reported a pulsating empyema in the left lumbar region. These openings may persist for many years, and if the suppurative process persist, amyloid disease of the liver and kidneys may ensue.

Diagnosis.—The differentiation of empyema from serous effusion is to be made by the presence of the septic symptoms just named, and by the use of an aspirating needle to determine the character of the fluid. The localized types of empyema are those which offer real difficulty in diagnosis, since the pus may be between two lobes of the lung, or at the base of the lung next the diaphragm, or be extended over a considerable area while very shallow, or, again, the inflammatory process in the adjacent lung tissue causes the presence of the physical signs of consolidation of the lung or of large effusion, when in reality the purulent collection is a small one. In these cases the introduction of the aspirating needle may fail to reveal the presence of pus, because the instrument does not happen to exactly strike the purulent focus.

When the pus is localized by adhesions in the neighborhood of the heart, this organ may be displaced by the pressure and transmit its impulse to the effusion, so giving rise to the belief that a purulent pericarditis is present; the absence of this more serious state being revealed only when the pus is set free.

A still more difficult condition to discover is interlobar pleurisy with effusion. In such cases if the accumulation is pus, it may rupture into the bronchi and give rise to the belief that the patient has true pulmonary abscess.

Prognosis.—The prognosis in empyema in children, over three years of age, if the condition is due to the pneumococcus, and if the pus is allowed to escape before it has done much damage, is surprisingly good, both as to rapid and complete recovery. Not only do many of these cases soon cease to form any more pus, but the compressed lung expands with remarkable rapidity, and may, in the course of a few months, fill the pleural cavity so well that nearly all traces of the disease may disappear. When the disease affects infants the outlook is bad, because of their susceptibility to wasting processes and their low vital resistance.

In streptococcus infection the prognosis is not so favorable, nor is it good in tuberculous empyema, for in the first type the formation of pus is persistent and the deformity of the chest is very apt to be great, while in the second type a primary infection elsewhere is usually present.

It has been stated by some authors that an empyema may undergo absorption. While a decrease in the size of the effusion may result from the absorption of some of its fluid constituents true disappearance of pus from the thorax does not take place unless it is let out or escapes spontaneously. It may, however, become inspissated and encysted.

Treatment.—There is but one thing to be done in cases of empyema, and that is to let out the pus, treating the case as one of ordinary abscess. If the quantity of the fluid is sufficiently large to compress and displace adjacent organs, particularly the heart, it is better to first relieve some of this pressure by aspiration, as in a case of serous effusion, removing enough of the pus to permit the heart and vessels to slowly regain their normal position. As the pus in these cases is often under great pressure, so that it squirts several feet when an incision is made, I am confident that this preliminary modification of the pressure is wise in most cases. On the following day or, if need be, immediately after aspiration, an incision should be made between the sixth and seventh, or seventh and eighth, ribs in the middle axillary line, and this opening should be maintained by the insertion of a doubled, or extra-large, drainage tube or by a gauze drain. If the ribs have been drawn so closely together by the contraction of the parietal pleura that free drainage cannot be obtained, then the upper surface of the lower rib should be cut away until drainage is free, or, if need be, several inches of the rib or of several ribs should be resected. This is usually necessary in streptococcus infection. As a rule, the milder measures suffice in children, but if the formation of pus persists resection should always be performed. For the details as to the exact technique of these operative procedures reference should be made to a surgical work.

CHRONIC PLEURISY.

Definition.—Chronic inflammation of the pleura may be nothing more than a sequence of some of the acute conditions already discussed. If a fluid accumulation, serofibrinous or purulent, be allowed to remain within the chest cavity, important alterations take place in the serous membrane. With the subsidence of infection reparative efforts lead to the production of fibrous tissue, which greatly thickens both parietal and visceral layers and ensheaths the collapsed lung, eventually forming such a dense investing membrane that re-expansion becomes impossible. In other cases the fluid is absorbed and the pleural surfaces coated by inflammatory products come in contact, coalesce, and become fused by permanent organization of the exudate. In the latter group of cases the pleural cavity may be obliterated, or partial adhesions only may form. In some cases unattended by frankly expressed acute inflammation, hyperplastic thickening of large or small areas, usually with adhesions, occurs. In such cases the newly formed inflammatory tissue may attain a thickness of 1 cm. or more and not infrequently contains calcareous plaques.

The third form is called "primitive dry pleurisy" in the sense that it begins without effusion and often without pain, and is not associated with fever. The patient may himself feel the pleural friction. Finally, limited adhesions occur between the layers of the pleura, but they do not cause marked interference with the lung nor deformity of the chest.

The fourth type is the so-called "*primitive dry pleurisy*" described by Sir Andrew Clarke, in which the layers of the pleura become adherent and thickened as in the forms just described. From the visceral layers of the pleura bands of connective tissue penetrate and traverse the lung almost as if they were true trabeculæ. The effect of these bands as they contract is to produce bronchiectasis and some distortion of the lung in its lower lobe, where the process is nearly always situated. The condition is really a pleurogenous interstitial pneumonia. These cases are not identical in character with those due to old empyema.

Occasionally in chronic pleurisy large calcareous or even bony plates are developed in the newly formed inflammatory tissues.

HYDROTHORAX.

This condition is to be clearly separated from ordinary pleural effusion due to inflammatory changes. Pleural effusion due to inflammation is usually unilateral, but hydrothorax is generally bilateral. The fluid in the pleural cavities is present as a result of transudation in cases of renal disease, cardiac disease, profound anæmia, or any cause which tends to impede circulation or to increase the readiness with which the serum can escape from the bloodvessels. Thus it may develop in cases of thrombosis of the vena azygos during the course of typhoid fever. While it is true, as already stated, that hydrothorax is usually bilateral, it sometimes happens that it is unilateral, if perchance the obstruction to the flow of blood or lymph is produced by some lesion which affects only one side of the chest. This occurs much more frequently on the right side than on the left, in those cases in which the cause is cardiac disease. Many years ago Jaccoud pointed out that this is due to pressure upon the major azygos vein by the dilatation of the inferior vena cava and the right auricle, and more recently Steele and Stengel have called attention to this class of cases.

When it is present in the course of cirrhosis of the liver it is probably due to the presence of associated pulmonary tuberculosis. Osler asserts that such an effusion may occasionally occur in what he designates as "perfectly healthy men."

The fluid in hydrothorax is usually of low specific gravity (see Pleurisy with Effusion), and clear or but slightly opalescent, yellowish or straw-colored. It is not rich in cells, and those cells that are present consist largely of relatively voluminous flat endothelial cells. Fibrin is usually absent. If an injury or obstruction of the thoracic duct is present, the fluid may be chylous in character.

Again, if such a patient is given iodide of potassium and the fluid is withdrawn by aspiration iodine will be found in it, whereas if the fluid is due to inflammation iodine is absent. The fluid is placed in a test-tube, a few drops of fuming nitric acid are added, and then it is shaken with some chloroform, when if iodine is present a red color will appear, which sinks to the bottom of the tube with the chloroform.

Hydrothorax can often be relieved by the free use of a saline purgative, such as half an ounce of magnesium sulphate given every morning before breakfast and by cardiac tonics if they are needed. If it causes symptoms by pressure it must be removed by aspiration. (See Pleurisy with Effusion.)

Bloody effusion into the pleura is met with in cases of cancer of the pleura and of Bright's disease. It is more indicative of the presence of the former malady. I have more than once seen a simultaneous pleural and abdominal bloody effusion due to a general carcinomatosis.

When pleural effusion is tapped a second time, or when the needle has been introduced more than once in the search for fluid, it not infrequently happens that the liquid obtained is blood-stained, owing to wounding of a bloodvessel by the instrument. Of course, this possibility must be remembered when a bloody effusion is found. Sometimes a true hæmothorax arises from this cause, or it is due to a leakage from an eroded bloodvessel.

PNEUMOTHORAX, HYDROPNEUMOTHORAX, PYOPNEUMOTHORAX.

Definition.—Pneumothorax—that is, the presence of air in the pleural spaces—is rarely present as the result of disease unless it is associated with fluid (hydropneumothorax) or pus (pyopneumothorax). As the result of injuries to the chest and to the lungs it not rarely appears as true hydropneumothorax, as after the fracture of a rib, or as the result of a stab wound.

History.—As long ago as the time of Hippocrates a succussion sound on shaking a patient suffering from empyema or pleural effusion was recognized, but it was not until the time of Laennec, about 2200 years later, that the value of this sign was appreciated as indicating the presence of both fluid and air in the chest.

Etiology.—The most common cause of pneumothorax is pulmonary tuberculosis, and it arises as a result of the perforation of the pleura by the rupture of a cavity through the visceral layer; in order that air may enter the pleura the tuberculous cavity must directly or indirectly communicate with a patulous bronchus, thereby affording a communication between the lung and the thoracic cavity. West believes that fully 90 per cent. of the cases are due to this cause. In many cases this accident is prevented by an acute or subacute pleurisy occurring at the area diseased, so that the pleura is thickened or the two layers glued together. This is particularly prone to be the case when a cavity has formed; and were it not for this protective process the condition of pneumothorax would be commonly met with. In still other cases, however, these very adhesions result in pneumothorax, for during some severe exertion they are torn, and so the air finds an opening through which to escape.

As a rule, the perforation occurs in the lower part of the upper lobe, or in the upper part of the middle lobe. Pneumothorax develops on the left side nearly twice as often as on the right. West, however, believes that the two sides are nearly equally affected. At times the opening through which the air escapes is so small that it cannot be found. Sometimes there is more than one perforation.

Very much more rare as causes of pneumothorax are bronchiectasis, pulmonary abscess, and pulmonary gangrene. So rare are they that when cases of this kind occur they should be reported. This holds true as well of cases which develop from rupture of a vesicle in cases of emphysema of the lungs. Pneumothorax has arisen in the course of whooping-cough, diphtheria, and typhoid fever.

Pneumothorax occurs three times as often in men as in women.

Symptoms.—The onset of pneumothorax is often very sudden and severe, but at times it develops so insidiously that no signs of its presence are noted by the patient until he attempts to make some exertion, when *dyspnœa* ensues. In cases of sudden onset there is not only *urgent dyspnœa*, but sometimes *syncope* to the point of unconsciousness. These severe symptoms are much more prone to develop in a patient who has slight pulmonary disease than in one who has well-advanced lesions, because in the latter case, the lung being already partly useless, the other lung is ready to compensate for the inactivity of the diseased part. When the accident occurs on the comparatively healthy side death may speedily ensue. In some instances the pain may be so severe that angina pectoris is thought to be present.

PHYSICAL SIGNS.—The physical signs in these cases of pneumothorax consist in bulging of the interspaces on the affected side, and at times the development of *subcutaneous emphysema.* There is also in many cases a distinct increase in the size of the chest on that side. If the air escapes on the right side the *liver is markedly depressed* and the heart is displaced to the left. In left-sided cases the heart may be pushed to the right of the median line.

Percussion reveals hyperresonance unless there has been an old pleurisy with secondary pleural thickening. If the lung is adherent to the chest wall and collapsed by pressure, or consolidated by tuberculosis, a dull note may be present. In a case of this character recently under my care, in a patient whose general health seemed to negative the possibility of tuberculosis, this state was confirmed at autopsy, much relief being given before death by frequently permitting the air to escape from the chest, over part of the chest wall through a hollow needle.

Auscultation reveals an absence of vesicular murmur over the area of hyperresonance, and, perhaps, loud amphoric breathing over the lung, particularly if it contain a cavity which freely communicates with the pleural space.

When *hydropneumothorax* is present the lower part of the chest is flat on percussion as in ordinary pleural effusion, above this is an area of hyperresonance, and above this again is the Skodaic resonance due to the compressed lung. On shaking the patient a succussion sound is heard, and when the patient is at rest *auscultation* reveals "metallic tinkling," which is supposed to be due to the dropping of fluid into the liquid at the base of the chest. *Succussion* and *metallic tinkling* are the most important signs of hydropneumothorax. Another valuable sign is the so-called "coin sound" produced by striking a large coin, held against the chest wall, with another coin. The physician listens to the back of the chest as the percussion is done, by an assistant, on its anterior surface, and closes his unoccupied ear with his finger-tip. If the coin be struck so that the sound has to be transmitted through the chest at the level of the fluid the sound is very indistinct. At the level of the air it is transmitted with startling clearness, and at the level of the lung its transmission is again impaired.

Diagnosis.—In considering the possible presence of pneumothorax in the type which is insidious the following conditions must be included: A large cavity may give somewhat similar physical signs, but the limited area over which they are manifested or demonstrable separates the two states. Emphysema of the lungs is excluded by the universal presence of breath sounds and the fact that the condition is bilateral. Rupture of the diaphragm with diaphragmatic hernia should be considered if some injury has been suffered, and pyopneumothorax subphrenicus must be excluded by study of the pulmonary signs above the area involved and of the condition of the epihepatic and epigastric areas.

Prognosis.—The prognosis of pneumothorax depends largely upon the cause of the condition and the associated states of effusion and empyema. Much depends upon the suddenness of onset. I have seen death occur in twelve hours in cases with sudden onset, and cases are on record of death in twenty minutes. On the other hand, if the dyspnœa is not severe the pulmonary condition may be actually benefited by the temporary rest enforced by the collapse of the lung. West has placed the mortality at 70 per cent., and of these fatal cases 75 per cent. died within two weeks and 90 per cent. within a month. In those cases which do not die soon after the onset of this condition death may result either from empyema and exhaustion, or from the progress of the underlying disease. If the heart is feeble, if the other lung is far advanced in disease, and if the strength of the patient is badly impaired the prognosis is, of course, bad. Recovery takes place in about 10 per cent. of the cases of simple pneumothorax without fluid.

Treatment.—The treatment of pneumothorax consists in the relief of pain, if it be very severe, by a small dose of morphine—say, $\frac{1}{8}$ of a grain given hypodermically. If the dyspnœa is marked a large hollow needle, or aspirating cannula, should be introduced into the chest, but not attached to the aspirator. The pressure in the chest will cause more air to escape than will flow in, and as the lung is already collapsed any damage caused by its entrance is done. To prevent the air from entering, the finger may be temporarily used as a valve on each inspiring movement until a wash-bottle can be so arranged that the air will escape through the water it contains and then cannot return. If the air constantly re-accumulates the case should be treated by a drainage tube inserted as in the treatment of empyema. (See Empyema.)

If œdema of the other lung is threatened, dry cups should be applied over its base.

When serous effusion is present, or when empyema is a complication, the conditions should be treated as described when discussing these conditions.

A most exhaustive study of this subject has been made by West in London, and more recently by Emerson in Baltimore during 1903.

DISEASES OF THE MEDIASTINUM.

Under this heading are considered diseases of the mediastinum other than those of the heart and aorta. In my Fothergillian Prize Essay I collected 520 cases of mediastinal disease, and the facts there presented form the basis for the following views. The statistics on their face show that there were 134 cases recorded as carcinoma, 98 as sarcoma, 21 as lymphoma or lymphadenoma, and 115 as abscess. In other words, a large proportion of cases of disease in this area are due to malignant growths, for the remaining lesions were non-malignant or inflammatory. The statistics apparently indicate that cancer is by far the most frequent form of individual growth. While we have no right to go "behind the returns," in the sense that cases reported as cancer may be regarded as sarcoma, it is nevertheless probable that sarcoma is really the most frequent growth in the mediastinum, because tissues favorable to its growth are found there in great abundance and tissues susceptible to carcinomatous growth are scanty. Again, it is well known that up to the middle of the last century, and later, little distinction was made between cancer and sarcoma, and so many cases of sarcoma were probably reported as cancer. Finally, lymphoma and lymphadenoma are so nearly allied to sarcoma that it is fair to add them to the so-called sarcomatous cases, making the total 119 reported as sarcoma.

The non-malignant tumors of the mediastinum are fibroma, teratoma, dermoid cyst, and hydatid cyst.

Notwithstanding the fact that the middle and posterior mediastinal spaces are more richly provided with lymphoid tissues than the anterior mediastinum, the statistics prove that malignant growth is more common in the latter space than in the other spaces. Thus, of the cases reported as sarcoma and cancer, in which the space affected was stated, 81 were in the anterior space as against 28 in the posterior space, and only 5 in the middle space alone, although in many other instances the entire mediastinum was invaded.

For this reason we should expect to find that sarcoma in a very large proportion of cases occurred as a secondary growth in the mediastinum; but an examination of the literature of the subject, both as regards general opinion and reports of cases, shows such a conclusion to be erroneous. Indeed, the mediastinum seems to rarely suffer from any form of this disease save the primary, and even in those cases in which the lesions were scattered all through the body from head to foot, this space seems to have escaped secondary contamination. Should the growth appear in the mediastinum, secondarily, it generally affects the posterior or middle spaces, owing to the large number of lymphatic glands and like tissues which are found in these cases.

It is a curious fact that mediastinal growths are twice as common in men as in women, although women so much more frequently have malignant growths in nearby tissues, and in them, as already stated, malignant growth of the lung is said to be more common.

The average age affected by mediastinal tumors is about thirty-seven years.

Symptoms.—The symptomatology of mediastinal tumor is by no means clear and well defined, since so many other conditions may produce signs of the same character, and it has been stated very positively by certain writers that such a growth cannot be diagnosticated during life.

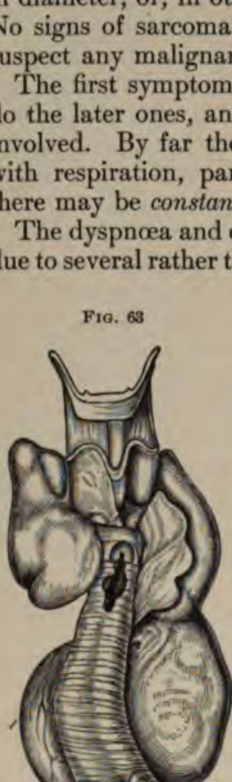
Although this assertion seems rather sweeping, there is, nevertheless, some truth in it, and in many cases, where we have no history to guide us and no evidence of a growth elsewhere, the diagnosis may be well-nigh impossible.

Large tumors are found, in the anterior mediastinum, which have not been diagnosticated or suspected until a postmortem has been made, not from any lack of ability on the part of the physician, but because the symptoms of mediastinal disease have either been entirely absent or masked by others of more importance elsewhere. Thus, in a case reported by Bruen, an old woman, aged seventy years, entered the Philadelphia Hospital with decided symptoms of renal disorder, which in a few days caused her death. Although an examination was made of the chest, as a matter of routine duty, no special physical signs were discovered, and the disease, which was sarcoma in the anterior mediastinum, was not discovered until the body was placed on the postmortem table. The only symptoms of such a condition of affairs before death consisted in slight dyspnœa and cough, both of which were supposed to arise from the renal lesions; and this is the more remarkable, since the growth weighed fourteen ounces, was six inches long by five inches broad and four inches in diameter, or, in other words, was about the size of a normal adult heart. No signs of sarcoma existed elsewhere in the body from which one might suspect any malignant disease.

The first symptoms complained of by the patient vary quite as much as do the later ones, and depend, as do their successors, upon the parts most involved. By far the largest number of sufferers notice some interference with respiration, particularly on exertion, which soon increases, so that there may be *constant dyspnœa*, and even attacks of *partial suffocation*.

The dyspnœa and other disturbances of respiration are, in many instances, due to several rather than any single cause, since, in addition to the mechanical pressure by the growth on the air-passages, we may also have such interference with the circulation of the blood, particularly in the thoracic veins, that pleural, pericardial, or mediastinal effusions of serum may occur.

FIG. 63



Kronlein's case of retrotracheal tumor of the mediastinum.

Effusions into the abdomen may occur, owing to involvement of the ascending vena cava, but such a condition is rather rare, probably owing to the fact that the ascending cava more frequently escapes than does the descending. *Dropsy* of the lower extremities, without abdominal effusion, sometimes comes on.

In still another class of cases the pulmonary vein may be obstructed, and œdema of the lung may develop. Hypostatic congestions are by no means rare, the patient often being forced, by cardiac weakness, pleural effusion, or pressure on the trachea, to lie in one position. In some cases loud venous murmurs can be heard in the jugular and other large superficial veins, and care has to be exercised as to the diagnosis of the true cause of the distress. The ribs and sternum may undergo gradual erosion and destruction from pressure, and the growth appear on the surface of the body.

In a certain number of cases the nerves of the thorax seem to be more affected than the rest of its contents, and involvement of the vagi or the recurrent laryngeal nerves may bring on a long train of obscure and dangerous symptoms, both as regards the circulation, respiration, digestion, speech, and swallowing.

Tumors of the mediastinum invading the lungs have frequently been mistaken for chronic and even acute pneumonia, growing, as they do, along the larger bronchial tubes and bloodvessels.

Without doubt, in a certain number of cases, either hypostatic pneumonia, or pneumonia due to pressure on the bronchial vessels, develops as the tumor invades the lung, and in such cases it is absolutely impossible to make a diagnosis unless there are symptoms of pressure in the mediastinum. Walsh has stated that if the lesion be due to a tumor, the affected side will increase in bulk rather than diminish, and that dyspnœa out of proportion to the degree of consolidation points to a mediastinal disorder rather than one confined to the lungs.

In a very large proportion of cases of mediastinal disease the condition is one of *abscess*. There were 115 cases in my collection of 520 of mediastinal disease. The proportion of acute and cold abscess in 79 cases in which the differentiation was made was 48 to 31.

The most constant and severe symptom is, in nearly all cases, the deep-seated pain, which increases in severity from first to last, seldom remitting until suppuration has taken place and the pus has found some outlet. If the case be one of cold abscess, these painful symptoms may be masked by other more pressing ones, such as dyspnœa and œdema from pressure; although it should not be forgotten that such symptoms may appear with equal severity in both varieties of the disease. In the acute variety all the symptoms of ordinary inflammation appear, such as rigors and periodical or constant fever.

As recovery took place in about 40 per cent. of the cases of mediastinal abscess according to the statistics of preantiseptic days, it ought to occur much more frequently now.

Mediastinal growths are usually of such a nature as to be beyond either medicinal or surgical treatment, but abscess, dermoid cysts, and teratomata are sometimes operable.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Acute Pericarditis. **Definition.**—Acute pericarditis, sometimes called acute fibrinous or acute serofibrinous pericarditis, is, as its name implies, an acute inflammation of the pericardium, the serous membrane which envelopes the heart.

Etiology.—Acute pericarditis is practically always due to the presence of some infecting micro-organism, although certain conditions existing simultaneously may predispose to the infection by lowering vital resistance. In the great majority of cases it is due to acute articular rheumatism, in comparatively few it develops as a complication of croupous pneumonia, the pneumococcus being its cause, and it also develops as a complication of scarlet fever, in which disease the streptococcus associated with this malady is probably the provoking factor. Acute pericarditis is also frequently associated with renal disease, and is often, under these circumstances, a form of terminal infection. So, too, it may develop in the course of various infectious diseases, such as smallpox, erysipelas, typhoid fever, and even in measles. Septic infections, such as general septicæmia and ulcerative endocarditis, may cause it. In diabetes it occurs as a terminal infection. Of course, tuberculosis and syphilis, diseases which affect every tissue, may also affect this one.

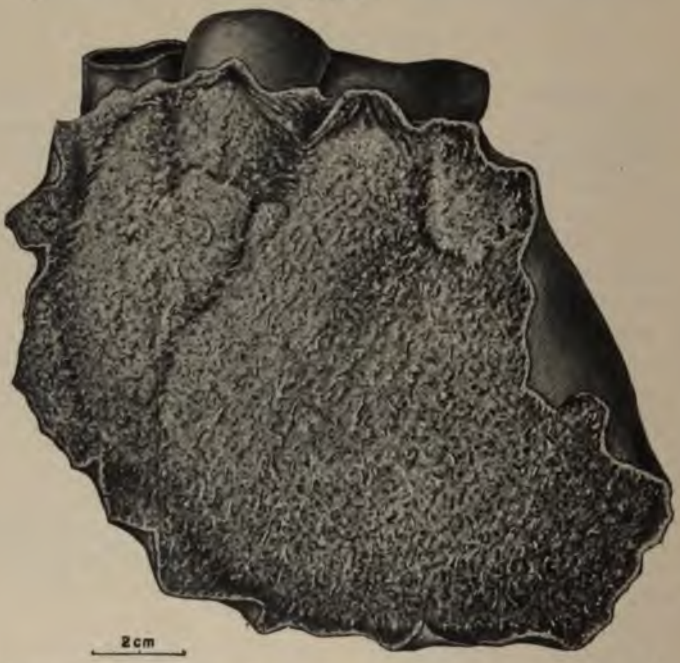
Acute pericarditis also develops by direct extension from inflammation in neighboring parts, in distinction from infection which takes place through the blood. Thus, inflammation of the mediastinal tissues may produce it, as in diseases of the bronchial glands, of the sternum, or of the vertebræ. So, too, pneumonia and pleurisy affecting nearby portions of the lung may cause inflammation of the pericardium. In infections involving the myocardium the overlying serosa—that is, the visceral layer of the pericardium—rarely escapes. So, too, pericarditis may be the first indication of impending rupture of that part of the aorta covered by pericardium.

Pericarditis may also be due to injury to the chest wall or to the membrane itself.

It is evident, therefore, that acute pericarditis is nearly always a condition secondary to some other affection, and that it is very rarely primary. When it is primary it is usually due to tuberculosis. (See articles on Croupous Pneumonia, Acute Rheumatic Fever, and Typhoid Fever.)

Frequency.—The frequency of pericarditis as a primary disease is very limited, but as a secondary affection it is great. Very many cases present no sign of it during life, yet the condition is found at autopsy. It occurs almost as frequently in children as in adults, although at one time this class of patients was supposed to be not so commonly attacked as older persons. Sturges found it present in 94 out of 100 cases of fatal heart disease in children. Of these cases 54 were of rheumatic origin. Indeed, it is probable that the disease is less prevalent after than before puberty. It occurs far more frequently in males than in females, and this is particularly true after puberty, when the greater exposure and activity of males become dominant factors in causing rheumatism and other infections. At this time the proportion is from 4 to 1 to 6 to 1.

Fig. 64



Heart and pericardium, acute serofibrinous (pneumococcal) pericarditis. Most of the anterior parietal layer of the pericardium has been cut away, showing the villous (shaggy) irregular projections of the fibrin. To the left, where the parietal pericardium is reflected over the right auricle and great vessels, cohesion of the layers may be seen; later, had the patient recovered, such fusion of the layers would have constituted the basis from which organized fibrous adhesions would have formed.

Pericarditis, as we would expect, develops more commonly in severe cases of acute rheumatism than in mild cases, but, on the other hand, it is to be borne in mind that even in those cases with very mild joint symptoms severe pericardial involvement may occur. Prior attacks of rheumatism seem to increase the frequency of pericarditis in subsequent attacks. The condition usually comes on during the first week of the disease. This is, however, by no means always the case, and it may appear as late as the sixtieth day of the illness or during a relapse.

Pericarditis due to renal disease, the *Pericardite Brightique* of the French, is distinctly a state of advanced years, occurring most commonly after forty-five or fifty years of age. It is more commonly met with in patients suffering from contracted kidney than in those that present the parenchymatous form of renal disease.

Pathology.—As in inflammation of the pleura, so in inflammation of the pericardium, it is well to recognize three forms of acute pericarditis, viz., the *acute dry* or *fibrinous*, the *acute exudative* (*serofibrinous*), and the *purulent type*. In the first stage of all these forms of pericarditis the lining surface of the pericardium is lustreless, opaque, and somewhat roughened by a delicate fibrinous exudate. It is also hyperæmic and may be dotted with petechiæ. It is the rubbing together of the two layers of the pericardium at this stage that causes the characteristic friction sound of the disease. As the inflammation progresses the membrane becomes completely covered by the exudation of fibrin, which may assume a villous formation. Adhesions between the layers of the pericardium also take place.

In the serofibrinous form a considerable quantity of serum is poured out into the sac, and particles of fibrin and leukocytes are found in it. The quantity may be so large as greatly to distend the sac, displace the heart, and interfere with its function, particularly by pressure upon the auricles and cavæ. In many cases as much as three pints have been found in the sac. West quotes cases in which the pericardial sac contained no less than five pints (due to scurvy), yet recovery occurred after aspiration.

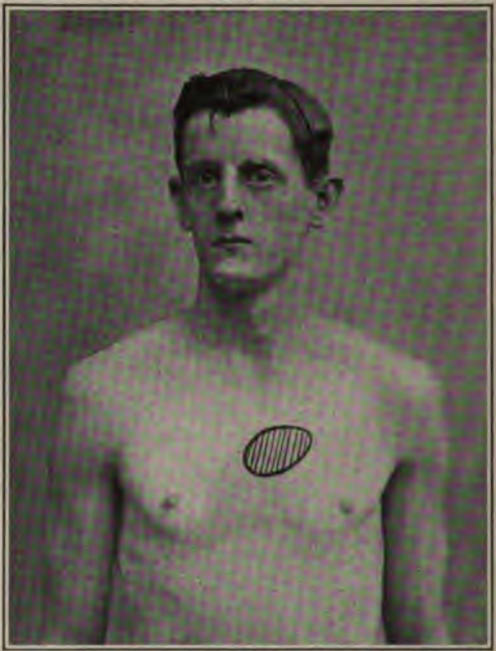
In the purulent form (*pyopericardium*) the serum and fibrin are mixed with pus cells and erythrocytes. Pyopericardium may arise as a primary purulent pericarditis or be converted from the serous form by infection with the *Streptococcus*, *Pneumococcus*, or *Staphylococcus pyogenes aureus*. Sometimes the *tubercle bacillus* acts as a pyogenic organism in this space.

There is some difference of opinion as to the frequency of pyopericardium. Two opinions which represent the two sides of this question are as follows: Samuel West speaks of it as "one of the rarest of clinical rarities," but Battin says "it is a disease seldom suspected, still more rarely diagnosed, and hardly ever treated, and yet it is one that is present in 3 per cent. of the deaths in the records of the Children's Hospital."

That the latter view is correct is shown by the fact that Breitung in 324 cases of pericarditis found that 108 were serofibrinous, 30 hemorrhagic, and 20 purulent, or 6.1 per cent. In 769 autopsies collected by Still 24 instances of pyopericardium were found, and 11 were due to infection by the pneumococcus. Scott found it in no less than 16 cases out of 40 (40 per cent.), and in 38 cases of croupous pneumonia the same reporter found pyopericardium in 17, or 44.7 per cent.

Symptoms of Acute Fibrinous Pericarditis.—The symptoms of acute fibrinous pericarditis are often not pronounced. *Pain* would supposedly be a well-marked symptom, but it is not present in all cases, although when it is present it may be severe. Fortunately pain from this cause is rare in children. When it occurs it is usually felt from the right edge of the sternum to the left nipple and is fairly constant, although it has sharp exacerbations. In other cases the pain is chiefly situated in the epigastrium, or a sense of *præcordial distress* develops, and the *breathing* may be *oppressed*. The action of the *heart is rapid*, often reaching 100 or 120 and even 160 beats per minute in severe cases. The *temperature* is usually increased, but seldom rises above 102° or 103°. In some cases it is not abnormal. Nervous symptoms are sometimes notable and *great restlessness* or even *active delirium* may ensue, resembling that of delirium tremens. *Vomiting* is a common symptom.

FIG. 65



Area in which pericardial friction sound is best heard.

PHYSICAL SIGNS.—The physical signs of acute fibrinous pericarditis are as follows: *Palpation* over the base of the heart at the third interspace may reveal friction fremitus in well-marked cases, and *auscultation* will show a distinct friction sound in the same area when the disease is established. This sound is creaking and dry and has been called the "saddle-leather sound," in that it resembles the creaking of an ordinary English leather saddle when it is first used. It differs from the friction sound of pleurisy in that it is to and fro and does not occur with the respirations. At times it has a gallop rhythm with a triple sound as of a horse galloping. If the stethoscope be pressed against the chest the sound can usually be intensified. These sounds vary with the severity of the inflammation of the pericardium and the action of the heart, being more sharply defined when the action is violent than when it is depressed.

Indeed, variation in the action of the heart may cause a loud friction sound at one visit and a lack of it at the next. As a rule the pericardial friction sound is limited to the area of the cardiac base about the third or fourth interspaces, but it may be heard at the apex or along the sternum.

Diagnosis of Acute Fibrinous Pericarditis.—Acute dry pericarditis can scarcely be confused with any other state, but several conditions resemble it somewhat. Thus in cases of early phthisis there is occasionally heard, near the apex of the left lung, a cardiopulmonary murmur or puffing sound during inspiration, occurring with each beat of the heart, and persisting if the patient holds his breath on a full inspiration, but disappearing on expiration. Another similar condition is the so-called "pleuropericardial friction sound," which is apparently due to the beating of the heart against the margin of the lung. Both of these sounds are, however, more in the nature of murmurs than friction sounds.

Symptoms of Serofibrinous Pericarditis.—With the development of effusion, in cases which go on to that state, symptoms of *cardiac embarrassment* begin to show themselves. The evidences of cardiac disturbance are not always, however, in direct proportion to the amount of fluid, for in some instances large accumulations of fluid cause so little inconvenience as to be overlooked, while in others in which the fluid is moderate in quantity they are severe. It is important that these variations in the severity of the symptoms be remembered, because it is humiliating in the extreme to find after some time that an unsuspected pericardial effusion is present. Probably the most constant symptoms are *dyspnœa*, a *dusky skin*, an *anxious facies*, and a *rapid pulse*, which varies in volume and speed with the respirations. The *voice* is somewhat *husky*, and *active delirium* may be present as in the dry form.

Diagnosis of Pericardial Effusion.—When the pericardium is well filled with fluid the sac presents a peculiar pear-shaped swelling which consists of two spheres superimposed, the smaller one above the larger one. It extends across the middle zone of the chest from a little to the left of the right nipple to a little to the left of the left nipple, and from the central tendon of the diaphragm nearly to the top of the sternum. The pressure of this fluid upon the heart and its great vessels may very markedly interfere with their proper movement, but it does not greatly change its position.

PHYSICAL SIGNS.—The physical signs of pericardial effusion are as follows: On *inspection* the apex beat is absent and in many cases it cannot be found on palpation unless the patient is turned on his face. The chest over the heart may be slightly bulging, and *palpation* may reveal the fact that the first rib can be felt projecting more prominently from beneath the clavicle than in health. So, too, inspection may reveal some prominence of the epigastrium, and there may be unusual tenderness on palpation of this area.

The heart sounds, on *auscultation*, are distant and feeble and the area of cardiac dulness is greatly enlarged. Thus it extends to the right of the sternum to a level below that which forms the base of the cardiac triangle, and to the left of the nipple. There is also enlargement of the area of cardiac dulness at the base toward the left. The presence of dulness in the fifth interspace, to the right of the sternum (Rotch's sign), of dulness as high as the second cartilage or second interspace (Sansom's sign), combined with dulness over the sternum between these points, are pretty sure signs of pericardial effusion, particularly if at the left infrascapular angle there is dulness on percussion (Ewart's sign). The area of dulness is that of a flattened cone, or, as Ewart has well said "it is that of a bag of fluid spreading out at the base." This view has been combated by Dr. Frederick Shattuck, but the distended sac does undoubtedly take this shape, although inability to demonstrate its outline does not exclude effusion by any means.

In doubtful cases, resort to the *x*-ray may aid greatly in deciding the question of the presence of effusion.

The most important states to be differentiated from pericardial effusion are cardiac dilatation and hypertrophy, and aneurysm of the aorta with some leakage into the pericardial sac. The first is the condition most apt to mislead the physician. I have seen this occur several times and I have seen the heart punctured on two occasions in an endeavor to aspirate the pericardial sac in the belief that effusion was present when in reality the condition was one of great cardiac dilatation with pericardial adhesion. The presence of feeble and distant heart sounds, the absence of a definite apex beat, and the manifest cardiac embarrassment all aided in producing an erroneous view in these cases. In such a case the history of old valvular difficulty and the diffuse character of the apex beat should help us to a clear view of the condition. In cardiac hypertrophy the distinct apex beat, the strong action of the heart, and its clear sounds separate the two conditions. An aneurysm of the root of the aorta with some pericardial effusion may be most misleading. In a case of this kind, seen by me, aspiration of the pericardial sac caused rupture of the aorta and instant death.

Very large pericardial effusions have been taken for left-sided pleural effusions, and an encapsulated pleural effusion has been taken for an effusion into the pericardium.

Prognosis of Acute Pericarditis with and without Effusion.—This is good in cases in which the heart is not seriously crippled by the effusion or by associated endocardial changes. The outlook is favorable in proportion to the smallness of the effusion and the benignity of the disease causing it. In pneumonia and renal disease the prognosis is worse than it is in rheumatism, in which disease it is good.

In the great majority of cases pericardial effusion undergoes absorption. This happens in this sac far more frequently than in the pleural sacs, perhaps because of the constant action of the heart. When the effusion persists it must be removed. (See Treatment.) Life is sometimes prolonged for many weeks even after pyopericardium develops. Coutts has recently reported the case of a child of four years that lived seventeen weeks and died only after operation. Nevertheless, pyopericardium is a very fatal condition.

Treatment.—In the early stages of acute pericarditis, if the heart is overacting and irritated, tincture of aconite may be given with advantage to quiet its action and to diminish friction. An ice-bag may be placed over the præcordium in cases of pneumonia with this complication, and in rheumatism several flying blisters may be used. Later if the heart becomes feeble the best stimulants are the aromatic spirit of ammonia, Hoffmann's anodyne, and alcohol. Digitalis except in small doses is rarely of any value, and may be prevented from acting properly by reason of the fever or because there is not room in the pericardial sac for full diastole to take place under its influence.

If the quantity of effusion be very great it must be removed by aspiration or incision. The latter operation is always essential if pus is present, and even if serum is present incision is safer because it is by no means easy to diagnosticate the presence of fluid beyond a doubt and more difficult still to be sure of the part of the sac farthest away from the heart. Aspiration may be therefore in the nature of a plunge in the dark. Incision carefully made is safer. The best spot for operative interference depends upon the individual conditions and the position of the apex beat. The usual areas of election are in the fourth interspace at the left edge of the sternum and at the fifth right interspace at the edge of the sternum.

When incision is practised for pyopericardium the mortality is high, but nevertheless it must be performed if recovery is to occur. Out of 51 cases collected by Porter 20 recovered and 31 died.

The use of purgatives, diuretics, and diaphoretics to cause the removal of the fluid is almost useless. If any drug is of value for this purpose it is iodide of sodium in doses of 20 grains three times a day.

Chronic Pericarditis (Adhesive Form). Definition and Pathology.—By chronic adhesive pericarditis is meant a condition in which one or all of the following pathological conditions arise as a result of an inflammatory process, which involves the pericardium and often the tissues that surround it:

(1) There may develop a state in which partial or localized adhesions take place between the visceral and parietal layers of the pericardium. Several such adhesions may be present in the pericardium at the same time. These adhesions may be immediate or consist in long strings of fibrous tissue stretching across the pericardial sac. They are commonly found near the base, but also occur at the apex.

(2) In the second class of cases the two layers of the pericardium are closely adherent, and the walls of the sac in some cases are much thickened. Here again the adhesion may not be universal, but in patches, although at times the entire sac is obliterated so that the heart is surrounded by a thick and tough capsule composed of the two layers, which cannot be separated.

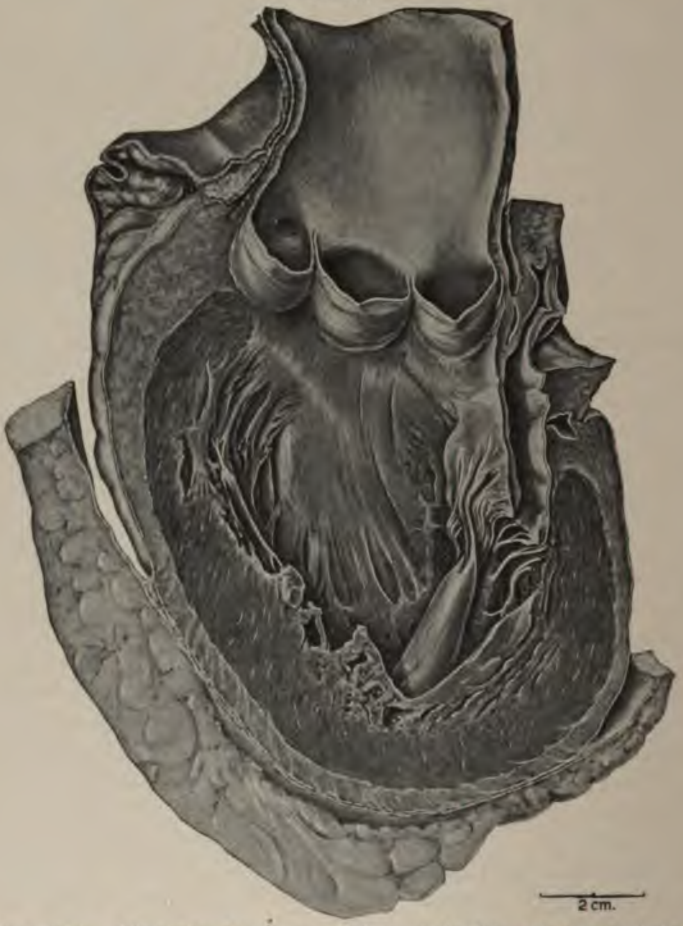
(3) In still a third class the inner surfaces of the pericardium are not so much involved as the outer surface, and as a consequence we find adhesions to the chest wall or to the pleura. It is perhaps hardly fair to class this type with those already named, because the pericarditis in these instances is usually the result of a spread of inflammation from neighboring parts, as from the pleura or mediastinal tissues.

(4) Another type, and the most serious of all, is that in which the internal and external layers are glued together, and the external layer is adherent to neighboring tissues so that the heart, its membranes, and adjacent parts are bound up in an inflammatory mass or mat. If the mediastinal tissues are not affected the condition is called "pericarditis externa et interna," but if the mediastinal tissues are included it is given the name of "indurative mediastinopericarditis." In some cases the tissues for nearly the whole length of the left edge of the sternum may be involved.

(5) Finally, a form of chronic pericarditis occurs which affects the visceral layer of the pericardium almost solely, and encloses the heart in a thickened inner layer.

Related to the latter condition is the so-called "multiple serositis" or the "pericarditic pseudocirrhosis of the liver" of Pick, to which the name "iced liver" has been given by Curschmann. In such cases the

FIG. 66



Heart, left ventricle. Adherent pericardium, with lipomatosis of the adhesions and slightly fatty infiltration of the myocardium.

pericardium suffers from a chronic hyperplastic or fibroid inflammatory process, which likewise affects the serous membranes elsewhere, whence the name "multiple serositis." In other words, in this diseased state all serous membranes of the thoracic and upper abdominal zones are involved in a hyperplastic process which is prone to affect the pericardium in particular. In some instances the pericardial sac contains fluid, but in others it is closed by the adhesions between its walls and even go on to calcification. When the condition is well developed the pericardium and pleuræ are adherent to one another and to all adjacent tissues, and the peritoneum is also thickened and adherent to nearby organs. The liver is adherent to the diaphragm and even to the stomach, colon, omentum, and belly wall. It is the profuse hyperplasia of the peritoneum which causes the organs it covers to look as does a cake which has been "iced."

The two symptoms of this condition which are most constant and characteristic are large ascites and a gradually increasing failure of cardiac power. The cause of the excessive ascities is the perihepatitis and the compression of the abdominal vessels by the newly formed connective tissue. Beyond these two symptoms the manifestations of the process are practically identical with those of adherent pericardium, as will be described below. The malady is a very slow and chronic process lasting, it may be, for years.

The result of these adhesions is enlargement or hypertrophy and dilatation of the heart. This change, however, does not occur in all cases and it is chiefly present in cases in which valvular lesions coexist. The pressure produced by the thickened membranes results in some obstruction to the flow of blood in the great veins at the cardiac base, and this causes jugular distention.

Symptoms of Adhesive Pericarditis.—The symptoms of the milder forms just described are so moderate that no thought of their existence is had till autopsy reveals them. It is in the well-developed types that the condition may cause symptoms which are definite, although they may be very indefinite. Indeed, in some cases it may be impossible to correctly diagnosticate even the most severe forms. The subjective symptoms are *pain in the præcordium* or a sense of constriction. This pain may be dull and constant or paroxysmal, arousing the suspicion of true angina pectoris. *Palpitation* is another common symptom and in some instances the action of the heart is irregular and hobbling. *Shortness of breath* on exertion is also present and at times the right ventricle becomes engorged and secondary engorgement of the liver and lungs ensue. Finally, what is taken for ordinary cardiac dropsy due to valvular disease develops. In other cases pleural effusion develops from this cause.

Diagnosis of Adhesive Pericarditis.—The physical signs to be searched for in making a diagnosis are as follows:

Inspection may reveal depression of the præcordial area and a drawing together of the ribs so that the intercostal spaces are narrowed. In place of this condition in this part of the chest, there may be distinct bulging. Again, the apex beat is often much displaced and as the pericardium is adherent to the chest wall changes in posture do not alter its position. The usual displacement of the apex is upward and outward. In other instances the apex beat cannot be seen or felt in its usual place, but a transmitted impulse can be found in the epigastrium. Perhaps the most important diagnostic symptom is retraction of the chest wall at systole. This retraction may be at the apex or along the left edge of the sternum in the third, fourth, and fifth interspaces. Roberts states that if the right ventricle is greatly enlarged the impulse can be seen to the right of the sternum. Broadbent has called attention to the fact that in many of the well-marked cases of adherent pericardium marked systolic retraction of the lower ribs on the posterolateral aspect of the chest is visible, if the patient is in a good light and the physician regards his back and side from a distance. This retraction is in the nature of a tug at systole. It is emphasized by a deep inspiration. Again, if the physician will place his head directly against the chest in this area he may have transmitted to it a shock at the time of systole. In some cases the adhesions between the pericardium and the diaphragm may prevent the normal epigastric respiratory movements.

Percussion may reveal in some cases an increase in the area of cardiac dulness, but it is not constant. *Auscultation* may show reduplication of the pulmonary second sound, and a rough and widely distributed friction sound, and a somewhat prolonged presystolic murmur at the apex which is not necessarily due to mitral stenosis.

The pulse often presents irregularities, particularly at the time of the inspiratory movements, the so-called *pulsus paradoxus*. Sudden collapse of the cervical veins on diastole may also be present.

It must be distinctly understood that these signs are often absent or are difficult to discover. There is perhaps no more difficult diagnosis than that of some cases of adherent pericardium. The history of a severe or repeated attacks of acute rheumatic fever, is an important point in judging of the likelihood of its presence, particularly if there is also a history of pericarditis. Progressive cardiac weakness without valvular lesions in a young person should raise the suspicion of the presence of this state, particularly if an insidious ascites and dropsy of the lower extremities develop.

Prognosis of Adhesive Pericarditis.—The prognosis is of course very grave if the symptoms are severe and if the occupation is strenuous. With adequate rest life may in some cases be prolonged for years if the inflammatory state is stationary.

Treatment of Adhesive Pericarditis.—The treatment cannot be curative for obvious reasons. It can only be palliative, by rest, good food, and the relief of dropsy by purges and diuretics. Digitalis usually cannot act usefully because the heart is too firmly bound down to permit of increased diastole.

HYDROPERICARDIUM.

This is a state of fluid in the pericardium due to transudation from vessels which are pressed upon by growths, or it results from hæmic or vascular changes due to renal disease. It is not an inflammatory effusion.

Symptoms.—Symptoms and physical signs are absent except when the fluid becomes copious enough to cause cardiac embarrassment, when the condition of the patient is found to be like that produced by an ordinary serous pericardial effusion due to true pericarditis.

Prognosis.—The prognosis depends upon the cardiac state and the underlying cause. It is usually grave, because of the underlying malady.

Treatment.—The treatment consists in free purgation, if the patient is able to stand it and the use of diuretics. Otherwise it is that of general dropsy. (See Chronic Parenchymatous Nephritis.)

HÆMOPERICARDIUM.

Blood in the pericardial sac arises from stab and other wounds, and it is also present in cases of purpura and of profound diseases of the blood. In other cases it arises from aneurysm of the aorta early in its upward course, and finally it may be due to rupture of the heart or aneurysm of one of the coronary arteries. Even in these cases death may be postponed a number of days. If a large amount of blood escapes into the pericardium the heart is stopped by the pressure on its surface and on the great veins at its base, so death is not due to actual loss of blood. This result is what one would expect, and I proved its truth some years ago in an experimental research.

When the condition is due to disease it usually is hopeless. When due to injury the pericardium should be opened, the blood removed, and if a wound in the heart exists it should be closed. There are many cases of this sort on record in which operation has been performed and in which life has been saved.

PNEUMOPERICARDIUM.

Air or gas in the pericardium appears as the result of injury, whereby air enters the sac, or again as the result of a cavity in the lung perforating, through adhesions, into this space. It also may develop in cases of cancer or other ulcerative lesions of the œsophagus, with adhesions between this tube and the pericardium, followed by perforation of the growth. Again, it occurs rarely, as the result of perforation, in pneumopyothorax. Cases are on record in which a gastric ulcer has caused adhesions to the diaphragm followed by perforation, and so pneumopericardium has developed. In still other instances the gas which is present is due to the presence of the *Bacillus aërogenes capsulatus* or other gas-producing organisms.

The mere presence of air, or gas, in this cavity is such an abnormal state that some serous effusion nearly always takes place within a few hours; so that all cases of pneumopericardium are really to be considered as hydropneumopericardium primarily, and as they speedily become purulent by infection they are usually instances of pyopneumopericardium.

Symptoms.—The symptoms resemble those of pericarditis with effusion, except that a considerable part of the area of cardiac dulness may give a high-pitched resonant note on percussion. This area of resonance varies greatly with the posture of the patient, being larger when he is recumbent than when he is erect. Another symptom which is quite characteristic, if it can be discovered, is a peculiar crackling sound due to the action of the heart in stirring up the fluid and the air. Sometimes these sounds are gurgling or churning in character. They have been compared to the splashing of water on a mill-wheel. There is but one condition which can produce symptoms like these, and that is a large cavity in the lung near the heart in which the fluid contents are disturbed by the movements of the heart.

Prognosis.—The prognosis is, of course, very grave, but recovery has occurred.

Treatment.—The treatment depends largely upon the cause. If the case is one of perforated œsophageal cancer it is of course hopeless; in an instance of traumatism with perforation surgical interference may give good results.

PYOPERICARDIUM.

(See Pericarditis with Effusion.)

DISEASES OF THE HEART.

HYPERTROPHY AND DILATATION OF THE HEART.

Definition.—*Hypertrophy* of the heart is a condition in which there is a growth above normal of its muscular fibres resulting in an increase in the size and particularly in the muscular power and weight of the viscus.

In *dilatation* one or more of the cardiac cavities is more capacious than normal; the wall may be normal, increased or decreased in thickness, with a cardiac power less than its muscular development would indicate.

These conditions, which at first glance seem diametrically opposed to one another, are in reality nearly always present simultaneously in varying degree. For this reason I consider them side by side.

When the wall of the ventricle is increased in thickness without any alteration in the size of its cavity the condition is called simple hypertrophy. When the cavity is larger than normal it is called eccentric hypertrophy, or hypertrophy with dilatation, and when the cavity is decreased in size it is known as concentric hypertrophy.

When dilatation is combined with hypertrophy it is called *active dilatation*. When there is no hypertrophy, but thinning of the walls alone, it is called *passive dilatation*.

Hypertrophy of the Heart.—The existence of concentric hypertrophy has been denied. It certainly is very rare. Simple hypertrophy is also rare, but eccentric hypertrophy is one of the most common of secondary pathological changes

A large number of causes produce cardiac hypertrophy with dilatation. Sometimes they act singly, but not rarely several of them are associated. The most common cause is valvular disease, which by its resulting regurgitation or obstruction increases the labor of the heart. The second cause of importance is a state of the bloodvessels which renders the propulsion of the blood more difficult than in health. This obstruction to the free flow of blood may be general, as in cases of arteriocapillary fibrosis; or localized, as when there is roughening or narrowing of the aorta, or when a tumor presses upon the aorta; or, again, in cases of aneurysm. Other localized causes of hypertrophy are emphysema of the lungs or chronic phthisis and adherent pericardium.

The increase in the size of the heart in cases of hypertrophy is sometimes very great. Thus, this organ, which in the healthy man weighs about nine ounces, or 270 grams, and in the healthy woman about eight ounces (240 grams), may weigh as much as 53 ounces (1590 grams). Usually, however, the increase does not go beyond fifteen ounces (450 grams).

A heart which has undergone hypertrophy is broadened or widened at its apex, but the actual increase in the size of this organ at this point depends largely upon the part of the heart which is chiefly affected. Thus, in cases in which the right ventricle is chiefly involved, this part of the heart is often far larger than that part formed by the left ventricle, which seems small by contrast. When a hypertrophied heart is incised, its walls are found to be much thickened and the columnæ carneæ and papillary muscles larger than normal. The enlargement is due to both an increase in the number and size of the muscle fibres.

Symptoms and Physical Signs of Cardiac Hypertrophy.—The chief symptom of ordinary eccentric hypertrophy, when it is adequate to compensate for the valvular lesion, or to overcome resistance, may be said to be the maintenance of a comfortable life and a normal circulation. Many persons develop this state without any knowledge of there being present any valvular disease, and remain in perfect health for years.

If the physician chances to examine such a case he may find a cardiac murmur and then, on closer study, discover the following physical signs, provided the process is well developed. On *inspection* the præcordium is bulging, but the impulse transmitted to the chest wall is regular and deliberate in distinction from the cardiac hurry and irregularity present when compensation is ruptured. The apex beat is more diffuse than is normal and is often in the sixth or seventh interspace instead of in the fifth, and it is farther toward the axilla than in health. On *palpation* the apex beat is found to be forcible and it may be heaving, but if the patient be a full-chested individual these local signs may not be present. If emphysema of the lungs causes these enlarged organs to overlap the heart they may hide much of the hypertrophy. *Percussion* may reveal increase of the area of cardiac dulness to the left, to the right, and downward. *Auscultation*, instead of revealing an exaggeration of the first sound, reveals that it is more distant, perhaps because the thickness of the heart walls muffles the sound, but the aortic sound is accentuated unless the aortic valves are diseased.

If the patient takes violent exercise he may complain of *palpitation* and the thumping of his heart.

When hypertrophy begins to fail the patient complains of *shortness of breath* on exertion, of *palpitation* and *oppression*, and if he persists in keeping on his feet the symptoms of cardiac failure (see Valvular Disease) develop. The first physical signs of the failure will be some *reduplication* of the *first sound* and diminution in its clearness. This reduplication is best heard just inside the apex beat when it first develops, but later it can be heard over a large area. The period between the first and second sound is prolonged as if the ventricle was able, only with the greatest endeavor, to slowly expel its blood. This means diminution of the period in which the heart muscle can obtain nourishment through its coronary vessels.

The causes for this failure are various. In some instances the degenerative changes in the myocardium and in the bloodvessels which are incident to old age are the determining factors. In others some acute illness—as typhoid fever, influenza, pneumonia, or renal disease—may be the cause; or, again, severe exercise may produce so much exhaustion of the heart muscle and acute dilatation that the cardiac power is impaired for all time.

Diagnosis.—Cardiac hypertrophy must be separated from several important conditions which are by no means rare. From dilatation hypertrophy is differentiated by the facts that the impulse in the former is feeble, in the latter it is strong, and by the feeble heart sounds in the former as compared to the stronger ones in hypertrophy. So, too, palpation of the apex in dilatation reveals a diffuse and feeble impulse and in hypertrophy a forcible beat.

From pericardial effusion it is differentiated by the fact that, though the area of cardiac dulness is increased in both states, the cardiac impulse and cardiac sounds are muffled in effusion and exaggerated in hypertrophy. From displacement of the heart it can be differentiated by the fact that though the apex beat is displaced the general area of cardiac dulness is not increased in cases of displacement.

Again, in certain cases in which the chest wall is thin and the lung is retracted so that it fails to cover the heart as in health, the heart may be so close to the chest wall that its area of dulness will be abnormally large and its apex beat unduly forcible and diffuse. In such a case the careful study of the state of the lung and pleura will make the condition clear.

Of the functional disorders that produce overaction of the heart and so cause apparent but not real hypertrophy, "tobacco heart," the irritable heart of exophthalmic goitre, and that of neurotic individuals must be remembered.

Prognosis.—As cardiac hypertrophy in its common form is compensatory in character, and as it very rarely becomes excessive, in the sense that it is beyond the needs of the patient, the prognosis in a case in which it is present deals not with the question of how much greater will hypertrophy become, but rather how much longer will hypertrophy enable the heart to supply the bloodvessels with blood in satisfactory quantities. Unlike most alterations from the normal as the result of disease this change is distinctly advantageous to the patient.

The question as to how long the hypertrophy will be maintained can only be answered after its provoking cause or causes have been determined. If the hypertrophy following valvular disease of the heart is adequate, if the patient is a young and otherwise healthy adult, and if the valvular lesion is not progressive, the prognosis as to the maintenance of the condition is usually good, but will depend upon the good habits of the patient, particularly as to alcohol, hard work and exposure, and upon the particular valve which is diseased. (See Prognosis of Valvular Lesions.) If the hypertrophy is the result of arteriocapillary fibrosis with its associated renal changes, the duration of life except under the most favorable conditions is brief, because the arterial obstruction is constantly increasing and the heart is constantly exposed to increasing strain, increasing toxæmia, and is poorly nourished by its own coronary arteries.

Treatment.—There is no treatment for compensatory hypertrophy except to maintain it by care as to manner of life, and the use of digitalis and rest if its integrity or maintenance is threatened. I have seen a few cases of aortic regurgitation with great hypertrophy in which rest in bed and moderate doses of tincture of aconite have given better results than rest and digitalis, but in these cases the patient had been accustomed to severe toil, and when he was put at rest seemed to have excessive cardiac power, as shown by throbbing and oppression. On the other hand, when hypertrophy seems excessive, it is not rarely in reality lacking, and the violence of the heart movements may be abortive efforts at circulation. Often the use of nitroglycerin at such times will be advantageous if the arterial pressure is high.

Dilatation of the Heart.—Passive dilatation (without hypertrophy) may be caused by valvular lesions, as the result of which the cavities of the heart become distended, but hypertrophy does not develop. Of these the chief cause is sudden and prolonged strain, and the feebleness often due to myocardial disease. Obstruction to the flow of blood in the pulmonary vessels may cause dilatation of the right side of the heart, as in cases of pneumonia, in cases of pleurisy with effusion, and in cases of acute pulmonary œdema complicating uræmia.

A common cause in men over fifty years is sudden effort, as in lifting a heavy weight or climbing rapidly a steep flight of steps. In the young and vigorous sudden strain may be followed by rapid return of the dilated heart to its normal size, but in those further on in years, or who have valvular or myocardial disease, an acute strain often results in permanent dilatation. It is very common for old men to try to prove that they are "as young as they used to be," and to attempt athletic feats which are followed by acute dilatation and perhaps immediate death or death in a few days or weeks. In other instances the heart suffers from a gradual dilatation from prolonged strain, as in soldiers on the march. Not rarely dilatation develops during the course of one of the acute infectious diseases or during convalescence.

When the strain is very gradual, instead of meeting the increased demand by increased effort, the heart slowly dilates and is perhaps never able to empty its cavities of blood.

The intrinsic causes of dilatation are myocarditis, fatty degeneration, fatty infiltration, and serous infiltration from pericarditis. In some cases, however, no adequate intrinsic or extrinsic cause can be found.

Symptoms of Cardiac Dilatation.—The symptoms and physical signs of passive dilatation are usually such that a diagnosis is readily made. When it is sudden in onset an acute or partial *syncope* with labored respiration and *thoracic oppression* may be present.

When the onset is more gradual the main symptoms are *impaired circulation*, a tendency to *syncope* on suddenly standing or sitting up, *congestion of the kidneys* causing *albuminuria*, and the poor capillary circulation which causes the skin of the hands to remain pallid long after pressure. Many persons so afflicted cannot lie down without *urgent dyspnœa* and *cardiac distress*. The *pulse* is small and irregular, and the *arterial tension low*. In other cases the pulse wave may be voluminous, but feeble.

An inspection of the præcordium shows that the apex beat, if visible at all, is diffuse and displaced outward and downward. On placing the fingertip on the spot where the apex beat seems most marked, the examiner is surprised to find no impulse or one which is very slight. There is often visible, but rarely palpable, pulsation near the ensiform cartilage or in the epigastrium. Care must be taken that overlying lung does not lead to an erroneous belief as to the presence of cardiac feebleness. If the whole hand is placed over the disturbed surface of the præcordium it is remarkable how little impulse is discernible. Percussion shows an increase in the area of cardiac dulness to the left, to the right, and downward. Often it is also increased upward.

On auscultation the first sound of the heart may be short and small, valvular and flapping, though fairly loud, and if the heart is strong enough there may be a systolic murmur at the apex, due to mitral regurgitation arising from stretching of the mitral orifice. As in ruptured compensatory hypertrophy the sounds of the heart may be equalized and the space between them may be altered so that the sounds are like those of the fetal heart or like the ticking of a watch. In other cases the first and second sounds may occur close together and the diastolic pause be prolonged. Very often great arhythmia is present. If in addition to these signs there is a history of acute strain near or remote, or symptoms of cardiac feebleness, and particularly if the onset has been sudden, without the presence of an acute infection to cause valvular disease, the diagnosis of dilatation may be made.

Prognosis.—The prognosis depends upon the degree of the dilatation, the state of the vessels and of the heart muscle and of the kidneys, and last, but not least, the lungs. When the latter are filled with rales the state is alarming, and if the vessels and kidneys are diseased the outlook is hopeless for much betterment. If the state of the vessels and the general condition of the patient indicate fatty myocardial degeneration the prognosis is also bad. If these states are absent, and rest can be maintained, distinct improvement can be hoped for, but a complete cure in the sense of old-time vigor is rarely reached.

Treatment.—The treatment is, first of all, rest in bed, or in an easy chair if bed is impossible because of orthopnœa. The second object to be gained is the removal of the cause of the dilatation, if that be possible, as the reduction of high arterial tension by the use of nitroglycerin. Third, the employment of digitalis and strychnine for effect, recalling the fact that once this drug has produced its action smaller doses will maintain its influence, and also bearing in mind the additional fact that when it is in full effect sudden changes of posture are dangerous, and that it may cause so much ventricular stimulation as to overdistend the auricles, which are poorly protected by the relaxed mitral ring.

When the lungs and kidneys are engorged, the application of several dry cups over them is useful, and if jugular distention and hepatic congestion is marked, the patient may be freely bled if he is plethoric. So, too, hydragogue cathartics, such as jalap and compound extract of colocynth, may be used to unload the bowels and liver, but care must be taken that the patient is not exhausted by purging. Blue mass in the dose of 8 grains once a week is useful, and the pill of calomel, squill, and digitalis mentioned under Endocarditis may be used. If ascites is a pressing symptom tapping is indicated, while for general anasarca the formula given under Endocarditis, or apocynum cannabinum may be used, or the digitalis given more liberally.

The diet should be light and nutritious, and often it is well to give pancreatized foods or starches with taka-diastase. Great care must be taken that the stomach is not distended by food or drink, and if gas accumulates in the stomach it should be expelled by the use of Hoffmann's anodyne in drachm doses, and by the employment of a turpentine stupe. When dyspnœa is urgent morphine and strychnine are useful drugs.

High altitudes should be carefully avoided and only gentle exercise on level ground be allowed.

DISEASE OF THE MYOCARDIUM.

Disease of the myocardium may be divided into two classes, viz., degenerative and inflammatory.

Degenerative Changes. **Etiology and Pathology.**—The degenerative conditions are as follows: In the *granular form*, sometimes called "parenchymatous degeneration," there develops in the protoplasm of the cardiac muscle fibres albuminous granules which differ in size and in number, and may be present in such an excess as to obscure the nuclei and striæ. The affected muscle is cloudy, softened, and paler than in health, its strength decreased, and the circulation is proportionately depressed. In a later stage some degree of fatty degeneration may also be present.

This type of degeneration is observed in the course of acute infectious diseases, as diphtheria, typhoid and typhus fever, the pyæmias, and even as a result of severe burns, and in debilitating conditions associated with severe cardiac work or the presence of toxic bodies in the blood.

In *fatty degeneration* of the heart the affected fibres contain fat-globules, which, in marked cases, replace the structural elements, both the nuclei and protoplasm. In some instances this degenerative process is restricted to a single focus, or it may be scattered about or diffuse; in others it is universal. When the heart is examined at autopsy it is seen to be mottled and the papillary muscles in particular will reveal the fatty areas, the so-called "Tiger Herz" of the Germans.

Diffuse fatty degeneration is caused by prolonged nutritional disorders.

Pernicious anæmia and leukæmia may also cause it, as may poisoning by arsenic, phosphorus, and antimony. Less commonly it is a sequence of various acute infectious diseases like diphtheria and scarlet fever or typhoid fever, and by degenerative or atheromatous changes in the coronary arteries. The local or circumscribed *forms* follow embolism or other *types* of rapidly developed coronary occlusions.

It is important that fatty degeneration be clearly separated from *fatty infiltration*, in which state the muscle fibres are not altered, but have been separated by the projection of fatty masses between them. This may cause some wasting or atrophy of the muscle fibres. This state is most commonly met with in very fat persons and in those who are addicted to excessive beer-drinking. Occasionally forms of *amyloid* and *hyaline degeneration* of the heart fibres occur.

Brown induration or atrophy of the heart is often seen in cases of chronic valvular disease and in old persons. The muscle is more dense than normal and reddish-brown in hue, and about its nuclei brown pigment is deposited. *Calcareous degeneration*, in which the muscle fibres become infiltrated with lime salts, is rare.

Under the name of *fragmentation* and *segmentation* there is seen a state of the heart muscle in which its fibres are broken across in fragments, or its cells are separated at the point of junction (segmented). These changes may occur in acute infectious diseases or in cases of central nervous disease. In some cases they are probably agonal, and it may be that similar appearances are of postmortem origin, but the frequency with which granular change is seen at autopsy strongly indicates that it may be present and unrecognized in life, not only in fatal cases, but in those who recover.

Symptoms of Myocardial Degeneration.—The symptoms of degeneration of the heart of the albuminous type cannot be considered as pathognomonic. Indeed, there may be no evidence of cardiac failure until a sudden and perhaps *fatal attack of syncope*, after a slight exertion, reveals the alarming state of the heart muscle. In other instances the *feeble cardiac sounds* on auscultation indicate the real condition of the heart.

When fatty degeneration is present the same absence of symptoms may exist until the final fatal syncope, or the patient may suffer from repeated attacks of *syncope*, or of *vertigo* with *anginoid seizures*. (See Stokes-Adams Disease, page 475.) The frequency and severity of these attacks are, however, by no means in direct proportion to the extent of the lesion in the heart muscle. In one instance a fatal syncope occurs, yet the heart scarcely seems altered in its fibres. In another case the life of the patient persists and fairly good health is maintained for years, yet at autopsy the heart muscle is so fatty and soft that the fingers can be pushed through it as if it were wet paper.

In some instances the symptoms complained of by the patient seem to be epigastric and due to *disordered digestion*. How often do we hear of a man of advanced years dying of *acute indigestion*, which is really cardiac failure with gastric symptoms, or cardiac failure caused by an overdistended stomach.

The *heart sounds* when the patient is in his average state of health are *distant* and *feeble*, and his *slow pulse* is *small* and of *low tension*. Not rarely his radial and temporal arteries are very calcareous, but in other cases they are soft and devoid of resistance on pressure.

There still remains to be considered several notable facts in connection with this disease. Notwithstanding the great feebleness of the heart in some cases and the exceedingly weak circulation of blood, dropsy in any form is a very rare condition. Indeed, if dropsy occurs it is certainly due to some complicating state. A second fact is that in some cases in place of anginoid attacks an *epileptiform* or *apoplectiform* seizure occurs. The epileptiform seizure is not that of *grand mal*, but *petit mal*, with this difference, that while, as in *petit mal*, there are no convulsions, there is a period of profound unconsciousness which is rather a syncope than a coma, such as is seen in true epilepsy.

The apoplectiform seizures may very closely resemble true cerebral hemorrhage, even to the stertorous breathing, the hemiplegia, the unconsciousness, and Cheyne-Stokes respirations. That the case is not one of apoplexy is usually proved by finding that the high-tension pulse of cerebral hemorrhage is absent and replaced by the low tension and slow pulse of fatty degeneration.

Prognosis of Myocardial Degeneration.—The prognosis in all cases of cardiac degenerative change is, of course, very grave. When it is present in children after acute infectious diseases recovery may ensue under a course of arsenic, phosphorus, and nux vomica, with absolute rest, and fresh air and sunshine, but even in this class of cases sudden death often intervenes. In the fatty heart of advanced age, whether the years be great or the patient prematurely old, the outlook is bad; but as no one can tell the extent of the lesions in the heart, a statement as to a brief duration of life is very prone to bring the physician's opinion into discredit if he attempts to name the time of dissolution. The wise physician rarely expresses a positive opinion as to the probable time of death in any case, much less in fatty heart.

Stokes-Adams Disease.—Cases of extreme slow pulse with vertigo, or syncope, or apoplectiform or epileptiform seizure, have been given the name of the "Stokes-Adams syndrome."

Associated with the slowness of the pulse there is marked pulsation in the veins of the neck, and to use Stokes' own words, written in the *Dublin Quarterly Journal of Medical Science* in 1846, the number of reflex pulsations is difficult to be established, but they are more than double the number of the manifest ventricular contractions. Experimental and clinical studies by Erlanger (*Journal of Experimental Medicine*, vol. vii., No. 6, November 25, 1905, pp. 676-724, and vol. viii., No. 1, January 25, 1906, pp. 8-58), with clinical and autopsy findings by Stengel and others, indicate that the symptom-complex in most if not all cases of this condition is due to what is now denominated "heart-block." In this state the auricles beat two, three, or four times as rapidly as do the ventricles. Erlanger has repeatedly produced at will any particular rhythm or even entire independence of the auricular and ventricular contractions in the dog by varying mechanical compression of the auriculo-ventricular muscle bundle of His. This muscle bundle, which is 17 to 20 mm. long, 2.5 mm. wide, and 1.5 mm. thick, extends from a point in the auricular septum below the foramen ovale downward through the auriculo-ventricular junction and terminates in the ventricular septum a short distance below the beginning of the aorta. The impulse which causes ventricular contraction appears to be transmitted from the auricles to the ventricles through this bundle and consequently lesions affecting it lead to slowing of the ventricle. When the latter chambers cease beating for some seconds syncope ensues. Most cases of Stokes-Adams disease appear to be primarily arteriosclerotic or syphilitic in nature though myocardial lesions of other origin may be the cause. Autopsy in some reported cases has shown no evident lesion and these have been pronounced neurotic in character. Erlanger states, however, that as yet there has been described no typical case of Stokes-Adams disease which might not have been caused by heart-block. A point to be noted in autopsies upon cases of this disease is whether lesions of the mesial leaflet of the tricuspid valve can interfere with the integrity of the muscle bundle of His.

Myocarditis. **Definition.**—This term is an unfortunate one in that it is often loosely applied to the degenerative changes just described as well as to those about to be mentioned. It is also unfortunate because it seems to indicate that there is a primary inflammatory state of the cardiac muscle fibres, whereas the changes in the fibres are secondary to inflammatory affections of the interstitial tissues of the heart and of its bloodvessels, which thereby cause atrophic and degenerative changes in the muscle.

There are several forms of so-called myocarditis, of which the most common is a slow, low-grade inflammatory change called *chronic interstitial myocarditis*, manifested by a wasting of the muscle fibres and the intercalation of fibrous or fibroelastic tissues. There is also an acute process, *acute interstitial myocarditis*, of which there are suppurative and non-suppurative varieties, the former being a manifestation of pyogenic infection of the heart wall.

In the great majority of cases the chronic form is the result of pathological changes in the coronary arteries. These vessels suffer from an obliterative arteritis in their finer branches, undergo atheromatous change, or become plugged by an embolus or thrombus. The lesions which result from these changes differ widely in character, but all greatly impair the usefulness of the heart. In all conditions lessening the vascular lumen and so decreasing the nutrition of the heart, there develops an overgrowth of interstitial tissue with atrophy of the muscle fibres. It is probable that the process is at no time a true inflammation, but rather one in which diminished blood supply causes atrophy of the muscle, followed by a substitutive fibrosis.

When a branch of a coronary artery is plugged the affected area may manifest the changes seen in an infarct, or when enough nourishment is available to prevent actual necrosis the deficient nutrition gives rise to fatty degeneration. In either case the affected area may become softened, and give way, causing rupture, or fibrous tissue gradually takes the place of the degenerated fibres. Later the scar tissue yields to pressure and a cardiac aneurysm ensues.

Hypertrophied hearts may show a slight increase in the fibrous tissue, and in failing compensation and progressing dilatation this increase in interstitial tissue may be conspicuous.

In some instances chronic myocarditis is not the result of vascular change, but of inflammatory processes in the pericardium and endocardium; and in syphilis there is often seen a marked increase in the interstitial tissues of the heart, which is not surprising in view of the serious changes produced by this disease in the small bloodvessels everywhere. Chronic myocarditis is more common in males than in females.

Symptoms of Myocarditis.—The immediate effects upon the patient produced by the lesions just named vary to an extraordinary degree. Plugging of one of the large branches of a coronary artery usually results in *sudden death.*

In some instances, however, the patient survives a severe attack of cardiac disturbance, but under these circumstances the plugging is usually in a small vessel, and a gradual substitution circulation is established, not by anastomosis, for these vessels are end arteries, but by the so-called vessels of Thebesius, which in some cases are able to supply the heart with an adequate quantity of blood.

When the closure of the vessel is gradual it not rarely happens that necrosis of the area deprived of blood is prevented by a blood supply through the vessels of Thebesius, so that the death of the patient is postponed until a very extraordinary degree of atheroma and narrowing in both coronary arteries is developed. The coronary arteries of a well-known member of the medical profession in Philadelphia, who died a few years since, were so diseased that only a thread-like passageway existed in these vessels, yet he led an active life to the end. Such patients may have no marked cardiac symptoms, but, as a rule, repeated attacks of *angina pectoris* of increasing intensity give warning of the sudden death to come. The other symptoms are the same as those described under fatty degeneration of the heart.

The physical signs of myocardial degeneration are *feebleness of the apex beat, equalization* of the *first* and *second sounds of the heart,* and evidences of feeble circulation in the lungs and in the peripheral systemic vessels.

The *prognosis* depends entirely upon the situation and the degree of the cardiovascular change. So far as recovery is concerned, that is impossible. The probable duration of life is also difficult to determine. Many cases with all the symptoms of severe myocarditis live a long period, while others die with unexpected suddenness.

Treatment.—We cannot expect very much from treatment in patients suffering from chronic myocarditis. It must be evident from what has been said, under the discussion of the pathological conditions which cause these states, that the harm is done before the physician has an opportunity to place the patient under treatment. The only hope is that by regulating the manner of life, by increasing the action of the kidneys, if they are sluggish, by attending to the digestive apparatus, and *by preventing undue cardiac strain* through excessive muscular or mental exercise, we may be able materially to prolong the patient's life. In instances in which the bloodvessels are distinctly atheromatous or fibroid, the use of the iodide of strontium or sodium, in doses varying from 10 to 40 grains three times a day, is usually advantageous. This treatment may be continued for several weeks, and then the patient may receive a course of Fowler's solution as a general tonic, with perhaps a small quantity of nux vomica or strychnine added to it.

If arterial tension is high, he should be given nitroglycerin in doses varying from $\frac{1}{100}$ to $\frac{1}{50}$ of a grain three or four times a day, in order that the resistance which is offered by tense vessels to the action of the heart may be lessened. Under these circumstances, too, small doses of digitalis sometimes act advantageously, particularly if nitroglycerin is given at the same time. To give digitalis to a failing heart and yet to permit the arterial tension to remain high is of little ultimate advantage to the patient, since it increases the labor of the heart without material advantage. It is much more important to diminish the labor by the use of nitroglycerin than to stimulate this viscus to increased endeavor by large doses of foxglove. Five minim doses of tincture of cactus grandiflora often do much good under these circumstances. It must be remembered that if the heart has undergone distinct degenerative changes there is little muscular fibre upon which the digitalis may exert its stimulating influence, and there may be danger by increasing intracardiac pressure of causing rupture of some area of white necrosis, thereby causing cardiac aneurysm.

It is hardly necessary to add that these patients should be warned against excessive muscular exercise or any severe cardiac strain, and they should be advised to lie down and rest several times a day, in order that the heart may at each period of rest recover as much strength as possible.

Digestive disturbances, which by accumulation of gas may disturb the action of the heart, must be prevented by the institution of an easily digested and simple diet, small quantities of food being taken often so as to avoid overloading the stomach. If there is a tendency to an accumulation of gas in the bowel salol may be given as an intestinal antiseptic, or in its place the capsule of taka-diastase, pancreatin, nux vomica, and capsicum, which is recommended in the article on Angina Pectoris, may be administered. Some of these patients seem to be greatly benefited by the use of gentle massage every day or every other day, with the object of aiding in the circulation of the juices of the body. Great care should be taken that the massage is not so vigorous that the patient is fatigued by it. Strychnine in the dose of $\frac{1}{20}$ of a grain three or four times a day is often exceedingly beneficial to these patients, particularly if there is any tendency to shortness of breath on lying down.

In many instances when the heart is feeble as the result of fibroid changes in its muscle, or when the patient is convalescing from some disease like influenza, which seriously impairs the functional activity of this organ, excellent results are sometimes obtained by the institution of what is known as the Nauheim baths, which were originally brought before the profession by Schott, of Nauheim in Germany. These baths are composed of water which is charged by nature with large quantities of carbonic acid gas. The water is also naturally warm. The patient is immersed in a bathtub, and immediately there is attached to the surface of his skin myriads of tiny bubbles of carbonic acid gas, which as they break produce a slight tingling sensation and exercise a stimulant influence upon the peripheral capillaries, as the result of which these capillaries are dilated and dermal hyperæmia is induced. In this manner the circulation is equalized, internal congestions are overcome, and the heart finds it easier to pump blood through the dilated superficial capillaries than under ordinary conditions. Not infrequently when the patient first enters the bath a primary contraction of the peripheral capillaries ensues, and this results in a momentary increase in the work of the heart, so that the patient for a time feels somewhat oppressed. Usually he remains in the tub for ten or fifteen minutes, but this period is governed by the physician who superintends the use of the baths. On his removal from the bath the patient is carefully dried by an attendant and has absolute rest for one or two hours. After the baths have been used for some time additional salt is added to the bath, and water containing larger quantities of gas is employed. In addition to these baths the patients are subjected to gentle resistance movements and massage so as to improve the circulation of blood and lymph in the muscles. Great care must be taken that these movements are not sufficient to tire the heart. When valvular disease is very marked, these baths are contraindicated.

The Nauheim baths are also contraindicated in cases of advanced arteriosclerosis, and in chronic Bright's disease if it is well developed, although if the renal difficulty is largely due to congestion this plan of treatment is advantageous. Aneurysm also contraindicates them, and bronchial asthma and chronic bronchitis contraindicate them, or at least require great caution in their use. Cases of pulmonary tuberculosis with cardiac disease also should not be subjected to this method, nor should patients who are suffering from far-advanced degeneration of the heart muscle receive it. These baths should never be taken except under the care of a local physician.

Attempts have been made to introduce artificial Nauheim baths in this country, but it has been found impossible to satisfactorily charge the water with carbonic acid, and the difficulties connected with the preparation of the bath have been such that its employment, except at Nauheim, has largely passed out of use.

It is important to note that the resistance exercises, which are carried out in connection with this plan of treatment, are probably equally beneficial, if not more beneficial, than the baths themselves. They consist in having the patient extend and flex his joints against the resistance offered by the attendant.

CARDIAC ANEURYSM.

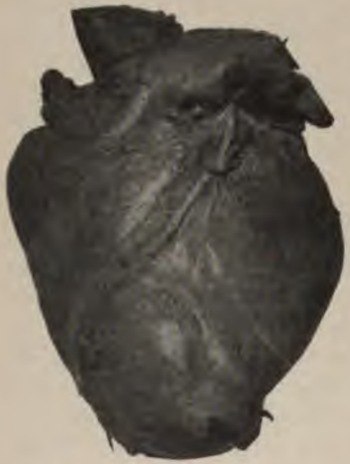
Aneurysm of the heart may occur in one of three forms, viz., aneurysm of the heart walls, aneurysm of the valves, and aneurysm of the coronary arteries. Aneurysm of the cardiac walls consists in a localized dilatation or pouching of the wall, and is to be separated from dilatation of the heart, to which the term aneurysm is sometimes applied by French writers. The aneurysm usually affects the ventricular wall. Hall has recently collected 112 cases, in which the site of the aneurysm was as follows: left ventricle, 92 cases; right ventricle, 1 case; left auricle, 2 cases; ventricular septum—(*a*) muscular part, 8 cases; (*b*) membranous part, 7 cases; auricular septum, 2 cases.

The left ventricle is therefore affected more commonly than all of the other chambers combined. The aneurysm is usually near the apex of the ventricle or in the anterior wall, just above the apex; 67 of Hall's cases were so situated.

The condition, as one would naturally expect, is found more frequently in males. In the relative frequency in the two sexes, Thurnam's, Legg's, and Hall's and my own statistics show a remarkable resemblance. Of Thurnam's 40 cases, 30 were males, 10 females; of Legg's 88 cases, 64 were males, 24 females; and of 80 cases collected by me, 59 males, 21 females. In a total of 208 cases, 74 per cent. were males, and 26 per cent. females.

Aneurysm of the heart is a sequel of the secondary myocardial changes already described. Thus the fibrous tissue which replaces the tissues which have undergone necrotic change may gradually yield before the blood pressure in the ventricle and form a sac, which is a true aneurysmal dilatation. This sac may communicate with the ventricle by a small opening. In other cases the ventricular wall at this point yields, so that the opening may be the full width of the sac. As in aneurysm of the bloodvessels, the wall of the sac is composed of several layers made up of the visceral layer of the pericardium, and perhaps of the parietal layer as well, if it has become adherent. Under this is the fibrous tissue, and beneath this again forming the inner layer is the endocardium. Rarely several sacs are present.

FIG. 67



Front view of heart, showing aneurysm of left apex of ventricle, which has perforated into the pericardium. The swelling of the aneurysm is visible externally, and the heart wall at the apex is no thicker than brown paper. No pericardial adhesion. The interventricular branch of the left coronary artery is dissected out, and is very atheromatous, and at the upper end of the groove is completely blocked by a thrombus, which extends downward for two and one-half inches. (From a specimen in Dr. Littlejohn's Museum.)

Three conditions may develop in such sacs. They may give way under pressure, causing sudden death, they may become filled with a clot, or their walls may be calcified. Sometimes an aneurysm of this sort forms in the septum and ruptures into the right ventricle.

In some cases the aneurysm may be due to fatty degeneration, without primary vascular disease. A softened spot in the heart muscle may bulge under strain, and rupture may occur before any real sac is formed. This is commonly called rupture of the heart, and usually involves the anterior wall of the left ventricle near the septum; but it may affect any part of the walls of the cardiac cavities.

Death by rupture of the sac does not occur as frequently as would be imagined, and in this respect cardiac aneurysm resembles aortic aneurysm. Out of 60 cases collected by Legg, only 6 died by rupture.

Aneurysms of the cardiac septa are so rare as to be curiosities. Hall states that only 2 cases have been reported in twenty years.

An *aneurysm of a valve* is sometimes formed as a result of endocarditis. This condition usually affects the aortic and mitral leaflets with about equal frequency. One leaflet is contracted or destroyed and another leaflet then yields or sags, partly because of deficient support, forming a pocket or sac which projects into the left ventricle. Sometimes this sac ruptures, and so the valve becomes perforated. In other instances the entire valve becomes sacculated.

Although atheroma of the coronary arteries is a very frequent lesion, aneurysm of these vessels is exceedingly rare. Hall could find only 25 recorded cases, of which 17 were in males.

Symptoms.—The symptoms of cardiac aneurysm are not definite at any time, and, unless the sac is large, there may be none. Hall tells us that out of 76 cases an antemortem diagnosis was made only once. When the sac is large it may cause a marked increase in the area of cardiac dulness near the apex, and produce distinct pressure symptoms. A skiagraph may give valuable information of the lesion.

WOUNDS OF THE HEART.

Wounds of the heart are by no means uncommon, as the result of shooting or stabbing. Cases of recovery after both of these forms of trauma are recorded in considerable number. The stab wounds probably recover in greater number than those which suffer from bullet wounds. Death is usually due not to the direct injury of the heart, but to the fact that the pericardial sac soon becomes filled with blood, and the heart is unable to expand. In other words, even severe injury to the heart is not fatal unless the hemorrhage be free, or the organ is damaged in some vital spot, as in Kronecker's "co-ordinating centre." I proved these facts in a research carried out on dogs many years ago, and a number of surgeons have now reported cases in which a stab wound of the heart has been exposed and sutured, and recovery has ensued. Further than this, I have seen the heart punctured and blood aspirated from its cavities without injury to the patient.

Gibbon and Stewart, of the Jefferson College Hospital staff, have recently reported interesting cases in which cardiac wounds were stitched with success.

ENDOCARDITIS.

Definition.—Endocarditis in an inflammation of the lining membrane of the heart, the endocardium. In the great majority of instances it chiefly affects the endocardium where it covers the valves (valvular endocarditis), and rarely it involves that part which covers the walls of the cavities (mural endocarditis). A distinction should also be drawn between the acute and chronic form of the disease and between the acute simple, or benign, form, and the so-called acute malignant, or ulcerative type. It is proper to state, however, that many persons deny the correctness of this division, and regard the two conditions as different stages or degrees of the same process. Finally, it is to be recalled that there are two types of chronic endocarditis, namely, that which is the result of the acute variety and that which arises in association with chronic arteriocapillary fibrosis and atheroma, which is a slow, retractile form of the disease.

Acute Endocarditis.—Synonyms: Verrucous Endocarditis, Simple Endocarditis, Benign Endocarditis, Papillary Endocarditis.

Etiology.—The bacterial origin of endocarditis is generally admitted, but all efforts to identify any particular organism as the specific cause have proved fruitless. Many organisms have been identified in the vegetations. Of these should be mentioned those found in acute rheumatism, pneumococci, streptococci, and staphylococci, and, less frequently, the colon bacillus, typhoid bacillus, influenza bacillus, tubercle bacillus, and a number of other bacteria.

In the article on acute Articular Rheumatism attention has already been called to the frequency with which this condition complicates that malady. So constant is this lesion during the course of acute articular rheumatism that it may be regarded as the condition next in constancy to the inflammation about the joints. It is probable that in all cases of acute rheumatism a slight endocarditis is present, but it may be so slight that no physical signs of its existence can be elicited.

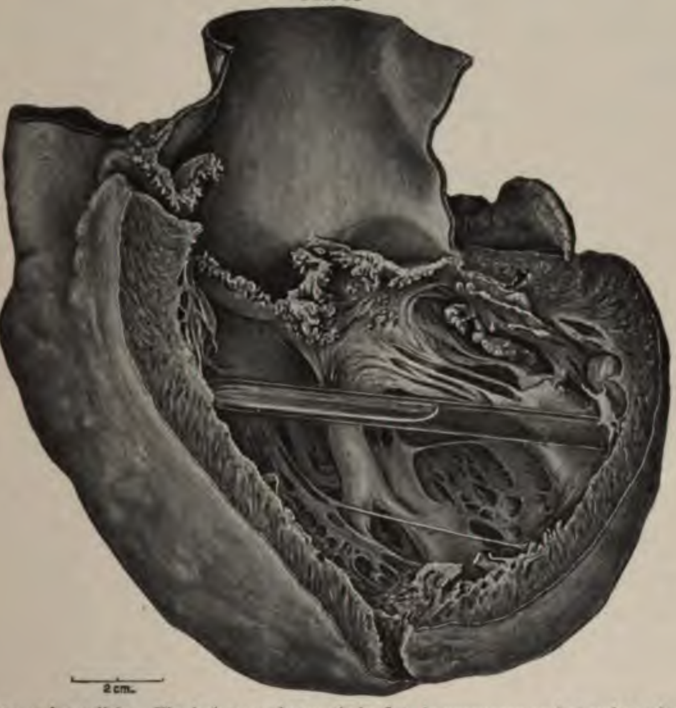
In children suffering from acute rheumatism the involvement of the endocardium is far more frequent than it is in adults. Thus, it is generally considered that from 60 to 80 per cent. of children who have acute rheumatism develop endocarditis; whereas, the percentage usually accepted for adults is about 21 per cent. Eighty per cent. is probably too high, and 21 per cent. is certainly too low an estimate. It is especially important to bear in mind that mild articular symptoms are not rarely accompanied by severe cardiac lesions, although, as a rule, the severity of the articular symptoms and the severity of the heart lesions go hand in hand. The first attack of rheumatic fever is more frequently the cause of cardiac lesions than subsequent attacks, and the signs of endocardial inflammation usually develop during the first ten days of the illness, although in rare cases a murmur may be heard before any arthritic signs develop.

When acute rheumatism causes endocarditis, it commonly affects the mitral valve. The aortic leaflets are comparatively rarely affected, and the valves on the right side of the heart only in very rare instances.

Next to acute rheumatism as a cause of acute endocarditis must be noted its association with chorea, in which disease, in its well-developed and typical forms, lesions of the lining membrane of the heart are very often present. The occurrence of endocardial disease in chorea is in direct proportion to the severity of the disease, but it is difficult to decide how frequently true endocarditis is actually present, because many choreic patients present on auscultation functional murmurs in the heart which disappear so rapidly that it is inconceivable that they could have been organic in origin. Again, so few cases of chorea come to autopsy during or immediately after the attack that it is impossible to study the exact state of the endocardium. If the various statistics of frequency of heart murmurs are added together, we find that these sounds occur in about 31 per cent. of cases of chorea.

In a very considerable proportion of cases endocardial infection takes place during acute tonsillitis. Another cause of endocarditis is gonorrhœa, which causes the ulcerative type of the disease more commonly than the benign form. Scarlet fever may also be a causative factor, but whether this is due to direct infection of the endocardium by the micro-organism which causes scarlet fever or by its toxins, or results from the mixed infection so frequent in this disease, is not known. Rarely endocarditis is apparently due to tuberculosis, for this bacillus has been found in the valve lesions. Traumatic forms and endocarditis due to syphilis have been described. Occasionally acute endocarditis develops as a result of an exacerbation of the chronic form of the disease.

FIG. 68



Heart, acute endocarditis. The lesion on the aortic leaflets is verrucose and at points ulcerating. On the lateral ventricular aspect of the mitral valves are a number of vegetations, although the contact line and auricular surfaces of this valve were not involved. The vegetations on the ventricular surface probably resulted from inoculation by projecting vegetations situated on the aortic leaflet. It is to be remembered that endocarditis affects the mitral valves more commonly than the aortic valves.

Endocarditis has been reported as present in the fœtus as a result of the infection of the blood through the placenta. In this period of existence the pulmonary valves are the parts affected.

Pathology and Morbid Anatomy.—The degree of inflammation in the endocardium covering the cardiac valves varies greatly in different cases. Its frequency on the left side, particularly in the mitral valves, is due principally to the greater stress to which these leaflets are subjected and also to the higher oxygen content of the arterial blood. The lesions develop in the auricular aspect of the mitral leaflets and the ventricular side of the aortic valves; this distribution depends upon the friction and impact of the blood, and the same factors determine the distribution of the vegetations along the lines of greatest pressure, where the leaflets impinge one upon another when closed.

The earliest lesions are rarely seen, because death seldom occurs at this time. The affected area is clouded and the valves slightly swollen, due to cellular infiltration and œdema. In this softened condition the impact of one valve upon another roughens the surface along the line of contact, and there is deposited at this point blood platelets, leukocytes, and fibrin, the quantity of each varying at different stages and in different cases. This deposit on the leaflet, tendon, or muscle is at first microscopic, but by accretion may attain relatively massive proportions, and constitutes what is called a vegetation; essentially it is a thrombus. Often a row of these wart-like bodies is festooned along the line of contact or project into the blood stream. Their disturbance of the blood current by obstruction and by rendering accurate coaptation of the opposed leaflets impossible, and thereby permitting regurgitation, causes a murmur, an appreciation of which is necessary for accurate diagnosis during life.

Once formed, these vegetations are subject to important changes. They may be detached and swept off into the circulation, or they may soften and possibly be absorbed. Adhesions between leaflets may occur, but commonly the vegetations organize and permanently alter the contour, flexibility, and elasticity of the valves, thereby interfering with their proper functions. Finally calcifying deposits may further add rigidity to the already damaged leaflets. In favorable cases the valve surface becomes smooth, but little thickening results, and function is more or less fully restored.

Acute endocarditis ends in one of three ways: 1. The acute inflammation may subside and leave little or no alteration behind it. 2. The vegetations may persist and form large masses of an almost nodular character upon valves. 3. The valves become eroded or adherent, or cicatricial contractions may lead to distortions and consequent immediate insufficiency or obstruction.

Symptoms.—In distinction from physical signs, there are no symptoms of ordinary acute endocarditis of the benign form. It is true that the pulse may be a little quicker than before the endocardium was affected and the fever a little higher, but neither of these symptoms are constant or characteristic. Some *palpitation* may be present, but so frequently are all symptoms absent that all too frequently the physician who is not careful fails to discover endocardial disease until the patient begins to move about and complains of *cardiac weakness* or *dyspnœa,* and then the damage is done and is almost irreparable. Even the presence, on physical examination, of a *murmur* over the mitral or aortic area does not prove the presence of endocarditis, because it not infrequently happens that a murmur due to anæmia or to relaxation of the orifice is present. The presence of the murmur, while not pathognomonic of acute endocarditis, is, however, sufficient ground for the physician to treat his patient as a case with this lesion.

Complications.—These consist most commonly of pericarditis and embolism of one of the cerebral or pulmonary arteries. Rarely acute cardiac dilatation ensues, and sometimes in infectious cases pneumonia and pleuritis develop.

Diagnosis.—Care must be taken, as just stated, that anæmic murmurs, murmurs due to relaxation of the orifices, and pericardial friction sounds are not mistaken for those due to endocardial disease. (See Valvular Disease.)

Prognosis.—Death is very rarely due to acute simple endocarditis in the sense that death comes during the acute stage of the disease. All too frequently it follows as a consequence of the changes produced in the valves and heart muscle months or years after the acute stage has passed. A bad prognosis can always be given if the physician does not strenuously insist upon the patient resting in bed for several weeks after all articular and valvular signs have ceased. Ill-health and death may not come soon in these cases, but it often comes years afterward, as time and chronic disease weakens the valves and heart muscle. Children who develop acute endocarditis and are permitted to move about too soon almost always succumb before or at puberty to these secondary changes.

Treatment.—To prevent endocarditis in the course of all infectious diseases, and particularly in acute rheumatism, the physician must insist on absolute rest in bed all through the illness and for some time after the attack has passed. The diet should be light and easily digested, and at no time should the digestive apparatus be overburdened, for active and prolonged digestion tires the heart. If acute rheumatism is present, the salicylates should be used freely at once, not that they protect the valves directly, but they shorten the illness and so diminish the chance of involvement of the endocardium. Over the præcordium, as a preventive of endocarditis and pericarditis, should be placed six or eight flying blisters, and alkaline diuretics, like citrate of potassium, should be freely used. An ice-bag may be placed over the heart if it is very irritable, and tincture of aconite may be given for the same purpose. After the endocardial symptoms have developed, rest of the most absolute character is the only useful plan of treatment. During convalescence rest is again the *sine qua non.* For the anæmia often present iron and arsenic are useful, as is also cod-liver oil. Subsequent cardiac feebleness is to be treated by small doses of digitalis, as 3 to 5 drops of the tincture with 10 drops of tincture of nux vomica three times a day.

Ulcerative Endocarditis. Definition.—Ulcerative endocarditis, of which the synonyms have already been given, is a state in which the endocardium is ulcerated, vegetations are present, and there is an actual loss of substance in the valvular tissues, so that a valve or even a septum may be perforated. A French physician, Bouillaud, first recognized this cardiac state with its associated signs of pyæmia, but Kirkes, of England, first emphasized the fact that the heart was the seat of the difficulty, and that the symptoms arose from its condition. Since his time a host of pathologists, including Virchow, Wilks, Murchison, Charcot, Vulpian, and Birch-Hirschfeld in Europe, and Osler in this country, have studied this malady. It is important to remember that ulcerative endocarditis occurs in an acute and chronic form.

Etiology.—The disease is always due to microbic infection of the endocardium. It may be due to a secondary infection, during the course of one of the acute infectious diseases like typhoid fever, scarlet fever, pneumonia, or tonsillitis, or it more rarely arises as a primary lesion. The organisms are usually the *Staphylococcus pyogenes aureus*, the *Streptococcus pyogenes*, and the *Pneumococcus*, the *Bacillus typhosus*, the *Gonococcus*, the *Bacillus coli communis*. Acute endocarditis due to the *meningococcus* of Weichselbaum is very rare. A number of cases have been reported in which the *meningococcus* has been found in the circulating blood: one by Gwyn in 1889, another by Salomon in 1902, and a third by Warfield and Walker in 1903. The last of these is the only one in which the *meningococcus* was demonstrated to be the cause of the endocarditis. Any damage, new or old, to the surface of a valve predisposes that part to infection.

Pathology and Morbid Anatomy.—Anatomically, the ulcerative form may be but a later stage of the acute simple type, and many cases occur in which no sharp line of demarcation can be drawn. Not infrequently it is engrafted upon an old or chronic valvular lesion, and patients having such lesions should be watched closely during an attack of any infectious disease associated with the constant or frequent occurrence of bacteræmia. The infective process in the ulcerative type leads to necrosis of the already formed or forming vegetations, and even of the affected leaflet or adjacent myocardium. The fragments thrown into the circulation cause infarction and metastatic lesions in many tissues, especially in the spleen, kidneys, and brain. Marrow lesions, joint complications, and other manifestations of septicopyæmia are often conspicuous. Whether the secondary processes be suppurative or not depends upon the character of the infecting organism. When involving the valves of the right heart (as it does more commonly than the acute simple form), pulmonary complications may be conspicuous, which, taken with the fact that it may be a sequence or complication of pneumonia, further tends to obscure the seat of the primary lesion. Occasionally the almost symptomless progress of the malady is due to the low toxicity of the infecting organism, while intensely toxicogenic bacteria reverse the picture and cause evidences of severe infection and toxæmia to be manifest.

Symptoms.—The objective symptoms of ulcerative endocarditis may be no more marked than those of the simple form. Fever may not occur. If the disease develops during the course of one of the acute infectious diseases or as a result of septicæmia, its existence may not be suspected. If it develops primarily the physician who does not carefully study the heart may believe that the *fever* in its acute period of rise and fall is the manifestation of one of the acute infectious diseases or he may suspect sepsis, typhoid fever, or laria. Because of these symptoms, the disease may be divided into the septic form, the typhoid form, and the malarial form. A cerebral form also exists.

In the *septic form* the patient presents the ordinary signs of septicæmia. Chill after chill develops, and between the chills high fever and sweats are present. The patient looks profoundly septic, the tongue is dry, the eyes sunken, and petechiæ may be present in the skin. The marked anæmia is very noteworthy, and its severity is diagnostic. Leukocytosis is present. Multiple metastatic abscesses may be found. The heart may or may not produce a murmur, but its action is hurried and feeble.

The *typhoid type* is closely allied to that just described. The dry tongue, the subsultus, the tympanites, the diarrhœa, the mental stupor, the swollen spleen, and the remitting fever may all present so typical a picture of a case of typhoid fever that only the constant recollection that such symptoms may be due to ulcerative endocarditis will save the physician from an error in diagnosis. Even epistaxis may develop.

In the *malarial type* the constant recurrence of moderate chills, moderate fever, anæmia, and some sweating may be very misleading.

In the *cerebral form* there is severe headache, unconsciousness, and convulsions due to an associated meningitis. In some instances the simultaneous development of septic arthritis makes the case resemble acute rheumatism. Burrows has reported a case in which vomiting and purging were so severe that he believed the patient to be suffering from homicidal poisoning.

Cases are recorded in which the *splenic enlargement* was so great that the patient was thought to be suffering from splenomedullary leukæmia. In other cases the diagnosis of acute tuberculosis has been made.

Albuminuria and *hæmaturia* are common symptoms, but renal infarction may take place without either albumin or blood being found in the urine.

Endocardial murmurs are not always present. They are shifting in time and in character and may be found one day and be lost the next. Further, it sometimes happens that the murmurs change in character from day to day, owing to the progressive character of the lesions. When murmurs exist they are more frequently due to old lesions than to the new ones produced by the acute infection.

Complications.—The complications of ulcerative endocarditis are many and serious. It not infrequently happens that a septic embolus not only plugs an important vessel, and so causes an infarct in such organs as the kidney, the lung, and the spleen, but it acts as a focus of septic development. Such embolic closure frequently occurs in the branches of the left middle cerebral artery, and causes temporary or permanent aphasia or hemiplegia. Peripheral vessels, such as the popliteal or brachial or lingual arteries, may also be affected. Occasionally violent abdominal pain, followed by bloody stools and signs of collapse, indicates that embolism of the mesenteric vessels has taken place. Sometimes uræmia, due to the septic nephritis which is present, ends the patient's life.

Diagnosis.—The points of value in diagnosis are the suddenness of onset in some cases, the up-and-down temperature waves, which form steep curves on the charts, the repeated rigors, the presence of distinct leukocytosis, the presence of pyogenic organisms in the blood, the absence of the malarial organism and of the Widal reaction, and, finally, the presence of great feebleness and irregularity of the heart's action. In some cases, however, the diagnosis from physical signs may be practically impossible. The Widal test may throw much light on the case if persistently positive.

Prognosis.—It is hardly necessary to state that the prognosis is most grave. Recovery rarely occurs, and death may take place in the first two weeks or earlier. Sometimes life is preserved for weeks. The duration depends largely upon the character of the infection, the condition of the heart and of its valves, and the occurrence of complications. Some cases extend over a period of several months, and most of them last for several weeks. That healing may take place and recovery occur in cases of true ulcerative endocarditis is proved by a large number of cases now on record, in which the condition has been proved by subsequent autopsies to have existed, the patient dying of another malady.

Treatment.—The treatment of ulcerative endocarditis is not very satisfactory. Antistreptococcic serum may be of benefit in a few cases, provided that the streptococcus is the cause of the disease, and provided that the variety of streptococcus used in the preparation of the serum is the same as that present in the heart. Aside from this specific treatment, the only thing to do is to support the system by the wise use of tonics, such as tincture of the chloride of iron and the tonic bitters. Full doses of quinine may be used. The most important function of the physician is to maintain nutrition by the use of good food and to order no drugs which, by disordering digestion, will interfere with the digestive and assimilative functions.

Chronic Endocarditis.—As already stated, chronic endocarditis is frequently a sequence of one of the acute forms just described, and, therefore, as a rule, it affects the left side of the heart and the mitral leaflets oftener than the aortic valves. In some instances, however, it depends upon alcoholism, gout, and syphilis, in which case associated changes in the heart muscle and bloodvessels are also found. A slowly progressing valvulitis or sclerosis of the valves, which comes on insidiously, is frequently associated with arteriosclerosis (arteriosclerotic endocarditis), with chronic renal disease, chronic metallic poisoning, especially that due to lead, and other conditions associated with high arterial tension, with or without the presence in the blood of some specific irritant to which the changes may be ascribed. As the acute inflammatory process merges into the chronic form, one of two changes appear in the endocardium. There is an overproduction of connective tissue in the endocardium, with thickening, stiffening, and lessened elasticity, which chiefly affects the valvular leaflets. Following this condition, as a result of further degenerative changes, we find contractions or localized yieldings of the valves which produce an unevenness of their surfaces, so that their edges can no longer be accurately approximated; nor do they permit the free flow of blood through the orifice which they guard, since they are unable to yield during the period at which a free flow of blood should normally take place. Even when the valves are thickened and distorted they may still be adequately covered by endothelium, but in some instances the endothelium may be absent, thereby exposing calcareous and roughened surfaces upon which fibrin is sometimes deposited. The chief factors in producing cardiac failure in chronic endocarditis are irregular contractions which distort the valves, causing their edges to become everted, inverted, or curled up. In addition the chordæ tendineæ which control the valves guarding the auriculo-ventricular orifices become shortened and thickened so that they interfere with the free movement of the valves. (See Diseases of the Myocardium.)

In that form of the disease in which mural endocarditis is present, patches of sclerosis or cicatrices may be seen over the walls of the ventricles. (See Chronic Valvular Disease.)

CHRONIC VALVULAR DISEASE AS A RESULT OF CHRONIC ENDOCARDITIS.

Chronic valvular disease of the heart is very constantly met with in medical practice, and its frequency is, as a rule, in direct proportion to the age of the patient examined. This is due to the sclerotic changes which are prone to take place in the valves as age advances, and to the fact that in those who have passed the period of middle life the heart in all its parts is unable to withstand the strains, which may come to it, as well as in earlier periods. The chief causes of valvular lesions may be placed in three divisions, namely: (1) those due to infectious diseases, particularly rheumatism, which may leave behind damage which only becomes apparent when age or some unusual strain weakens the heart muscle; (2) fibroid or sclerotic changes ensuing as a result of age, gout, syphilis, and alcoholism; (3) definite myocardial degeneration and dilatation which does not cause direct but indirect valvular failure in function, as described elsewhere. (See Relative Insufficiency.)

Experience in the larger London hospitals, some fifteen years ago, led me to believe that cardiac valvular disease was much more common in England than in America. It is interesting to note, however, that this view is incorrect, for out of 59,762 medical cases which are recorded in hospitals in London, there were 3059 cases of valvular heart disease, or a percentage of 5.1; and out of 91,985 medical cases in hospitals in different cities in the United States, there were 4108 cases of valvular disease, or a percentage of 4.4. The actual difference in frequency in England and America is, therefore, not very marked, not only as regards endocarditis, but acute rheumatism as well. (See Acute Rheumatism.)

VALVES AFFECTED.—Series of statistics differ somewhat as to the relative frequency with which different valves are affected. One difficulty is that there has never been a sufficiently large collection of statistics to give results free from error. Another difficulty lies in the differentiation of true and false aortic stenosis; for it is evident that certain statistics which give a large percentage of this lesion include cases in which there is not true simple stenosis (which is quite rare without regurgitation) and cases in which atheroma and aortic roughening cause a systolic aortic murmur.

All clinicians and pathologists are in accord in stating that mitral regurgitation is the most common lesion by long odds. Jürgensen has analyzed 2470 cases of valvular cardiac disease, with the following results as to the relative frequency with which valvular disease occurs: mitral disease, 1616; aortic disease, 457; pulmonary valvular disease, 56; tricuspid disease, 10; associated aortic and mitral disease, 224; associated mitral and tricuspid disease, 45; lesions at the mitral, aortic, and tricuspid valves, 24; and at the aortic and tricuspid valves, 2. Unfortunately he does not state what the lesions are—*i. e.*, regurgitant or stenotic. It is an open question, too, how many of the cases of so-called mitral disease and tricuspid disease were secondary murmurs due to dilatation of those orifices and not to true valvular defects.

Some years ago, T. G. Ashton, my then chief of clinic at the Jefferson Hospital, made an analysis of 1024 cases of valvular disease met with in life insurance examinations. His results showed that of these 557 were cases of mitral regurgitation, 136 were aortic stenosis, 47 were aortic regurgitation, 32 were mitral stenosis, and 11 tricuspid regurgitation. I believe that these statistics, while accurate in themselves, are to some extent misleading, and that the proportion of cases of aortic stenosis is too high. (See article on Aortic Stenosis.) The following figures obtained by the analysis of 908 cases of valvular heart disease treated in Westminster Hospital, London, show that the most common single lesions are mitral regurgitation, mitral stenosis, aortic regurgitation, and aortic stenosis,[1] in order of arrangement, and that of double lesions at one orifice the relative frequency is double aortic, double mitral, double pulmonary, and double aortic with double mitral. Mitral regurgitation quite frequently occurs as the result of aortic regurgitation, through dilatation of the mitral orifice.

Mitral disease affects more women than men; aortic disease more men than women.

Aortic regurgitation is the most fatal lesion. Mitral stenosis ranks second in fatality, aortic stenosis third, and mitral regurgitation fourth. The mortality of double aortic lesions is greater than that of double mitral.

The statistics of A. Lockhart Gillespie, based on a study of 1914 cases treated in the Edinburgh Royal Infirmary, are especially interesting in that they show the mortality of valvular lesions in the two sexes according to age. Gillespie found that the maximum mortality in males with aortic incompetence or stenosis occurs between the age of fifty and sixty-nine, but in those with double lesions the years from twenty to twenty-nine are those with the highest mortality. The female maximum mortality in aortic incompetence and aortic stenosis falls between the years of forty-nine and fifty. Mitral stenosis proves most fatal at from thirty to thirty-nine years in males, and from forty to forty-nine in females. The death rate in females between twenty and twenty-nine, forty and forty-nine, and fifty and sixty-nine is higher than in males at similar periods. The death rate in mitral incompetence in both sexes rises progressively with the age. In cases of double mitral lesion, the male maximum mortality falls between thirty and forty-nine, and in the female between fifty and sixty-nine.

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[1] This probably refers to true stenosis and not to cases in which only the aortic systolic murmur was present. (See Aortic Stenosis.)

Before proceeding to a consideration of the various valvular lesions it is essential that the mechanism of the valves in health and disease be clearly understood (Fig. 69). The cardiac valves are arranged in such a way that they prevent a reflux of blood into that cavity which the blood has just left in the progress of the normal circulation. As a rule, these valves are capable of fitting together so tightly that they completely and effectively close the orifice which they guard, but even without the presence of any condition of disease they may at times give way, and permit some reflux. The moderate reflux occurring during great muscular strain may be regarded as a physiological attempt to relieve the blood pressure in the cardiac cavities, and if it is not maintained for too great a length of time it does no harm.

FIG. 69

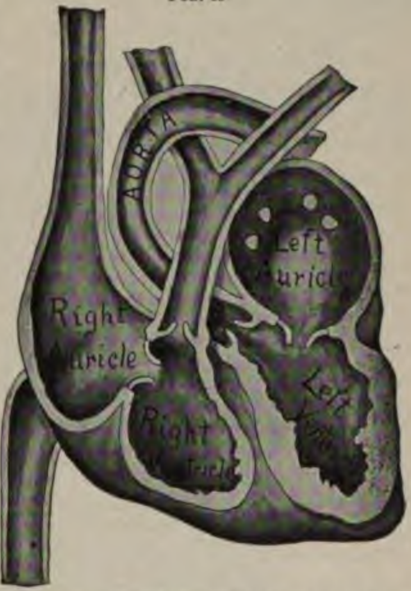


Diagram modified from Page to show the relation of the various valves. A study of this diagram will render clear the time of the various cardiac murmurs. Thus in mitral regurgitation the blood passes back from the left ventricle to the left auricle during systole, and is dammed up in the pulmonary veins, the openings of which are seen in the auricular wall, producing pressure on the pulmonary valves, the sounds of which are thereby accentuated.

It must also be recalled that there are at least two ways in which the cardiac valves may become incompetent to prevent reflux of blood. In the first, and by far the most common type, the valves are diseased, as already described in the article on endocarditis, so that they cannot become closely approximated, or they are glued together in such a way that the same result is achieved, and so they also obstruct the flow of blood. In the second type the rings, which form the bases of the valves and the margins of the orifices, yield, and as they relax the orifice becomes too large to be closed by the valves, which may still be practically normal in themselves. This condition exists for a brief space of time in acute cardiac strain, as just stated. It persists for a long time or becomes permanent in instances where the heart is feeble and the strain is very severe or prolonged, and it is frequently found in cases of dilatation and feebleness of the heart muscle.

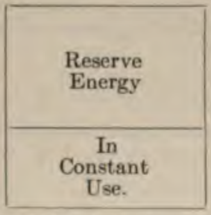
Those forms of valvular incompetence which occur in athletes or others after severe exertion can therefore be put aside as beyond the scope of these particular pages, although they will again be discussed under the head of functional disorders of the heart.

In those cases in which the valve becomes incompetent to close an orifice, and so permits regurgitation to take place, the failure of the valve is so gradual, as a rule, that there develops simultaneously an increase in the size and strength of the heart muscle, so that it may by increased power and activity compensate for the leakage which occurs. As a result it very frequently happens that this compensatory hypertrophy is fully equal to the increased demands made on the heart muscle, and not until the occurrence of a severe illness, or until advancing years impairs the power of its fibres, are any manifestations of valvular lesions to be found in the patient, save the physical signs of hypertrophy and the murmur caused by the regurgitating blood. Sometimes even the murmur may disappear for a time.

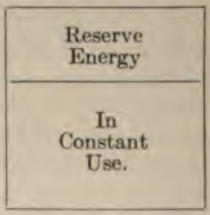
In cases in which a valve is ruptured or severely damaged by disease so that it fails in its function before the heart can undergo compensatory hypertrophy, we often see signs of great circulatory embarrassment from the very first part of the illness.

In all cases of valvular disease in their early stages much depends upon the inherent strength of the heart muscle and its ability to increase in power, and therefore it is evident that it is of vital importance for the patient to rest at this period in order that the strength of the heart may be conserved, and in order that it may not be subjected to a severe strain with associated dilatation at the most critical period of its existence. This is the more important because diseases which secondarily infect the valves usually impair, to some extent at least, the myocardium as well.

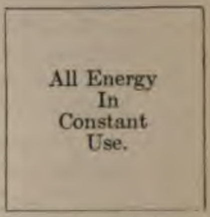
In health, when the valves are intact, the heart always possesses a considerable degree of reserve energy and power, using only a small part of its store of energy in a day's work. As a consequence a healthy man can run a considerable distance, or leave his desk and go hunting, without engendering anything more than fatigue of his voluntary muscles and some healthy cardiac tire. This reserve energy is kept for just such purposes. On the other hand, if a man who is a sufferer from valvular or myocardial disease, even if he is seemingly in perfect health, attempts to follow the first one, he soon begins to suffer from cardiac embarrassment, and if he persists may become very gravely ill from acute cardiac failure, and rupture his compensation by excessive exercise to such an extent that he may be bedridden for the rest of his days. In the latter instance nearly all his reserve energy is being used daily in the maintenance of a normal circulation and, having little reserve, he cannot undertake feats that demand great calls upon his reserve. This is illustrated in the following squares:



In health, large reserve.



In disease, small reserve.



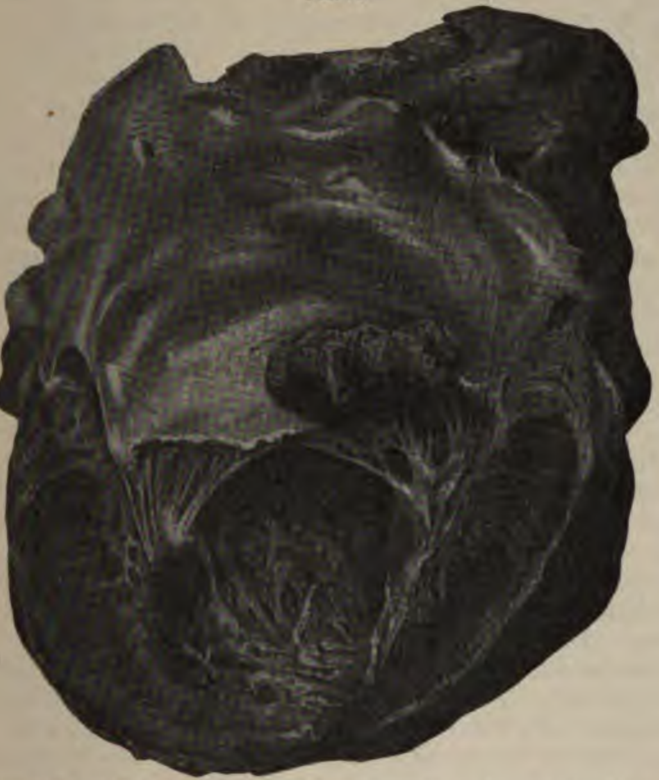
Far advanced disease,
no reserve

In the last square it is seen that all the reserve is in constant use, and, therefore, if any extra exertion is made, the heart promptly fails and death may occur. Even in those cases in which sufficient hypertrophy develops to adequately compensate for the leak in the valve, the heart is never as capable for work as in health because the reserve is never restored completely and the degree of leakage may increase at any moment of strain.

A consideration of these facts makes one therapeutic fact stand preeminent above all others, namely, that rest is the chief measure to be instituted whenever compensation is failing, as by rest alone can we expect to restore reserve energy.

These remarks have so far dealt with regurgitant conditions. In stenosis of the cardiac orifices the same facts hold true, for in such cases the question is whether the heart muscle possesses enough strength to drive the blood through the obstructed area.

FIG. 70



A large vegetation on the mitral leaflet. (Kast and Rumpler.) It can be readily seen that this would cause both a mitral obstructive and a mitral regurgitant murmur.

Given a case of valvular disease the prospects of survival depend almost entirely upon the ability of the heart to undergo compensatory hypertrophy, and therefore the prognosis depends largely upon the state of the muscle, the absence of arteriocapillary fibrosis, which, if present, strains the heart and wearies it, the ability of the patient to pursue an easy occupation and his willingness to avoid habits of life which strain the heart.

With these preliminary remarks we may pass on to a discussion of the individual valvular lesions, taking up first of all the most common of them, namely:

Mitral Regurgitation.—Mitral regurgitation, often called "mitral incompetency" or "mitral insufficiency," depends in the great majority of cases upon thickening, shortening, or distortion of the mitral leaflets, those bicuspid valves which in health guard the left auriculoventricular orifice, in such a way that the blood when pressed upon by the contracting walls of the ventricle cannot regurgitate into the left auricle.

Associated with this valvular defect there are usually vegetations on the edges of the valves which prevent proper approximation of their edges. The chordæ tendineæ, which extend from the ventricular wall to the leaflets for the purpose of giving them support, are shortened so that they will not permit the full movement of the valves. In some instances the valves, their fibrous bases, the chordæ tendineæ, and even the endocardium are so completely calcareous that the ordinary physiological functions of the part are impossible. It is evident that it is almost impossible for such advanced changes to be present without at the same time causing some obstruction to the flow of blood from the auricle to the ventricle, and therefore we find that in nearly all cases of well-developed mitral regurgitation some mitral stenosis also exists.

Pathology.—The morbid anatomy has already been discussed under the heading of Endocarditis. The morbid physiology or pathology of mitral regurgitation is as follows:

During systole the blood from the left ventricle in cases of mitral regurgitation flows in two directions. A larger part escapes into the aorta, as in health, and a smaller part of it regurgitates through the imperfectly guarded mitral, or left auriculoventricular orifice, into the left auricle. The results of this regurgitation are multiple. In the first place the auricle receives more blood during diastole than is normal, for it receives not only the blood which comes to it from the pulmonary veins, but it also receives the blood which regurgitates from the left ventricle. This excess of blood requires the auricle to dilate beyond its ordinary capacity, and if the excess of blood is great this dilatation of necessity means distention. If the regurgitation progresses gradually there is developed a certain amount of hypertrophy in the auricular walls which enables the auricle when it contracts to empty itself completely and to prevent continuous overdistention, but the muscular fibres in the auricle are never well developed as compared to those of the ventricle, and therefore compensatory hypertrophy can never be so complete.

The second result of this lesion is dilatation and hypertrophy of the left ventricle, which is due to several causes, namely, the fact that when the left auricle empties itself it delivers to the ventricle an excess of blood over the normal quantity, for the reasons just given. To hold this excess of blood the ventricle must dilate, and, to expel it on contraction, the ventricle must undergo hypertrophy. The general system still requires as much blood as before, and in order to provide it with that quantity the ventricle must increase its activity in order that the amount lost by regurgitation may be compensated for by increased cardiac action.

The third result is found in dilatation and hypertrophy of the right ventricle, the labors of which are increased by the fact that the engorgement of the left auricle renders it difficult for the pulmonary veins to empty themselves into it. As a consequence they, and their tributary branches, become engorged, raising the resistance in the pulmonary vessels, and so the right ventricle finds it less easy to pump blood through the lungs. If the pulmonary engorgement is very marked, and dilatation develops in the right ventricle more rapidly than does compensatory hypertrophy, there is produced an insufficiency of the tricuspid valves guarding the right auriculoventricular orifice.

Fourth, the right auricle now feels the same stress as was felt primarily by the left auricle, and it undergoes dilatation and hypertrophy, but this hypertrophy is rarely, if ever, adequate to the task set before it, and as it fails to properly empty itself, evidences of engorgement of the systemic veins becomes manifest in that they become swollen, the liver and kidneys are congested, and œdema develops in the lower extremities.

Fifth, certain definite changes take place in the lungs. As a result of their being constantly engorged with blood, they suffer from brown induration and atheromatous changes appear in the pulmonary arteries and veins as years go by. Finally, when the systemic veins are engorged we find in addition serious congestion at the bases of the lungs as a result of impotence of the right ventricle and obstruction to the flow of blood from the pulmonary veins.

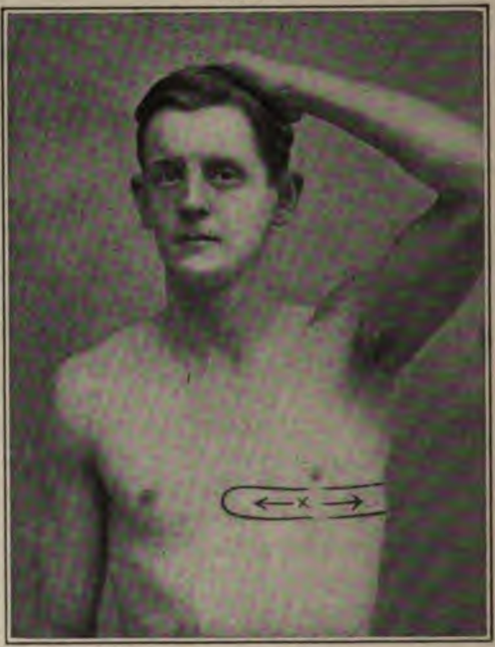
Lastly, we find, at autopsy, in these cases red atrophy of the liver and engorged, cyanotic kidneys.

In those cases in which there is no primary disease of the mitral leaflets, but in which they fail because of acute dilatation of the ventricle so that the auriculoventricular orifice is widened and the valves cannot close it, there is developed the same train of symptoms save that they are more rapid in onset and more severe. This condition develops after great cardiac strain, in which the aortic valves are ruptured or the left ventricle and its auriculoventricular orifice greatly dilated. The resulting engorgement of the lungs may be so severe that their bloodvessels may rupture and profuse hæmoptysis ensue. These patients nearly always succumb shortly, or remain chronic invalids, because the stress develops so suddenly that compensatory hypertrophy cannot take place. We have, therefore, an apparent paradox, namely, that actual disease of the mitral valves is rarely so serious in its consequences as is incompetency of these valves due to other causes.

Still another cause of mitral incompetence, aside from actual primary valvular disease, is Bright's disease, which increases arterial tension and thereby throws an increased strain on the left ventricle and the mitral valves when the blood is to be thrown out into the aorta. Usually in these cases the renal condition indirectly impairs the power of the ventricle, rendering its nutrition faulty through impoverishment of the blood and toxæmia, and valvular failure then arises as a result of gradual widening of the auriculoventricular orifice.

Symptoms.—It must be evident from what has just been said that many cases of mitral regurgitation may present no symptoms for years after the lesion is established, for as it develops so does a compensatory hypertrophy develop. It is only upon extra exertion, which the heart is not prepared to meet, that symptoms of cardiac embarrassment ensue, and if the exertion is not very severe and not repeated, the *dyspnœa* and *palpitation* from which the patient suffers may be considered by him as due to indigestion. If no illness impairs the heart muscle and no laborious pursuit causes it too great stress, the patient advances in comfort to old age, when symptoms develop as a result of the fact that his arterial tension gradually increases, thereby giving his heart more work to do at each beat, and at the same time his heart muscle undergoes the changes incident to advancing years. Under these conditions compensation "ruptures," to use the ordinary term applied to this unfortunate state, and subjective and objective signs appear.

FIG. 71



Showing at *x* the apex beat, where the murmurs of mitral regurgitation and obstruction can be best heard. The arrow pointing to the axilla indicates the direction in which the regurgitant murmur is transmitted, and the arrow pointing to the sternum the direction of transmission of the obstructive murmur.

The subjective symptoms (that is, those felt by the patient) vary considerably in the early and mild degrees of failing compensation. In some instances *shortness of breath* on exertion brings the patient to the physician, in other instances *digestive disturbances* due to hepatic congestion and secondary gastric catarrh are complained of, and in others the patient may complain of a *cough*, which is due to a mild pulmonary congestion and bronchitis having its origin in the cardiac failure. In still another class they may *suffer excessively from cold* in moderately cold weather, and perhaps become easily fatigued while walking in cold air because the cold contracts the peripheral capillaries and so increases the labor of the heart. When the failure of the heart is well marked, then dyspnœa, inability to lie down because of oppression, and pain in the epigastrium are perhaps the symptoms of which the patient will most complain.

The objective symptoms (that is, those seen by the physician) are even more characteristic. The capillary circulation is sufficiently impaired to produce some stasis and consequent *cyanosis of the lips* and finger-tips or even of the face. The *fingers*, particularly in young persons in whom the disease has lasted for some time, are *club-shaped*—that is, they do not taper, but have thickened tips; there is more or less *œdema* of the feet and ankles, and perhaps *blood-spitting* as a result of intense pulmonary engorgement or infarction.

Physical Signs.—The physical signs in such a case are usually well developed. *Inspection* of the præcordium reveals a diffuse and perhaps forcible apex beat. *Palpation* shows a distinct thrill in children, and in this class of patients this thrill can frequently be seen as well. The apex beat may be distinctly felt well outside and below the nipple line. If the compensating hypertrophy is well developed the apex beat may be forcible, but if compensatory hypertrophy is lacking it is feeble and diffuse. On *percussing* the præcordium the normal area of cardiac dulness will be found to be enlarged. This enlargement is usually transverse or lateral, so that it may extend to the right edge of the sternum owing to enlargement of the right ventricle, and as far as the left of the nipple from dilatation and hypertrophy of the left ventricle.

Auscultation provides us with the signs that determine the exact nature of the lesions, for the signs so far described are not distinctive of mitral regurgitation. When the ear of the physician is applied over the apex of the heart there is heard a soft, and often quite loud, murmur, which occurs synchronously with the apex beat, or with systole, or contraction of the ventricle. This murmur is transmitted to the left axilla, and it may be to the angle of the left scapula. If it is very loud it may be heard in any part of the chest. If the regurgitation is great enough to cause engorgement of the lung the pulmonary second sound, due to a quick shutting of the pulmonary valves guarding the orifice of the pulmonary artery, may be louder than normal. This is best heard at the third left costal cartilage. When the regurgitation is severe enough to have resulted in tricuspid regurgitation from engorgement of the right side of the heart, there may be heard at the fifth costal cartilage on the right side a comparatively soft systolic murmur due to this secondary leak, but in some instances the mitral murmur completely obscures the tricuspid murmur. In others the tricuspid murmur can be heard only at the ensiform cartilage.

It is of vital importance for the physician to recall the fact that a murmur so slight as to be almost inaudible is not an indication of the presence of a small and unimportant lesion of the mitral valve. On the contrary, the presence of a faint murmur often, but not always, indicates that the heart is too feeble to drive the blood with sufficient force to make the murmur clearly audible.

The pulse of mitral regurgitation varies, of course, with the extent of the lesion and the degree of compensation. It is usually nearly normal, though lacking somewhat in volume even when compensation is complete. It is irregular and small when compensation is insufficient. When compensation is ruptured, it is very small and hobbling.

Diagnosis.—It is desirable to separate mitral regurgitation due to dilatation of the mitral orifice from mitral regurgitation due to true valvular disease. This is in many cases impossible if the action of the heart is already seriously impaired, the more so because in many instances the secondary dilatation produces some widening of the orifice. While the physical signs may not be distinctive the absence of any history of rheumatism or other infection which affects the valves, and the presence, or history of the presence, of any exhausting illness which weakens the heart muscle, or of any strain which has dilated the orifice, would indicate that dilatation rather than true valvular disease is present. This is particularly true if the strain, though moderate, has occurred during convalescence, while the heart is weak. This differentiation is important because in some cases with returning health and strength the leak may cease and perfect recovery ensue, whereas if the valve is actually diseased good health can only come from compensatory hypertrophy and the murmur will probably always persist.

The possibility of a murmur being due to anæmia is also to be recalled, but hæmic murmurs are usually very soft and are best heard near the base rather than at the apex.

The diagnosis of mitral regurgitation is therefore chiefly based upon the presence of a murmur heard most clearly with systole at the apex and transmitted to the axilla.

Prognosis.—The prognosis in a case of mitral regurgitation depends upon the age of the patient, his occupation, the general condition of his vitality, and the severity of the lesion. When a child develops mitral disease it often does not survive puberty, chiefly because dilatation in such a case is usually excessive and because the constantly increasing demands of its growth require of its heart more than it can provide. In young adults who will rest after damage to the valves during an attack of rheumatism until compensation is really established, the prognosis is good, provided that the subsequent occupation is not too strenuous, although with the onset of old age vascular changes will probably cause breakdown. In feeble persons, however, the prognosis may be unfavorable from the onset, particularly if hypertrophy is lacking and dilatation is marked. In old persons the prognosis is bad for obvious reasons, since the heart cannot, at this time of life, readily gather new strength to meet new demands.

If syphilis or alcoholism are factors in the case the prognosis is grave, and if the kidneys are diseased the prognosis is, of course, very bad. So, too, the presence of arteriocapillary fibrosis with high arterial tension is a serious or grave factor, in that it gives the heart so much work to do. (For the relationship of this lesion to age and mortality, see page 490 in article on Chronic Valvular Disease.)

Treatment.—(This is discussed at the end of the articles on Valvular Disease.)

Mitral Stenosis. **Definition.**—By mitral stenosis we mean a condition of the tissues composing the mitral valves or surrounding the left auriculoventricular orifice whereby the blood is prevented from passing with normal ease from the left auricle to the left ventricle. It is sometimes called "mitral obstruction."

Etiology and Pathology.—Mitral stenosis is far more common in females than in males, although the reason for this is not clear. Sir Dyce Duckworth found that out of 264 cases of this disease 177 were females, and other statistics are practically in accord with Duckworth's. It is due in the great majority of instances to gluing together of the mitral valves as the result of acute endocarditis arising from acute rheumatism.

With this adhesion of the valves there is often associated a growth of vegetations on their edges and not uncommonly thickening and sclerosis of the chordæ tendineæ, the papillary muscles and the leaflets themselves, so that the parts lose their elasticity and are so stiffened that they are unable to move out of the way of the blood stream when it seeks to pass into the ventricle. This sclerotic process is so marked in some cases that the tissues may seem almost cartilaginous in character, and in advanced life lime salts may be deposited to such an extent that the fibrous tissue is calcareous and even the walls of the ventricle are infiltrated by calcareous masses. In some instances the adhesions are so complete that the valves form a funnel-shaped tube through which the blood must find its way. In other instances the edges of the valves are adherent and their margins thickened, leaving only a small orifice between them, forming the so-called "button-hole" mitral orifice, and in still other cases the conjoined edges of the valves are so drawn or puckered that the orifice when the auricle is opened looks like the normal anus.

The funnel-shaped opening is most commonly seen in children, but it is not always, as some have thought, a congenital lesion; the button-hole orifice is much more common in adults. Sansom states that the proportion is one button-hole to eight funnels in children, and that as adult life is reached the proportion changes to twenty-five button-holes to one funnel.

There can also be no doubt that chronic contracted kidney with associated arteriocapillary fibrosis not only causes mitral stenosis, but that even the funnel-shaped opening just described may be developed in this type of cases. The studies of Duroziez and Huchard in France, and of Goodhart, Sansom, and Pitt in England prove this fact. In 542 autopsies in cases of interstitial nephritis, Pitt found mitral stenosis 33 times.

The results of mitral obstruction are overdistention of the left auricle and scanty blood supply to the left ventricle. The engorgement of the left auricle results from its inability to empty itself, and leads to dilatation and to some degree of hypertrophy. In some instances the growth of muscle fibres is so scanty that no true hypertrophy ensues, in others they seem to become actually atrophied, but in other instances very complete compensatory hypertrophy develops, so that the auricular wall is distinctly thicker than normal. Indeed, it may appear as a firm, muscular mass which does not collapse at autopsy as the ordinary auricle is wont to do. Whether hypertrophy exists or not, dilatation is always present. When such an auricle is opened the endocardium is often found to be thickened and a laminated clot may line its cavity. In other instances polypoid or globular coagula are attached to the auricular walls, and these globular thrombi may almost fill the auricle. At times the thrombus is free, acting as a ball valve in the auriculoventricular orifice.

The wall of the left ventricle in these cases is often thinner than usual, but dilatation and hypertrophy may be present.

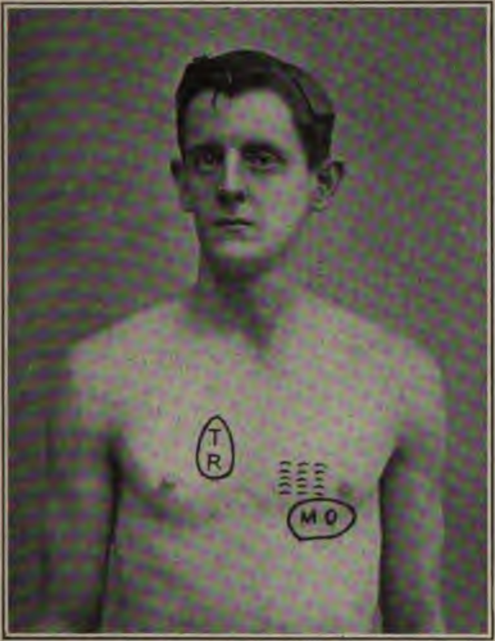
The accumulation of blood in the auricle leads to the engorgement of the lung, and the same changes occur in its vessels and tissues that have been described under "Pulmonary Congestion" and "Mitral Regurgitation." So, too, the right ventricle undergoes hypertrophy and dilatation from similar causes, and the tricuspid valves, even more frequently than in mitral regurgitation, give way, so that pulsation of the jugular veins, pulsation of the liver, and œdema of the lower extremities finally develops. Embolism often occurs in mitral stenosis, and, as in all cases of embolism arising in the heart, the embolus usually lodges in the left hemisphere of the brain.

If the narrowing of the auriculoventricular orifice is not progressive, and if the auricle undergoes compensatory hypertrophy, the signs of advanced disease may not ensue. It is only when auricular hypertrophy fails that the malady becomes manifest and causes the symptoms of cardiac failure which in all respects resemble those described under mitral regurgitation, save that the venous and hepatic pulsation just described are more frequently present.

PHYSICAL SIGNS.—The physical signs of mitral obstruction are as follows: *Inspection* of the chest may reveal pulsation at the apex of the heart near the nipple, and also near the base of the heart close to the sternum, in the second or third left intercostal spaces. Not rarely there may be, in addition, epigastric pulsation. If a straw, or piece of cardboard, be placed over the pulsating spots at the apex and base, it will be found that they do not move synchronously, but the lower one moves with the ventricle and the upper one with the auricle. It has been claimed that the auricle does not produce this upper pulsation, and that it is due to the movement of the conus arteriosus of the right ventricle, but this is seemingly disproved by the fact that the two movements are not synchronous with systole. If the patient be a child these pulsations are much more noticeable than if he be an adult, at which time of life pulsation may be absent. In children bulging of the chest wall close to the sternum and near the epigastrium may be quite marked, and if pulsation be well defined in this area and absent from the region of the nipple, indicating hypertrophy of the right ventricle, the diagnosis of mitral stenosis is strengthened, provided that adherent pericardium can be excluded.

Palpation does not always give us much information, but sometimes it practically decides the diagnosis, for there are *three signs on palpation* which are noteworthy in this lesion. There is a *thrill* which is *presystolic* in point of time and is felt in the fourth or fifth interspace inside the nipple line. It is characterized by sudden arrest at the moment of systole. This sign may be considered diagnostic of mitral stenosis provided we have excluded the possibility of aortic regurgitation, which sometimes causes a similar sign. This thrill may be present at one time and entirely absent at another, and it may be absent when the patient is in the dorsal decubitus and present when he is erect, or, again, it is present after exercise and absent after rest. The *second sign* of some importance in the diagnosis of this disease by palpation is the *heaving impulse*, felt just below the margin of the last costal cartilage on the left side without such impulse near the nipple. This has already been spoken of under inspection as indicative of hypertrophy of the right ventricle. Care must be taken, however, that signs of hypertrophy of the left ventricle are really absent, for it sometimes happens that an overlapping of the lung covers the left side of the heart so that it does not transmit its impulse to the chest wall. The *third sign* on palpation is the discovery of the edge of the *enlarged liver, which pulsates*, below the level of the floating ribs on the right side. Here, again, care is necessary, for it often happens that the liver is moved by a transmitted impulse from the heart muscle through the diaphragm.

FIG. 72



MO shows area of greatest intensity of a mitral obstructive murmur; TR shows area of greatest intensity of a tricuspid regurgitant murmur. The fine lines indicate the area in which is felt the characteristic thrill of mitral stenosis.

Percussion of the præcordium may reveal, in cases of mitral stenosis, a distinct increase in the area of cardiac dulness to the right, with comparatively little extension of dulness to the left. This is due to the enlargement of the right ventricle. When, however, the disease is far advanced and the heart is greatly dilated or hypertrophied the left margin of dulness is distinctly extended, and in such cases the area of cardiac dulness to the left of the nipple line may be very great.

Auscultation is, of course, the most important aid in the diagnosis of mitral stenosis, since it presents no less than *six points* of interest. The *first* of these is the presence of a murmur which occurs just before systole, a *presystolic murmur*, and is best heard between the nipple and the sternum, on the nipple level (Figs. 71 and 72). In most cases the murmur can only be heard in this area, but in some instances it is so loud as to extend all over the chest. The murmur is usually harsher than that of mitral regurgitation, and is vibratory in character. It is due to the passage of the blood through the obstructed auriculoventricular orifice, and it ceases with the close of auricular systole. In some cases the murmur may extend all through ventricular diastole. At times it is so metallic as to be musical rather than purring.

Another sign of importance is the *accentuation of the pulmonary second sound*, which is best heard at the third left interspace, and is due to the high pressure in the pulmonary artery, produced by back pressure on the column of blood in the lungs. This accentuation is, however, not so constant in stenosis as in regurgitation because of the greater irregularity of the heart in stenosis.

A third sign is the *reduplication of the second sound* of the heart so that it appears as a "tap-tap," or resembles the "postman's knock." It is very characteristic of mitral stenosis and is heard at two places: at the apex and at the base of the heart. It is, however, supposed to be due to different causes at each spot. At the apex Sansom believes it is due to the sudden rush of the blood into the ventricle through the narrow orifice under the pressure of the hypertrophied auricle. In other words, this sound is associated with the normal pulmonary and aortic second sounds. When this reduplicated second sound is heard at the base, and this is where it is usually heard, it is most diagnostic, and is supposed to be due to an asynchronous closure of the aortic and pulmonary valves, but in all probability the cause is similar to the sound at the apex. This is sometimes called the "gallop rhythm."

The fourth sign of importance is the loud and *sudden snapping sound* which is heard at the close of systole of the ventricle. It is supposed to be due to forcible snapping to of the bicuspid or mitral valves.

The fifth sign is not only somewhat indicative of stenosis, but much more of cardiac breakdown, namely, absence of the first sound of the heart at the apex.

Still a sixth sign of mitral stenosis is sometimes of value, namely, great *irregularity as to rhythm* and force. In no form of valvular lesion with rupture of compensation is the heart so tumultuous as in this disease. As already stated, the absence of a murmur may be more indicative of grave valvular disease than its presence.

When cardiac breakdown ensues it not infrequently happens that the presystolic murmur itself disappears because the auricle is too feeble to drive the blood through the auriculoventricular orifice with enough force to make a murmur.

Diagnosis.—Mitral stenosis is at times the most difficult of all the cardiac lesions to diagnosticate, chiefly because when compensation is ruptured no murmur may be present, and the action of the heart being exceedingly irregular its sounds are confused. Again, stenosis is so frequently associated with mitral regurgitation that the double murmur may cause confusion, the more so as only one murmur may be present at one time and both at another time, and also because the regurgitant murmur is often so loud that it covers the stenotic murmur, with the result that unless the physician is on the *qui vive* to discover the less noticeable sound it is overlooked. In cases which are manifestly ones of mitral stenosis the physician need not hesitate to express an opinion, but in the obscure forms of the disease he should always reserve his statement until he has had an opportunity of examining the heart several times under conditions of rest and exercise, and, perhaps, after the use of digitalis or some other drug to strengthen the muscle.

As the physical signs have already been thoroughly described, it is only necessary, at this point, to differentiate mitral stenosis from those conditions which resemble it.

The most important of these is the so-called "Flint's murmur" first described in 1862 by Dr. Austin Flint, of New York. It occurs in some cases of aortic regurgitation, and is supposed to be due to the regurgitating blood striking upon the mitral valves and chordæ tendineæ in such a way that they vibrate and so cause a sound. This sound is diastolic in point of time because it occurs after the blood has been sent out into the aorta and while the ventricle is receiving more blood from the auricle. Its time of occurrence is, therefore, practically identical with the murmur of mitral stenosis.

The following points make the differentiation between mitral stenosis and "Flint's murmur" in most cases, although in some cases the separation may be impossible. In cases with aortic regurgitation auscultation at the second right costal cartilage and along the sternum will reveal a diastolic murmur which will not be well defined in these areas in mitral stenosis unless the mitral murmur is so loud as to be heard pretty much everywhere in the chest. The pulse in aortic regurgitation is characterized by a full wave followed by a sudden fall—the "Corrigan pulse"—whereas the pulse of mitral obstruction is a fine thread, irregular and feeble. Again, in cases of aortic regurgitation with "Flint's murmur" there are rarely, if ever, those well-developed signs of pulmonary, hepatic, and splenic engorgement which have been described as occurring in mitral stenosis, nor does the patient so frequently suffer from hæmoptysis due to pulmonary congestion or infarction. Further than this, the sharp, snapping first sound of the heart characteristic of mitral obstruction is not present with "Flint's murmur."

A second condition resembling mitral stenosis is tricuspid stenosis. On general principles, this latter lesion can be excluded on the rule of probabilities, for tricuspid stenosis is an exceedingly rare lesion. If it exists it is usually heard best in the tricuspid area (the area of the fourth right intercostal space), but it may be clearly heard in the mitral area, and as tricuspid stenosis and mitral stenosis exist together in some cases and occur simultaneously, they may not be separable and nothing be known of the lesion on the right side of the heart until autopsy.

At times children suffering from adhesive pericarditis present a presystolic sound like that of stenosis. It is to be discovered by the signs of adhesive pericarditis (which see). In every case of valvulitis due to rheumatism we should bear in mind the possible if not the probable, presence of adherent pericardium. The presence of this condition is rendered likely if the liver is not only enlarged, but very firm, and if ascites develops in excess of that seen in cardiac dropsy. This point is of importance, because if the pericardium is adherent we cannot expect very good results from digitalis nor from any other method of treatment.

When a patient presents himself with a disordered circulation and confused or irregular heart sounds, and no murmur, it must be recalled that while such a state may be due to tobacco heart, it may also be caused by mitral stenosis with no murmur.

Prognosis.—The prognosis of mitral stenosis is not as favorable as is that of mitral regurgitation or aortic stenosis, and children nearly always succumb to it before they reach adult years. Adults who have a severe lesion also rarely survive for many years after it begins, but there are very marked exceptions to this rule. Thus, I have under observation at present a case of mitral stenosis which I examined twenty-two years ago, and who was told thirty years ago that the lesion existed. During all these years (he is now sixty-nine years of age) he has led a very active life, both physical and mental, with no cardiac embarrassment, although he had an attack of hæmaturia when I first saw him, which was due to an infarction of the kidney. During this time he has taken no treatment, except at rare intervals, his compensation being complete.

In young women with mitral stenosis, marriage and consequent childbearing often cause rupture of compensation and death.

The average age at death in cases of this disease is stated by Sansom to be about thirty-two and seven-tenth years. (See General Discussion of Valvular Lesions and Their Effect on Mortality, page 490.) (For treatment see close of these articles.)

Aortic Stenosis. **Definition.**—Aortic stenosis, often called "aortic obstruction," is a condition in which the left ventricle finds it more difficult than normal to expel the blood through the aortic orifice, because this orifice is narrowed by disease. The murmur which is produced by the blood under these circumstances is systolic in point of time, for it occurs as the left ventricle expels its contents. It is best heard at the second right costal cartilage or under the sternum, at its upper portion. It is of vital importance, however, to recall the fact that the presence of a systolic murmur at this point is not necessarily indicative of actual obstruction of the aortic orifice. An aortic systolic murmur does not necessarily mean an aortic valvular lesion. The murmur is usually due to roughening of the lining of the aorta by atheromatous plaques. Aneurysm may also be provocative of such a sound. So rare is true simple aortic obstruction that it may be said that the presence of a systolic aortic murmur is in most cases probably not due to this lesion, unless it is found associated with aortic regurgitation. Some clinicians of repute assert that they have never seen pure aortic stenosis without regurgitation. Cabot states that in 252 autopsies made at the Massachusetts General Hospital on persons with valvular disease, there was not a single instance of uncomplicated aortic stenosis.

Etiology.—The causes of aortic obstruction are multiple. In the first place, it may be the result of rheumatic endocarditis of so severe a type that not only the mitral but the aortic valves are involved, for it is only in rare cases that rheumatism attacks the aortic valves and leaves the mitral valves untouched. In such a case the endocardium covering the valves is roughened in patches, and upon these patches is deposited fibrin from the blood stream, which, with proliferated cells, forms granulations and vegetations, fibrous thickening, and, finally, the deposition of lime salts. If the inflammation is severe the edge of the valves may become glued together, and so a funnel-shaped opening is formed, which is much narrower than the normal aortic orifice. In rare instances, instead of the valves being adherent and thickened, they are adherent and thinned, so that they appear atrophied. Such a condition is found at times in children and is thought to be congenital, but even in young children the cause may be rheumatism, and Sansom asserts that the condition may be due to rheumatic endocarditis in intrauterine life. Rheumatism may be considered the usual cause of aortic obstruction in children or in those who have not as yet reached advanced years.

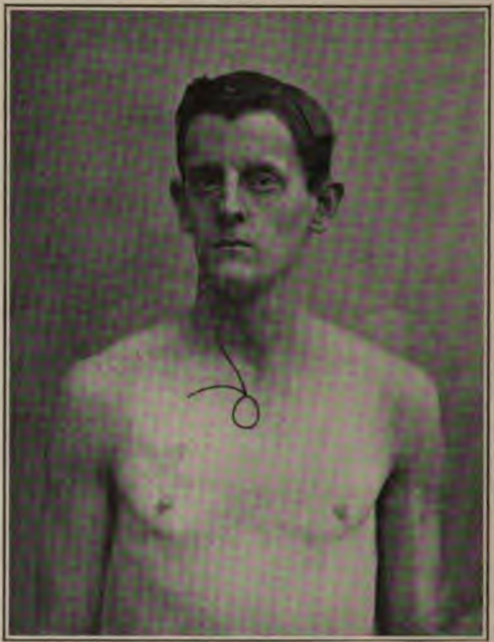
The cause of aortic obstruction is often not acute in character, as in the types just described, but chronic, being due to a gradual atheromatous change, which, having involved the aorta itself, spreads to the aortic valves, and causes a slowly progressive thickening and calcification of their tissues. This is the form of stenosis which is often of a very advanced type, so that the orifice may be but a small slit or chink through which the blood escapes.

From what has just been said, it is easy to understand how it is that obstruction to the flow of blood in the area of the aortic valve is exceedingly rare as a single lesion. The very nature of the morbid changes which take place in the tissues at this point renders the simultaneous existence of aortic obstruction and regurgitation probable, for the valves at the aortic orifice are either glued together as a result of rheumatic endocarditis, or, more commonly, are thickened by chronic endocarditis and calcareous deposits. In either instance they are not only in the way of the blood as it passes out of the ventricle, but they are incapable of preventing its regurgitation, since they are too thick and too stiff to approximate their edges. In other instances the presence of vegetations on the valves, in addition to these changes, adds to the impairment of their functional activity.

The secondary changes produced by aortic obstruction are chiefly connected with the left ventricle. Under favorable conditions this portion of the heart usually develops a satisfactory compensatory hypertrophy, the muscle fibres gaining in strength and size as the process of narrowing in the aortic area gradually progresses. As a consequence, it not rarely occurs that even an extreme degree of aortic obstruction is accompanied by such a complete compensatory hypertrophy that the presence of the lesion is only discovered at autopsy. It is interesting to note that the hypertrophy of aortic obstruction differs somewhat from that of aortic regurgitation in the fact that the ventricular walls increase in thickness without undergoing any great dilatation, whereas, in aortic regurgitation they both dilate and hypertrophy, causing eccentric hypertrophy.

So long as the compensatory hypertrophy of the left ventricle in aortic obstruction is adequate, practically no changes occur in the other parts of the heart. It is only when compensation ruptures that symptoms of impaired circulation ensue, or the mitral valves give way under the strain, and congestion of the left auricle and of the lungs develops. This may in time increase the labor of the right ventricle and lead to its hypertrophy.

FIG. 73



Showing area of greatest intensity and the direction of transmission into subclavian and carotid arteries of the aortic obstructive murmur.

Symptoms and Physical Signs.—Patients suffering from aortic obstruction rarely present or complain of any symptoms which are in any way characteristic of the lesion. At times a sense of constriction or oppression is felt over the aortic area, as it is in cases of aortitis or atheroma of the aorta, and as it is felt in some cases of true angina pectoris. When there is an associated aortic regurgitation, the symptoms are of that lesion. (See Aortic Regurgitation.)

The *physical signs* vary considerably with the type of patient examined and with the stage of the disease. In old men whose chest walls are thickened and rigid and whose lungs are often emphysematous, so that the edge of the left lung projects between the heart and the chest wall, it may not be possible to either see or feel an apex beat, even though the heart be considerably hypertrophied. On the other hand, if the patient be young, or the chest wall pliable, the apex beat may be seen and felt distinctly and forcibly, and it is usually a little below and a little outside the nipple because of the hypertrophy. If the action of the heart is forcible and the fingers are placed over the second right intercostal space, a distinct thrill can be felt. This thrill is in the nature of a vibration and is often transmitted down the sternum and even up into the carotids. This thrill felt in the area described is very characteristic of aortic obstruction, but it is also felt in aortic aneurysm.

The area of cardiac dulness on percussion is not materially enlarged, unless associated regurgitation has caused its well-known secondary cardiac changes.

Auscultation reveals a murmur which is loudest at the second right costal cartilage near the sternum.

This sound is transmitted in most cases into the vessels of the neck, and not infrequently it is heard over the sternum as low as the ensiform cartilage. It occurs with systole of the heart, and it is usually loud if compensation is preserved. Not only is it loud, but it is apt to be harsh and even musical, and it is long and blowing in character. The aortic second sound is usually absent.

When rupture of an aortic valve takes place the murmur is usually widely diffused, very loud and musical. I showed a patient to the College of Physicians of Philadelphia in 1902 that possessed a murmur capable of being heard when the ear was eighteen inches from the chest. It could be heard on top of his head, in his radial arteries, and if the stethoscope was placed so that his lips encircled it the murmur could be heard in his mouth. There was also a loud aortic regurgitant murmur in his case. The patient was a brakeman who suffered from sudden and severe dyspnœa and syncope on lifting a heavy weight, and who had a history of syphilis.

The pulse in aortic obstruction is small (*pulsus parvus*) because the heart cannot expel a large wave of blood through the narrow aortic opening. The wave rises gradually and then falls gradually, unlike the sharp upward jerk felt in aortic regurgitation.

Diagnosis.—Sufficient emphasis has already been placed on the fact that a systolic murmur at the second right costal cartilage does not mean, necessarily, aortic obstruction, but if the time of the murmur, the thrill, the peculiar pulse and atheromatous vessels are present in an old person the diagnosis is fairly certain. When the murmur is due to atheroma of the aorta alone the aortic second sound is usually sharp and clear instead of being impaired as it is in true obstruction. Aneurysm is excluded by the absence of the characteristic signs of that state. (See Aortic Aneurysm.)

Sewall asserts that an aortic stenotic murmur heard at the apex disappears on pressure of the stethoscope, and so separates itself from the systolic murmur of mitral regurgitation.

Prognosis.—The prognosis in a case of aortic stenosis is generally considered as more favorable than in any other valvular lesion, but in each individual case the physician must base his prophecy as to life upon the age of the patient, the state of his arteries, and the condition of the kidneys. The mere presence of far-advanced atheroma in aged persons who have an aortic stenotic lesion is not necessarily of evil import. As Clifford Allbutt well says, "We see well-to-do old ladies leading tranquil lives up to four-score years or more with systolic aortic murmurs of a quarter of a century's standing." On the other hand, in somewhat younger persons, who have more fibrous and less calcareous arterial changes, the prognosis is not so good, either because they are at a period of life when they are prone to resort to exercise and so strain the heart, or because there is a tendency to fibroid heart as well. Allbutt's view that "a person who in young or middle life begins to suffer overtly from the symptoms of aortic stenosis has but a few years to live" is certainly correct. (For treatment see end of article on Valvular Disease.)

Aortic Regurgitation. **Definition.**—Aortic regurgitation is often called "aortic insufficiency" or "aortic incompetency," and consists, as its names indicate, in a condition of the aortic leaflets, or of the aortic orifice, whereby the blood after being expelled by the contraction of the left ventricle into the aorta is permitted to return.

Etiology and Pathology.—By far the most common cause of this lesion is acute rheumatism, which causes the same changes in the valves at the aortic orifice as have already been described as taking place in the mitral leaflets, namely, distortion, retraction, stiffening, and the development of vegetations. As a rule, when rheumatism is the cause the mitral valves also suffer seriously, so that the aortic and mitral lesions coexist. A second common cause is aortitis, or atheroma of the aorta, which extends to the valves and causes sclerotic and degenerative changes which alter the position and functional ability of the aortic cusps. This atheroma may be due to mere senility, or to syphilis, gout, or even malarial infection. It is remarkable how many of these cases have a history of excessive toil, excessive venery, and excessive drinking, with the result that the bloodvessels and heart have had to stand strain, toxæmia, and infection. The cases of aortic regurgitation which are due to these causes suffer the greatest destruction of the valves, for their surfaces may ulcerate or the deposition of an excess of lime salts causes necrosis to such an extent that only stumps of the valves exist.

A third cause, which is much more rare, does not primarily involve the valves, but the aortic orifice. The aortic ring undergoes dilatation and as a result the valves cannot become approximated. In other words, the opening is too large for them to close it. This condition is met with in cases of aneurysm of the aorta. It is not by any means as frequent as dilatation of the mitral orifice, because the ring around the opening of the aorta is largely made up of fibrous and fibroelastic tissues, whereas that which supports the mitral orifice is largely muscular.

A fourth cause is rupture of an aortic leaflet as the result of violent strain. This accident rarely, if ever, occurs unless the valve has already been weakened by disease.

A fifth cause is the presence of vegetations on the aortic valves, developing in the course of the acute infections or chorea. Some of these lesions differ from those due to ordinary endocarditis in that they are not always permanent, but may entirely disappear.

A sixth cause is ulcerative endocarditis, in which great destruction of the valves may take place or abundant vegetations develop. This is usually due to the pneumococcus. (See Croupous Pneumonia.)

A seventh cause is congenital malformation, which is exceedingly rare. Indeed, aortic regurgitation due to this cause is more rare than are congenital defects themselves, for congenital defects in the valves may not be severe enough to permit leakage.

Finally (eighth) a functional relaxation of the aortic orifice occasionally is met with in which temporary regurgitation takes place for the same reasons as have been described elsewhere. I saw a case of this character while on duty at St. Clement's Hospital some fifteen years ago, in a young girl who had a loud, aortic regurgitant murmur and apparently a fusiform aneurysm of the innominate artery. At the autopsy the vessels seemed perfectly normal in size, but on testing them they were found to be unusually yielding and elastic.

The secondary effects of aortic regurgitation upon the left ventricle are most important and interesting. The left ventricle no sooner expels its contents into the aorta and begins to dilate in order to receive the blood from the auricle, than it also receives part of the blood it has just sent into the aorta by reason of the fact that the aortic valves permit regurgitation. The ventricle, therefore, contains not only the normal amount of blood from the auricle, but an additional quantity from the aorta, and so it becomes dilated to contain this excess and also undergoes hypertrophy in order to expel this excess into the aorta and empty itself. Any strain upon the heart increases the dilatation, and as a result we often see, particularly in those who live by manual labor, an extraordinary increase in the size of the heart, which is both greatly dilated and greatly hypertrophied, the so-called eccentric hypertrophy of aortic regurgitation resulting in the "ox heart" or *cor bovinum.*

The rapidity and degree of the hypertrophy is extraordinary in some cases. Sansom speaks of a case in which the heart was thought to have gained an ounce each week for four or five weeks, and Dulles has recorded a case in which the heart weighed forty-eight ounces. When the ventricle is much dilated the ring guarding the mitral orifice may yield and insufficiency of the mitral valves ensue and thus cause dilatation of the left auricle, congestion of the lungs, and hypertrophy of the right side of the heart. In some cases of aortic insufficiency, when the cause is atheroma, the pathological process of fibrosis and calcification gradually extends to the tissues around the opening of the coronary arteries, which are then unable to properly supply the heart with blood, or the coronary arteries themselves become atheromatous. Under such conditions compensatory hypertrophy may never be established, or if established rapidly fails, is ruptured, and death ensues.

Symptoms and Physical Signs.—The symptoms of aortic regurgitation are more characteristic than are those of any other form of valvular disease, and are more constantly met with in such cases, although compensation may be adequate in many cases for a time at least. *Dizziness* and *partial syncope* often appear on suddenly sitting up or on standing up, and at times are present even when the patient lies down. *Palpitation* and *dyspnœa* on exertion are pressing symptoms if the heart is feeble, and even when compensation is adequate *pain* in the region of the heart is often a severe symptom, being radiated into the arms or into the neck. At this time the attacks may be identical with those of true angina pectoris. *Sudden death* occurs more frequently in this form of valvular disease than in any other.

When compensatory hypertrophy is lost the patient is forced to sleep sitting erect or nearly erect in an easy chair; he is usually very pallid, but at times cyanotic. Cough and pulmonary complications do not ensue until the mitral valves give way.

When the cardiac failure is well marked, and it is evident that death cannot be many days away, distressing *mental symptoms* often appear. Hallucinations are pressing and even an active maniacal delirium may develop. At times the patient becomes suicidal in his insanity. In some cases these mental disturbances are due to disordered and inadequate cerebral blood supply, and at times they are due to a complicating nephritis arising as a late lesion of the general breakdown.

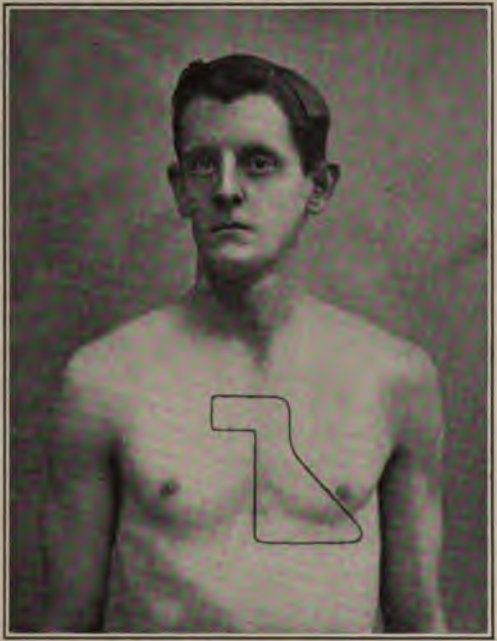
PHYSICAL SIGNS.—The physical signs are characteristic when well developed. On *inspection* the carotid arteries are seen to pulsate markedly, and even the head may be moved by the impulse transmitted to it by the heart. The thorax, in the præcordium and neighboring parts, heaves with each pulsation of the heart and is often bulging, owing to the cardiac hypertrophy and dilatation. An ocular examination of all the superficial arteries will show a characteristic throbbing or jerking, and if a glass slide be lightly pressed against the lower lip capillary pulsation is readily seen, in that the color of the mucous membrane rises and falls with the movements of the heart. Such capillary pulsation, often called "Quincke's pulse," can be seen under the thumb-nail when it is gently pressed upon, and in the red line produced on the skin of the forehead by drawing the end of a pencil over this part. As the arteries are often elongated, and therefore more tortuous than in health, the impulse of the wave of blood which tends to straighten the curves makes the vessels move laterally as they beat, and this increases the pumping effect which is produced by the so-called "Corrigan pulse."

The apex beat is always far below and far outside the nipple because of the dilatation and hypertrophy of the left ventricle, and also because of the elongation of the aortic arch. On *palpating* the heart the impulse on systole is powerful and diffuse except when compensation is ruptured, when it is feeble.

The *pulse* when felt by the finger-tips or when recorded by the sphygmograph feels as it looks on inspecting the arteries. With each systole of the heart the almost empty artery suddenly gives to the finger a short, sharp impulse, which equally suddenly disappears, the vessel being in an instant apparently as empty as before. The hypertrophied and dilated heart expels a large wave of blood into the aorta, of which a part at once falls back into the ventricle, so that what might be called the tail of the pulse is lost. This is the so-called "Corrigan pulse," sometimes called the "water-hammer pulse," or "trip-hammer pulse." It can be emphasized in the radial arteries by raising the hand above the head. This pulse is usually regular as to rhythm and force. If it becomes constantly irregular, it is a sign of grave cardiac failure.

On *percussion* the area of cardiac dulness is found to be greatly enlarged, so that it extends far over to the anterior axillary line in some instances, and three inches below the nipple. Dulness due to the enlarged heart is also found even as far as the right edge of the sternum.

FIG. 74



Showing the area in which the murmur of aortic regurgitation can be most clearly heard.

Auscultation.—Auscultation reveals a diastolic murmur, due to the return of the blood from the aorta after the systole has expelled it into the vessel, and while the ventricle is once more opening for a new supply from the auricle. This murmur is heard in its greatest intensity at the fourth left intercostal space, or at the second right costal cartilage in the so-called aortic area. It is heard at the fourth left intercostal space because the reflux of blood carries the murmur back into the ventricle, and because the aortic opening as a matter of fact is nearer this area than the second right intercostal space (Fig. 74). In still other cases the diastolic murmur may be very clearly heard at the cardiac apex as well as at the second right intercostal area, although between the two no murmur at all, or only a very faint sound, can be discovered. The explanation of this is that the right ventricle occupies the anterior surface of the heart, except at the extreme left edge, where the left ventricle protrudes and where the apex comes closely in contact with the chest wall.

The murmur of aortic regurgitation is not widely transmitted, but is limited, as a rule, to the area described, although when it is very loud it may be heard everywhere in the chest. The quality of this murmur is blowing or purring. There is no accentuation of the pulmonary second sound at the third left costal cartilage, unless there is an associated mitral regurgitation or stenosis. The first sound of the heart is loud or prolonged owing to the large amount of blood which has to be expelled at systole. In some cases a diastolic sound near the apex may mislead the physician into a diagnosis of mitral obstruction, but it is in reality the so-called "Flint's murmur." (See Mitral Stenosis.)

If the stethoscope is placed over any one of the large superficial arteries there is sometimes heard, in cases of aortic regurgitation, a sharp systolic, "pistol-shot" sound, which is said to be due to sudden filling of the vessel. This sound is not transmitted from the heart, but is local in origin. It is a modification of the arterial sound which can be elicited in most persons with a strong pulse, if the artery is occluded by pressure with a stethoscope. Very rarely a diastolic arterial sound can be heard, the so-called "Duroziez sign," which is thought to be due to a transmitted cardiac sound.

The question as to whether a diastolic aortic murmur can be present without disease of the aortic valves has been much discussed, particularly of late, by Cabot and Locke, of Boston, and Gibson, of Edinburgh. As is well known, the aortic ring is so firm that it seems impossible that it can yield as do the rings of the pulmonary and mitral orifices. Cabot and Locke believe that diastolic aortic murmurs are not uncommon in connection with diffuse or localized dilatation of the aorta, but in view of the evidence presented by Gibson, we must, I think, agree with him that diastolic murmurs, without valvular lesions, are rare at the aortic orifices, and that when they occur they are due to defective approximation of the different aortic cusps rather than to yielding of the aortic ring.

Diagnosis.—The symptoms and physical signs of aortic regurgitation having been described, it remains to separate them from those conditions which possess a resemblance. Occasionally in some persons a diastolic cardiopulmonary murmur is heard, but it disappears with change in posture and is dissipated or is accentuated by forced expiration and inspiration. At the level of the second and third rib there is heard at times a hæmic murmur or hum due to anæmia, but this is systolic in time. Cabot has, however, reported cases in which diastolic murmurs were hæmic and due to anæmia.

Finally, it is to be recalled that the aortic regurgitation murmur is often inconstant, and if not heard at one examination may be at another, or when the patient exercises or changes his posture.

Prognosis.—Facts in regard to the relative fatality of aortic regurgitation have already been given. Allbutt says ten years constitute a long time of life in any case of aortic regurgitation. It is not only a serious lesion, but it is the valvular lesion above all others which produces sudden death; but death in aortic regurgitation is not always sudden. A majority of cases gradually "play out" with dyspnœa, dropsy, and cardiac distress. Much depends on the age of the patient, his previous habits, and the cause of the disease. In a young adult with a good history, and in whom the lesion has followed rheumatism without great injury to other parts, compensatory hypertrophy may carry him along for many years unless he ruptures it by severe strain, or it is dissipated by illness. When the lesion comes on as the result of atheroma it is more serious because it is evidence of general cardiovascular degeneration, and the coronary arteries, through which the heart is chiefly nourished, no sooner share in the degenerative change than feebleness of the heart ensues. If perchance the kidneys share in the cardiocapillary fibrosis, the outlook is all the more grave. When fast living and debauchery are factors in the case, and an old syphilitic infection is impairing the myocardium, the outlook is worse still. (For treatment see end of article on Valvular Disease.)

Tricuspid Regurgitation. **Definition.**—Tricuspid regurgitation, or insufficiency, is a condition in which the blood flows backward into the right auricle from the right ventricle, upon the contraction of the latter.

Etiology and Pathology.—This is a rare lesion and is due to two chief causes. It occurs most commonly as a result of disease on the left side of the heart, whereby the blood is dammed back into the lung and so, by preventing free pulmonary circulation, an undue strain is thrown on the tricuspid leaflets. In other instances the primary obstruction exists in the lungs, as in pulmonary emphysema, fibroid phthisis, and bronchiectasis.

As indicating the relative frequency of these causes, Newton Pitt reports from Guy's Hospital that out of 405 cases of tricuspid regurgitation examined at autopsy, in a period of twenty-five years, 200 cases were due to left-sided failure with valvular disease. Of this number 64 were cases of mitral regurgitation with mitral endocarditis or adherent pericardium, 66 were due to mitral stenosis, 61 to mitral with associated aortic disease, and 9 had valvular lesions not named. In 71 cases the tricuspid condition was due to left-sided failure without valvular disease, in 56 cases the cause lay in muscular failure of the whole heart, and in 55 the lesion was not left-sided, but was right-sided alone. Seven cases were due to disease of the pulmonary valves, of which 5 were stenosis and 2 pulmonary regurgitation. In 4 cases no cause could be found, and in 12 the reports as to the exact state of the heart were too imperfect for analysis.

The valves in these cases are usually healthy and fail to close the right auriculoventricular opening because the orifice is enlarged as a result of dilatation of the ventricle.

The second cause of this lesion lies in the heart itself, that is, an endocarditis involving the right side of the heart. This is generally stated to be very rare, although in fetal life endocarditis is more commonly on the right side of the heart than the left. Bramwell, however, combats the statement that acute endocarditis rarely affects the tricuspid valves, and believes that endocarditis of the right heart often exists and is overlooked. Out of 28 cases of recent simple endocarditis he found disease of the tricuspid valves in 14, or 50 per cent. Nevertheless, the lesions are not so pronounced as those due to this cause on the left side of the heart. In ulcerative endocarditis Osler found the tricuspid valves affected in 19 cases out of 238 instances of that disease.

A case in which tricuspid disease was discovered during intrauterine life is reported by Peter, of Paris!

Symptoms and Physical Signs.—When the cause exists in the left side of the heart the symptoms are those naturally arising in cases of pulmonary congestion and engorgement. A *low-grade bronchitis*, with some *dulness at the bases* of the lungs posteriorly from hypostatic congestion, with *cough* and occasionally blood-stained sputum, is discoverable. The *jugular veins* become distended and *pulsate*, the *liver becomes enlarged* and may pulsate, and there is marked *cyanosis*. It is a noteworthy fact that the presence of jugular pulsation, while a sign of well-developed regurgitation, is not by any means as grave a sign as is its absence in the presence of other evidences of cardiac embarrassment, because if the right ventricle is strong it may drive the regurgitating blood with sufficient force to cause jugular pulsation, whereas if it be weak no jugular pulsation can ensue. On the other hand, the presence of jugular pulsation shows that the regurgitation is a grave one, because it does not occur until a considerable quantity of blood flows into the auricle and distends the veins sufficiently to interfere with the valves, so that they cannot prevent auricular regurgitation as they do in health.

A distinct impulse in the upper epigastrium is often present.

The *pulsation of the liver* is best discovered, if not seen, by placing the finger-tips or hand against the floating ribs at the side, and the other hand near the ensiform cartilage, and then exerting gentle pressure upon the liver from both directions. Care must be taken that a directly transmitted impulse from a hypertrophied heart is not taken for true expansile pulsation of the liver.

On *percussion*, increase in the area of cardiac dulness to the right of the sternum is demonstrable.

On *auscultation* at the apex a soft systolic murmur, or purr, can be heard, which is found to be loudest at about the fifth interspace, to the right of the sternum or at the base of the ensiform cartilage, and which may be in a few cases transmitted to the right axillary area. The murmur of tricuspid regurgitation has been said to resemble the sounds made by a small jet of escaping steam, and it may be singing or musical. Duroziez says that venous blood "sings" more than arterial blood.

Diagnosis.—True tricuspid regurgitation must be differentiated from regurgitation at this orifice, which is temporary and not constant. These valves not rarely give way from severe strain, as in athletes during exertion. They act in this way as a "safety-valve" to relieve undue pressure, and as soon as the strain ceases the heart gradually returns to its normal size and the murmur disappears. So, too, a similar murmur may develop in cases of profound asthenia resulting from prolonged fevers, particularly if the heart is strained by the patient attempting to do too much. Such a murmur may or may not pass away with rest.

Prognosis.—The prognosis depends entirely upon the exciting cause, the age of the patient, and his general state and manner of life. In those who have to do manual labor the prognosis is usually bad.

Tricuspid Stenosis.—This condition sometimes arises as the result of an attack of endocarditis involving both sides of the heart, and is usually associated with mitral stenosis. In other instances it is congenital. In the vast majority of cases it is found as an associated lesion, and it is exceedingly rare.

In 1899 Newton Pitt collected 87 cases of tricuspid stenosis from the post-mortem records of Guy's Hospital extending over a period of twenty-six years, and comprising a total of 12,000 autopsies. Leudet, in his Paris thesis on tricuspid stenosis, gives the following figures, based on 114 post-mortem examinations which were collected from various sources, and which include the cases collected by Fenwick:

Stenosis of the tricuspid valve alone	11
Stenosis of the tricuspid and of the orifice of the pulmonary artery	3
Stenosis of the tricuspid, mitral, and pulmonary	1
Stenosis of the tricuspid, mitral, and aortic	21
Stenosis of the tricuspid and mitral	78

F. W. Griffith, of Leeds, has examined Leudet's tabulation of cases and has shown that probably only two of them were unassociated with lesions of the other valves. These figures show the rarity of uncomplicated tricuspid stenosis.

Diagnosis.—The diagnosis of the lesion during life is usually very difficult, but it can be made. The murmur, if present, is presystolic in time and has its greatest intensity at the fifth right interspace near the sternum. As already stated, this murmur is usually associated with that of mitral stenosis, and its existence may be completely masked by the presystolic murmur at the mitral orifice. Additional physical signs of tricuspid stenosis are distention of the jugular veins without pulsation, or with very slight pulsation due to the feeble auricular regurgitant impulse.

As dropsy is a late symptom of severe mitral stenosis, the early development of dropsy in a case with a presystolic murmur which is not aortic strongly indicates tricuspid obstruction.

Disease of the Pulmonary Valves.—Lesions of the pulmonary valves are so rarely met with that many practitioners of large experience have never seen a case presenting them. When actual lesions occur they are nearly always congenital, and the usual lesion is that of pulmonary stenosis or obstruction, for pulmonary regurgitation is the rarest of cardiac lesions. On the other hand, there is no area at which we listen, for the purpose of determining the state of the heart valves, in which murmurs are so constantly found as the so-called pulmonary area, at the second and third left intercostal space. These murmurs are not due to actual disease of the pulmonary valves or of the pulmonary artery, but they arise from causes of a non-organic character and are usually systolic in point of time. These functional murmurs may be due to one of several causes, as, for example, anæmia and chlorosis, producing the so-called hæmic murmur. They also occur in pregnant women and in women after childbirth. In other instances they are present as a sign of Graves' disease or of nervous tachycardia, and in still others they arise from some abnormal position of the heart, caused by conditions which alter the relationship of the heart muscle to its great vessels. These conditions all produce a pulmonary murmur which may be considered as within the area of the pulmonary artery or near its valves. In addition we sometimes hear at this point a so-called, cardiopulmonary murmur, which is supposed to be due to the effect of the bloodvessel upon the lung, or *vice versa,* in that the murmur occurs during forced inspiration, or expiration. This murmur is not rarely seen at this point in early tuberculosis of the apex of the left lung.

PULMONARY STENOSIS.—The systolic murmur which is due to actual organic disease at the pulmonary valve is due to stenosis, as already stated, and is commonly due to gluing together of the cusps of the pulmonary valves in antenatal life. This murmur is more harsh than the soft purring murmurs of the functional type just described. The patient usually has a history of having been cyanotic all his life, with dyspnœa on the slightest exertion. Percussion and palpation will usually reveal a distinct increase of cardiac dulness to the right from hypertrophy of the right ventricle. Palpation also will reveal a distinct systolic thrill over the area of the pulmonary valves.

The points by which the true systolic murmur is to be separated from the functional murmurs already described are the absence of systolic thrill in the case of the functional murmurs, the harshness of the true murmur, and the hypertrophy of the right ventricle in cases of actual pulmonary disease. From aneurysm of the descending portion of the aortic arch the true pulmonary murmur is separated by the fact that in such a case the hypertrophy chiefly involves the left side of the heart, by the additional fact that there is a bruit, not a murmur, and by the presence of a bruit posteriorly on the left side between the vertebræ and scapula. There are also pressure symptoms in aneurysm in many cases. Finally, if it be aneurysm, percussion of the third left costal cartilage will reveal dulness, which is absent in pulmonary stenosis. The murmur of aortic stenosis is heard louder at the second right costal cartilage than at the second or third left cartilage, and is transmitted into the carotids, but the pulmonary murmur is not.

PULMONARY REGURGITATION, the rarest of all valvular lesions, is usually fetal in origin, but cases have been recorded in which it has arisen as a result of ulcerative endocarditis. The murmur due to this cause is, of course, diastolic, and is produced by the blood falling back from the pulmonary artery into the right ventricle. It is to be separated from aortic regurgitation by the fact that it is heard best to the left of the sternum instead of to the right, and by the absence of the Corrigan pulse of aortic disease. There are signs of dilatation and hypertrophy of the right ventricle, and the pulmonary second sound may be accentuated. It is by no means uncommon for the existence of pulmonary regurgitation to be unsuspected until autopsy.

Treatment of Chronic Valvular Disease.—It is of vital importance that the physician remember the fact that the mere presence of a valvular lesion in the heart does not indicate drug treatment. On the contrary, much harm is frequently done by the administration of cardiac stimulants to patients who are found to possess a valvular lesion, with the result that natural compensation is disturbed and cardiac symptoms may be noted by the patient for the first time in his history. It is only when the patient presents symptoms which indicate failure of cardiac function that the physician should think of administering remedies which have a direct influence upon the heart. In other words, when compensation is complete, no cardiac treatment is indicated, but when it is ruptured therapeutic measures should be instituted. I have again and again seen patients who have presented some ailment involving other organs in the body than the heart, in whom the physician, on examination, found a mitral regurgitant murmur, and immediately proceeded to administer digitalis, forgetting that the presence of a murmur in the absence of evidences of circulatory failure is not an indication for the use of the drug.

Another important point to be borne in mind when the physician is called upon to treat a case in which compensation is failing is that, far and above all drugs in value, is rest for the patient; not only rest of the body, but rest of the mind. The heart is an organ which, of course, cannot have complete rest at any time; but its work can be diminished one-half if the patient can be made to take no exercise, and if he will carefully abstain from business worries and cares. It is a remarkable fact that disease of the coronary arteries and of the heart muscle is more common in brain-workers than in those who gain their living by manual toil, and this is an indication of the fact that mental labor throws a severe strain upon the heart. Careful observation will promptly prove that patients who are not benefited by digitalis and other cardiac stimulants, when suffering from ruptured compensation, will at once improve if rest is insisted upon; for, manifestly, the rupture of compensation is largely the result of cardiac fatigue, and this fatigue cannot be put aside by the mere administration of stimulants. In order that the juices of the body may be kept moving, it is useful in these cases, when they are made to rest in bed, to use more or less vigorous massage daily, its vigor depending upon the strength of the individual. If the massage is too vigorous, it may produce very considerable fatigue, and it should not be employed to this extent.

In all cases of ruptured compensation the patient should be warned of the danger of sudden, severe effort, since, even if death does not ensue under these circumstances, the heart may be so dilated or fatigued that irreparable damage is done to it.

Digitalis is without doubt *facile princeps* the best of cardiac stimulants. I have proved that it actually increases the muscular development of the heart, and the manner in which it acts results in an increased nerve and blood supply to this viscus which is not equalled by the results obtained from the administration of any other remedy. Digitalis is, in a large number of cases, given in too large doses. Not infrequently as much as 10, or even 20 minims of the tincture are given three times a day, with the result that in the course of a few days the heart is overstimulated by the drug; its cumulative effect is produced, and instead of doing good it may do serious harm.

I have known of cases in which the use of full doses of digitalis, persisted in for a considerable period of time, have resulted in the sudden death of the individual.

When digitalis is given in overdoses it produces a curious irregularity of force and rhythm in the heart, with imperfect systole, followed by wide diastole, and this causes a hobbling pulse. The urine may also be reduced in amount instead of increased, as it should be, under the influence of the remedy.

It has been my experience that if the patient is made to rest, small doses of digitalis produce satisfactory results, not more than 5 drops of the tincture being given three times a day. The only conditions in which I think that the use of large doses is justified are when the condition of the heart is found to be exceedingly feeble, and the patient's condition so critical that immediate stimulation is necessary; and again, when because of idiosyncrasy or other cause the heart is found not to respond to smaller doses. In the first class of cases it has been my experience that it is better to overcome pressing cardiac weakness by more rapidly acting and diffusible stimulants such as Hoffmann's anodyne, strychnine, and caffeine for the first twenty-four of forty-eight hours until the digitalis has a chance to act; for it must always be remembered that digitalis is a drug which produces its effects very slowly, and maintains these effects for some time after its use has been stopped. When large doses are given, it has been my experience that they may be decreased to about one-half or one-quarter the original quantity at the end of a few days, and their effects maintained by the use of smaller quantities. I do not think that I am exaggerating the case when I state that digitalis, through its abuse, does almost as much harm as it does good. (See my *Text-book of Therapeutics*, article "Digitalis.")

The fluid extract of digitalis may be given in the dose of from ½ to 5 minims, according to the needs of the case. One-half to 1 minim every eight hours is usually sufficient. A physiologically tested preparation should always be employed.

When dropsy is present it has been held that the infusion of digitalis is the best preparation on the ground that it is more diuretic. But any slight increase in diuretic power is, I think, counterbalanced by the fact that it is much more apt to disorder the stomach, which is in a disturbed condition in any case of grave disease, and particularly so in heart disease.

When digitalis has been given in full doses to a patient suffering from ruptured compensation, he should be warned against getting up suddenly, and particularly against evacuating the bladder when in a standing position, as dangerous syncope may occur under these circumstances.

Before deciding upon the administration of any cardiac stimulant in a case with ruptured compensation, the physician should make a careful study of the state of the bloodvessels, and if the arterial tension is higher than normal he should remember that the use of nitroglycerin to reduce this tension, and thereby diminish the work of the heart, is a more important therapeutic procedure than the administration of a stimulant which simply urges the heart to do more work in the face of vascular obstruction. In such a case the fact that digitalis stimulates the vasomotor system and raises arterial tension should also be remembered. Nitroglycerin can often be given to advantage to prevent this arterial effect of the drug. Strophanthus, which is a much less powerful stimulant than digitalis, possesses the advantage that it does not raise arterial pressure by stimulating the vasomotor system, and can often be given in the dose of 5 to 10 minims of the tincture every eight hours with advantage. Large doses of strophanthus are prone to produce an irritative diarrhœa.

There is a host of drugs which have been recommended for ruptured compensation, but none of them approach these three in value, and often when these fail the failure is due to a mistake in the dose rather than to a fault of the drug. Should there be any tendency to hypostatic congestion of the lungs or pulmonary œdema, digitalis may be freely given, and strychnine and atropine administered as vasomotor stimulants. Two or three dry cups over the base of each lung are also useful under these circumstances, and sometimes a sharp purgative may do good if dropsy is present; but the possibility of the purge weakening the patient must always be borne in mind.

Attacks of acute cardiac failure are to be combated by Hoffmann's anodyne in the dose of 1 or 2 drachms every hour or two, or aromatic spirit of ammonia in the dose of ½ to 1 drachm. If, associated with the cardiac failure, there is high arterial tension, the nitroglycerin should be used hypodermically in the dose of $\frac{1}{150}$ to $\frac{1}{100}$ of a grain, and repeated every half-hour until the tension is lowered.

When marked dropsy is present, the use of magnesium sulphate in concentrated solution, a heaping teaspoonful to one-half glass of water taken before breakfast, will often do good by removing large quantities of fluid from the patient's body. A very useful remedy in cardiac dropsy, both by reason of its action on the heart and because of its diuretic effect, is apocynum cannabinum given in the dose of from 5 to 30 minims of the tincture twice or thrice a day until it produces slight purgation. Care should be taken that a fluid extract of real apocynum cannabinum is obtained. Much of that on the market is apocynum androsimæfolium, which has no such therapeutic properties, but which so closely resembles apocynum cannabinum that its leaves are often unintentionally substituted for the true drug.

Another very useful prescription in cardiac dropsy is a tablet composed of

Extract of sourwood leaves	2 grains.
Extract of elder flowers	2 grains.
Extract of squill	¼ grain.

Take one or two tablets three times a day.

For generations physicians have been in the habit of employing a pill composed of—

Powdered squill	1 grain;
Powdered digitalis leaves	1 grain;
Calomel	1 grain;

three times a day in the treatment of cardiac dropsy, for its stimulant and diuretic effect. If free diuresis is not produced by this pill at the end of the third day it should be stopped, and a saline purgative administered to sweep out the calomel from the alimentary canal.

In many cases of cardiac disease, particularly if the presence of anæmia is marked, iron and arsenic are indicated, both on general principles and because we cannot expect to improve the nutrition of the heart when it is supplied by impoverished blood.

It is important to remember that in nearly every case of failing compensation there is a certain amount of hepatic congestion. This is best relieved by the use of 5 or 10 grains of blue mass given every week or ten days. Frequently digitalis and other stimulants will act much better after the liver has been unloaded by the mercury than they will when this gland is congested.

When the rupture of compensation has resulted in dyspnœa and anxiety, morphine proves itself a most valuable remedy. It should not be given except in cases in which there is dire need of rest, since if used too frequently it loses its good effects, and frequently causes constipation and disorder of digestion. On the other hand, the quiet and rest produced by ⅛ to ¼ of a grain of morphine will often cause marvellous improvement. It has seemed to me that it is most satisfactory in mitral lesions. If dropsy is present, it cannot be given hypodermically because it will not be absorbed.

When there is great engorgement of the jugular veins, and manifest distention of the right side of the heart with pulmonary congestion, venesection to the extent of about 8 ounces to a pint, according to the size of the individual, is often very valuable as a means of relief. It also is a remedy which is to be reserved for somewhat desperate conditions, as manifestly it is not proper to bleed frequently.

It is generally considered that digitalis does less good in cases of aortic regurgitation than in other valvular lesions, and I believe this opinion is correct. Some have taught that it is contraindicated in aortic regurgitation. While this may be the general rule, every now and then we meet with cases in which cautious use of the drug in this state produces excellent results, provided compensation is ruptured.

In some instances when the heart's action is very irregular and excitable an ice-bag applied over the præcordium is advantageous.

When the patient has had syphilis, chronic rheumatism, or gout, or manifests evidences of arterial capillary fibrosis, iodide of sodium or iodide of potassium in the dose of 10 or 15 grains a day is advantageous.

The diet in cases of valvular disease should be simple, but nutritious. The patient had better eat four or five small meals a day than two or three hearty ones, and the greatest possible care should be taken that foods which are prone to produce gaseous distention of the stomach and bowels are avoided. Fatty substances are very apt to produce such symptoms, as are also the starches, when they remain in the stomach undigested for a considerable period of time. This, however, can be put aside in the case of the starches by the use of taka-diastase or pancreatin and by the use of powdered capsicum, either in pill or upon the food, for the purpose of stimulating the gastric mucous membrane, which is often atonic or catarrhal as a result of the impaired hepatic circulation.

NEUROSES OF THE HEART.

Definition.—By neuroses of the heart it is meant to include a number of conditions widely separated in their actual causes, but depending upon a disorder of the nerve supply of this organ, so that, without there being necessarily present any grave organic lesions, its functional activity is impaired or perverted.

Palpitation.—Palpitation of the heart may be due to any of the valvular or other lesions that cause the heart to beat rapidly or irregularly when the individual takes exercise. In other cases it arises from the accumulation of gas in the stomach or colon, which by its pressure causes cardiac disturbance. In other instances it is due to intense nervous erethism, and in still others it arises from the excessive use of tobacco or of coffee or tea. So, too, sudden nervous shock may cause repeated attacks of this character. Probably the most frequent cause of cardiac palpitation is not connected with the nerves supplying the heart, but is due to irregularities in the nervous control of the vasomotor system, whereby the tension of the vessels is relaxed and as a result the heart beats rapidly because the ordinary pressure in the arteries is suddenly removed. It is remarkable how cases of palpitation get well if attention is paid to the state of the bloodvessels rather than the heart. The so-called "irritable heart of soldiers," first described by J. M. Da Costa, is due in all probability to this cause as well as to the imperfect action of the vagus. Associated with the disordered cardiac action it will often be found that the peripheral capillaries suddenly become dilated, so that the blood can flow more rapidly than normal into them. Profuse diuresis, in which the urine is found to be pale and clear, shows that a similar relaxation of the renal vessels has ensued. These symptoms often cause the patient great alarm and bring him at once to the physician with the statement that heart disease is feared. It is usually the case that a patient who says he has heart disease has only a neurosis, unless some physician has found a real lesion and told him of its existence.

Tachycardia.—Many cases of neurosis are closely allied, if not identical, with that condition of rapid heart called tachycardia. This may be due to vasomotor palsy, to a deficient action of the vagus nerves, or to some central nervous lesion. (The tachycardia of exophthalmic goitre is not considered here.)

Such attacks occur in young hysterical women and are called "pseudo-angina," because a sense of cardiac expansion is often present with the rapid beating of the heart. They also appear in women near the menopause, and in men who have been guilty of excessive sexual abuse. A few cases in men are apparently due to some organic nervous lesion. The pulse may rise as high as 220. The tachycardia may be paroxysmal or continuous. In a case under my care in 1890, a woman who had seen her husband and sons drowned in the great Johnstown flood, and had been swept from the roof of her floating cottage, presented a pulse rate which was uncountable, it was so fast. This persisted for months and was present two years after the catastrophe.

Rapid, feeble heart sounds, in which the first and second sounds appear alike, are met with in prolonged exhausting fevers such as severe typhoid fever, and to this state is given the name "fetal heart sounds" or "embryocardia."

Bradycardia.—Great slowness of the heart's action (bradycardia) is caused, to some extent at least, by conditions which are the antithesis of those that cause tachycardia. A sudden or persistent rise of arterial pressure may cause a very slow pulse, as the heart endeavors to force blood through tightly contracted vessels. This may be called vascular bradycardia. Again, it occurs as the result of irritation of the vagus nerves by poisons such as digitalis and opium, or in chronic lead poisoning, or again in cases of jaundice when the slowing is due to the biliary salts in the blood. It is also met with in cases of apoplexy, cerebral tumor, and in the coma following epilepsy.

A pulse below 60, or even as low as 40, is sometimes felt after a woman has given birth to a child.

Occasionally cases are met with in which the bradycardia becomes extraordinary. The late D. W. Prentiss reported to the Association of American Physicians in 1889, 1890, and 1891 the case of a man whose pulse at times fell to 11 per minute and rarely rose over 40 per minute for two years. In this case no very distinct morbid lesions were found at autopsy, although the patient died suddenly in an attack. (See Stokes-Adams Disease.

Arhythmia.—There are other cases met with in which cardiac arhythmia—that is, irregularity as to the speed and the force of the heart beats—occurs. In many cases this is due to a neurosis of the vagus. It is very commonly met with in persons who have taken excessive doses of digitalis, and it is also a common symptom in mitral stenosis with ruptured compensation. The so-called gallop rhythm may appear in these cases. (See Mitral Stenosis.) Occasionally arhythmia is a notable symptom in cases of myocardial degeneration, but cases are recorded in which arhythmia has lasted for several decades in seemingly healthy men.

Treatment of Cardiac Neuroses.—In the treatment of the various cardiac neuroses it is essential that the physician shall first determine what portion of the circulatory system is chiefly affected by disordered innervation. Not infrequently, however, it will be found that both the heart and vasomotor system are out of order, and therefore the treatment will have to be devoted to regulating the nerve supply of the functions of both of these important vascular areas.

When it is believed that attacks of tachycardia have their origin in a condition in which the pneumogastric nerve fails to control the action of the heart, digitalis is, of course, a valuable remedy in that it exercises a powerful stimulating influence upon the pneumogastric nerves. In some instances, however, the action of the heart is already sufficiently vigorous and the administration of digitalis, while stimulating the pneumogastric nerve, also stimulates the heart to such an excessive degree that its action becomes too violent. Under these circumstances it is well to combine with the digitalis a little aconite, which drug also stimulates the pneumogastric nerve, and thereby aids the digitalis in controlling the heart, and at the same time combats the influence of the digitalis upon the heart muscle itself, thereby preventing overstimulation. A prescription made up as follows will often be of advantage, the quantities of the ingredients being varied to suit the needs of the individual case:

℞.—Tinct. digitalis (physiologically tested)	f ʒ j (4.0).
Tinct. aconiti	f ʒ ss (2.0).
Tinct. belladonnæ	f ʒ ij (8.0).
Tinct. cardamom. comp. . . . . q. s. ad	f ℥ iij.

A teaspoonful to a dessertspoonful three or four times a day.

The efficiency of this prescription may be increased by the application of a belladonna plaster over the heart.

In those cases in which the cardiac irregularity depends chiefly upon alternate spasm and relaxation of the bloodvessels, so that they are at one moment offering too much resistance and at another too little resistance to the flow of blood, much benefit can be produced by hydrotherapeutic measures, such as directing that the patient shall take an alternate hot and cold sponging in the morning on getting up; first, drenching himself with a sponge dipped in as hot water as he can bear, and next drenching himself with a sponge dipped in cold water. In this way he does not become chilled, but the elasticity and tone of the bloodvessels is much improved. The prescription just recommended is also beneficial in many of these cases, and not infrequently moderate doses of tincture of nux vomica are advantageous. When the disorder depends upon the excessive use of tobacco, this drug must, of course, be prohibited; and if the patient is overworked, and suffering from nervous strain, he must be sent away on a vacation; or, if his condition is grave, be given a "rest cure." When a disordered circulation is associated with pain in the neighborhood of the heart, small doses of antipyrin, 2 to 3 grains, are often useful; and if the tension is high, nitroglycerin should be given.

ANGINA PECTORIS.

Definition.—By angina pectoris is meant a condition in which a patient, usually in or past middle life, is seized by a severe, agonizing pain in the cardiac area, which extends in most cases down the left arm even to the wrist, and suffers from intense mental anxiety and a sense of impending dissolution. It is important that pseudoangina be not confused with it.

Etiology and Pathology.—The cause of true angina pectoris is usually atheromatous change in the coronary arteries, although this is by no means always the case. Thus, Potain found stenosis of both coronary arteries in 20 out of 45 cases at autopsy, and Huchard in 38 out of 70 cases. It is distinctly a disease of the brain-worker rather than of him who gains his bread by manual labor. The laborer and artisan present to us very commonly the most surprising degenerative changes in their arteries in the way of calcification of their radials and temporals, but they rarely have true angina. On the other hand, the man who is subjected to nervous strain rarely shows extraordinary calcareous changes, but he is the unfortunate victim of this terrible malady. Physicians are peculiarly prone to it. The number of deaths among the leaders of the profession in Philadelphia during the last decade from this cause is extraordinary. Mental strain with a sedentary life are, therefore, causes. Gout, syphilis, and renal disease are also causes. The disease affects men much oftener than women. Out of 65 cases collected by me only 4 occurred in women, and out of 290 cases collected by Forbes, Huchard, and Lartigue only 47 occurred in women. Aside from the narrowed and thickened coronary arteries and the fibroid changes in the heart which result from their state, there are no characteristic lesions of true angina pectoris. Among the exciting causes of an attack are to be named nervous wear and tear, anger, or muscular exertion, particularly if it be made in the face of a cold wind, which contracts the capillaries and so increases still more the labor of the heart. So, too, errors in diet, by causing gastric disorder, may reflexly cause an attack.

Sir Douglas Powell and Merklen give the age incidence for true angina at twenty to forty years. This is probably too young. The actual age is forty to sixty years.

Symptoms.—When angina pectoris is well developed there is no symptom-complex so characteristic and dramatic. Seized on a sudden or with but a few moments' warning, the patient stands or *sits transfixed* with pain and fear. It is difficult to tell whether his *arrested respiration* is controlled by the disease or his own will-power. The *sense of dissolution* or of impending death is so real that the patient expresses the belief that death is at hand, if he can find breath to speak. The expression of the face is one of *intense anxiety* or *horror*, the *skin is pallid* to the degree of cadaveric hue, and the brow is marble white and perhaps bedewed with sweat as the attack advances. The *pulse* during an attack is usually *slow, small,* and *very tense,* becoming feebler and more relaxed as the paroxysm passes away. The pain is often beyond the patient's power of description after his recovery, both as to severity and character. Some patients say that the heart feels as if it were being crushed in a vice; others that a huge stone is crushing the chest wall; others that a heavy bar of steel is laid across the thorax. At times the pain not only extends down the left arm to the fingers, but to the right arm as well. As the attack passes off the patient's expression of keen suffering is modified. A flush may supplant the pallor and a sigh reveals that the seizure has passed. At this time the patient not rarely belches up large volumes of gas, and this seems to give much relief. It is this which gives rise to the euphemistic diagnosis of "acute indigestion" in some of these cases. Sometimes more than one attack may occur in an hour, but this is rare.

Modified forms of severe angina are constantly met with in which the pain is not so severe as in the cases just described. The degree of modification may be so great that little or no pain is felt, this form being called *angina pectoris sine dolore.*

Diagnosis.—When angina pectoris presents itself in its well-developed form, there cannot be much doubt as to its true character. The question of the character of an attack is, however, often in doubt when the symptoms are not all present. While it is a rule that valvular disease of the heart rarely causes pain, it is a fact that attacks of anginoid pain are occasionally met with in cases of aortic regurgitation, particularly if dilatation of the heart is marked. This state can be determined by the diastolic murmur and the "Corrigan pulse." Again, aortitis may cause symptoms practically identical with those of true angina, and it may be impossible to separate the disease of the aorta from that of the coronary arteries because the lesion spreads from the aorta to the openings of these vessels.

It is of some importance to differentiate the angina pectoris due to ordinary coronary sclerosis and secondary myocardial change from that due to syphilis. This is probably impossible by the physical signs in the circulation, but can be made if a history of late syphilis can be obtained, or if the patient is prematurely aged. While these changes, when due to syphilis, cannot be treated as successfully as can secondary syphilis, for they are of the nature of parasyphilitic affections, more aid can be given by the use of the iodides than in those cases which present changes in the coronary arteries from other causes.

A form of pseudoangina is occasionally seen in nervous women and in men who resort to wine, tobacco, and women to excess. It differs from true angina pectoris in the facts that the man is usually under thirty years rather than over forty years of age; his vessels are usually in fair condition; there is a history of neurosis or excessive venery and of the excessive use of tobacco, and the sensation about the heart is that of distention instead of constriction.

Prognosis.—From what has been said as to the state of the vessels and the heart muscle in true angina pectoris it must be evident that the prognosis is most grave, for death may ensue in any attack and an attack may come on at any time. On the other hand, patients sometimes go long periods without an attack, particularly if the mode of life can be quiet and the pulse tension can be reduced by the iodides and nitroglycerin. Much depends in prognosis upon the degree of vascular and myocardial change which can be found. In many of these cases the feeble first sound shows how weak and dilated the ventricles have become.

Treatment.—The treatment of angina pectoris may be divided into that which is devoted to the improvement of the circulatory condition with the object of preventing paroxysms of the disease, and to that which is devoted to the relief of the patient during the paroxysm. As the treatment of a paroxysm requires very active procedures, it will be considered first. If seen as a paroxysm is commencing, the patient should inhale from 3 to 5 minims of nitrite of amyl, or if this drug is not at hand a few whiffs of chloroform should be used. Nitroglycerin should also be given hypodermically in the dose of $\frac{1}{100}$ or even $\frac{1}{50}$ of a grain. If the patient has become accustomed to this remedy, larger doses are indicated. Sometimes it is advantageous to give simultaneously with the nitroglycerin $\frac{1}{4}$ of a grain of morphine; but in those cases in which the paroxysm is not of long duration, the attack commonly passes away before the morphine has an opportunity to exercise its pain-relieving influences. If the patient can swallow, a very useful remedy is 1 or 2 drachms of Hoffmann's anodyne given in a little cracked ice and water; or, if this is not at hand, ½ to 1 drachm of spirit of chloroform may be given in a similar manner. The employment of nitrite of amyl, nitroglycerin, and chloroform is useful in direct proportion to the degree of arterial spasm which is present. In that somewhat unusual class of cases in which attacks of angina occur with a state of low arterial tension, these drugs, manifestly, cannot be of the same value as in those patients in which the systemic arteries are tightly contracted. Should the patient be seen in a paroxysm and the physician possesses none of the remedies just named for his relief, a drink of hot water, containing some capsicum, or some brandy, may be given, since this not infrequently causes the belching up of a considerable quantity of gas followed by some degree of relief to the patient.

The treatment between the paroxysms is dietetic, hygienic, and medicinal. The patient must take a sufficient quantity of food for the purpose of maintaining his nutrition, and must be forbidden to eat anything more than is absolutely necessary for this purpose. Sweet and fatty articles of diet should be entirely avoided, as should be champagne and all sweet wines. If any alcoholic stimulant is required, Scotch or rye whiskey, or a dry gin with lime-juice and sparkling water should be ordered. There is no objection to the diet being largely one of meat, if the kidneys are in a fairly healthy state; the more so, as starchy foods are prone to cause the formation of gas in the stomach and bowels, which may reflexly upset the cardiac balance and precipitate an attack.

The hygienic treatment consists in having the patient take as much sunshine and fresh air as possible; in forbidding him to expose himself to blustering winds, and to warn him that if his peripheral circulation is chilled the consequent contraction of his capillaries may result in an attack of heart pang. Flannel should be worn next the skin both winter and summer. Sudden effort, as going up stairs rapidly or running for a car, or entering into any heated debate, either in court or in a business argument, should be avoided.

As distention in the stomach sometimes causes an attack, it is often necessary to feed the patient with small quantities of food four or five times a day rather than to permit him to eat two or three hearty meals.

Care must be taken that the bowels do not become overloaded with feces, and that they be moved every day by some vegetable laxative or one of the mild saline waters.

The medicinal treatment consists in the administration of iodides in as large doses as the patient can readily bear without danger of disordering his digestion. If there is a history of syphilis in the case, larger doses are needed than if the history is not specific. The patient should take at least 60 grains a day of the iodide of sodium or iodide of strontium if possible, divided into four doses, which should be taken one hour after food. In some cases syrup of hydriodic acid, in the dose of ½ to 2 drachms three or four times a day, may be given with advantage, well diluted with water.

The iodides under these circumstances lower arterial tension and do whatever can be done toward arresting the process of fibroid overgrowth in the bloodvessels. The patient will also be much benefited if he receives nitroglycerin in the dose of $\frac{1}{100}$ of a grain four or five times through the twenty-four hours, the dose being controlled, not by the number of doses administered, but by the effects which it produces upon arterial tension. In some instances it is wise to alternate the nitroglycerin and iodide. If the heart muscle is very feeble, full doses of nux vomica, 10 or 20 drops of the tincture, or moderate doses of digitalis—say, 5 minims of the tincture—may be given three times a day; the nitroglycerin being actively employed at the same time to prevent these drugs from raising arterial tension while they are stimulating the heart. The patient should be instructed to carry glass pearls of nitrite of amyl in his pocket and to crush one and inhale its contents if at any time he feels threatened by an attack.

Symptoms of gastrointestinal dyspepsia are to be relieved not only by regulating the diet in the way indicated, but by the use of pancreatin and taka-diastase to aid digestion. A very useful capsule under these circumstances is one which contains 2 grains of taka-diastase, 2 grains of pancreatin, 1 grain of capsicum, and $\frac{1}{4}$ grain of extract of nux vomica. This should be taken thrice daily.

CONGENITAL CARDIAC DEFECTS.

Two abnormal conditions may arise in the heart of the fœtus and persist after birth, namely, defects of development and defects produced by an attack of endocarditis. In some instances the endocarditis is responsible for the defect in development. The most common of these defects is the persistence of the *foramen ovale*, which permits the blood to make a short circuit through the interauricular septum instead of passing into the right ventricle and thence through the lungs. Sometimes this opening is partly guarded by a membrane, but in other cases no such membrane is present. If the opening is large and entirely unguarded by a membrane the patient is apt to present intense cyanosis, particularly if any effort is made; but in some cases the defect does not produce this symptom and the patient lives to adult years, no one suspecting the presence of such a defect, the existence of which is revealed only at autopsy. Thus, my colleague, Coplin, made an autopsy upon a woman, dead of croupous pneumonia. During her life and in her final illness there were no signs of cardiac disease, but at the autopsy a twenty-five-cent piece could be dropped flatwise through the foramen ovale. More or less oblique communications between right and left auricles are present in about 2 to 5 per cent. of adult hearts.

A much more rare condition is that in which there is an absence of the septum between the right and left sides of the heart, and as a result "bilocular heart" is present. In others again the absence of an interventricular septum produces a "trilocular heart." At times a condition is met with in which a perforation exists in the upper part of the ventricular septum, in the so-called "undefended space."

Valvular anomalies also occur. The three aortic semilunar valves may be replaced by two leaflets. This state while not at all incompatible with life is, nevertheless, prone to become grave, in that the semilunar valves usually become thickened and distorted. The pulmonary valves are much more rarely abnormal, and the valves protecting the auriculoventricular orifices on both sides of the heart are even more rarely anomalous from defective development. These valves may, however, be the subject of endocardial disease prior to birth, and the result is, in one sense, not very different from that met with in the heart of the ordinary individual who suffers from rheumatic endocarditis; for we find the auriculoventricular valves thickened and the chordæ tendineæ broadened and shortened so that they interfere with the free action of the valves. That form of acute or chronic endocarditis which results in the production of granular or warty nodules on the valves is rarely encountered in fetal endocarditis.

The orifice of the pulmonary artery is very commonly found to be in a state of stenosis, as a result of gluing together of the valves and contraction of the ring around the orifice itself. The agglutination of the segments may be so perfectly accomplished as to leave a smooth, funnel-like opening, or the valves may be roughened by vegetations. Patients with this defect may live for years, but it is a curious fact that they are very prone to die of pulmonary tuberculosis.

Stenosis of the right conus arteriosus, of the pulmonary artery, and of the pulmonary orifice are often associated, and form a large proportion of the congenital lesions seen at autopsy in persons who have suffered from these defects, but lived for years. It is a noteworthy fact that these lesions are not rarely complicated by a patulous interventricular septum, an open foramen ovale, and an open ductus arteriosus. Considerable hypertrophy of the right ventricle is naturally found in these cases if life is prolonged.

Narrowing of the aortic orifice is a rare congenital defect. Malposition or transposition of the heart is sometimes seen. Transposition is always associated with transposition of the other viscera.

Occasionally ectopia cordis, a state in which the heart is not protected by the chest wall, is met with. The heart has also been found in the abdominal cavity. Peacock reported one such case in a man of forty-seven years, and Rezek one in a man of thirty-two years.

DISEASES OF THE ARTERIES.

The tubes carrying blood are subject to many alterations, some of which depend upon changes in the perivascular tissues, including with these para-arterial inflammations, infection, trauma, etc. The most important group of vascular changes, however, result from alterations in stress and tension under which the circulation is maintained, and, to a greater degree, are the results of the irritant action of poisons circulating in the blood. The intravascular irritants may be bacterial or of bacterial origin (toxins), unusual quantities of normal salts, or the presence of abnormal compounds that irritate the endothelium.

Of the acute and subacute inflammations involving the intima (endarteritis), and the relation of this change to later alterations in the vessels, we are at present, through the studies of Thayer and others, becoming more familiar. The clinical importance, however, of these alterations is not as yet fully appreciated. Hyaline and fatty degenerative changes occur in the intima and subintimal tissues in a number of pathological processes. The deposit of pigment in the vessel walls, infiltration by lime salts (calcification), and amyloid disease are rarely, if ever, primary in the vessels, but depend upon a number of primary conditions, and therefore are rarely, if ever, recognized independently of the conditions by which they are caused. Infections acting within the vessels give rise to proliferative or necrotic changes in the endothelium with the formation of thrombi (thromboarteritis and thrombophlebitis), which, by mechanical interference of the circulation, influence the nutrition of the organs, or, by causing embolism and distribution of infectious material through the system at large, constitute the basis of septicæmia and pyæmia as already considered.

ARTERIOSCLEROSIS.

Definition.—Arteriosclerosis, as we understand it to-day, evidently comprises a number of pathological processes, the exact relation of which, one to the other, is still somewhat uncertain. Two important processes, often if not constantly associated, are, first, an affection involving particularly the smaller arteries (arterioles), and commonly termed arteriocapillary fibrosis; and, second, a type of arterial change involving particularly the larger vessels and called atheroma, or, on account of the changes in the conformation of the affected vessels, arteritis deformans. Writers are not agreed that these two processes are independent, but they are very constantly associated, and the clinical picture embraced under the term arteriosclerosis includes them both.

Etiology.—Certain individuals, and often whole families, seem peculiarly liable to arteriosclerosis. The change is often a manifestation of age, and the trite but true saying "that a man is as old as his vessels" indicates the belief in preliminary aging of those in whom arterial change occurs in early life. Alcohol, and intemperance in work and eating, and overexertion, mental or physical, are also causes. The autointoxications, lead poisoning, syphilis, and gout are important factors in the production of arterial disease. Certain forms of chronic interstitial nephritis may precede, accompany, or follow arterial change. (See Etiology of Chronic Interstitial Nephritis.)

Recent studies, experimental and pathological, seem to indicate that possibly arteriosclerosis may bear some definite relation to morbid processes affecting the adrenals. Josué, Ziegler, Erb, Pearce, and others have produced atheroma, or a closely allied lesion, by the intravenous injection of adrenalin. Vaquez reported an instance of adenoma of the adrenal associated with heightened arterial tension, and Josué and Bernard, and, more recently, my colleague, Coplin, have shown that in patients having arteriosclerosis the adrenal is rarely, if ever, a normal organ. The observations are not, however, as yet conclusive.

The relation of heightened stress to arteriosclerosis is one of the problems upon which authorities are not agreed. Allbutt recognizes a mechanical arteriosclerosis depending upon prolonged high tension of whatsoever origin. There can be no doubt that in some cases prolonged stress is an important etiological factor, as is shown by the fact that typical arteriosclerosis is rare in the pulmonary artery and its branches, except when mitral disease or pulmonary lesions increase the tension in this vessel, under which circumstance sclerotic changes are not of infrequent occurrence. Heightened stress in the veins also tends toward the development of phlebosclerosis, as is shown by the occurrence of this lesion in the veins of the lower extremity, when for any reason the tension in these tubes is heightened, and also by the development of similar changes in the portal area in cirrhosis of the liver with venous obstruction.

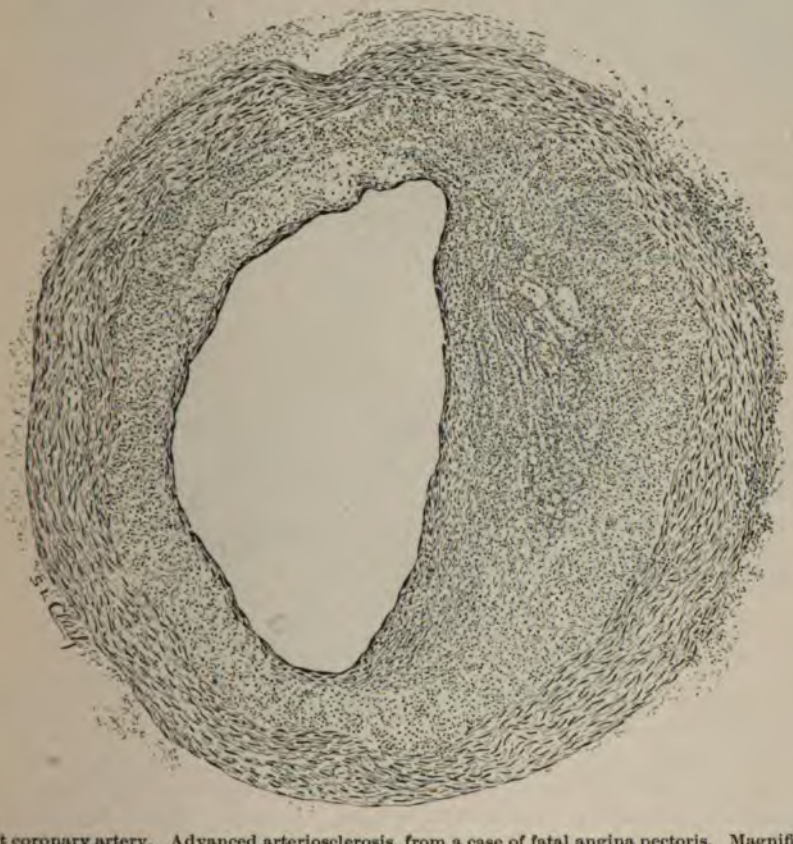
Pathology and Morbid Anatomy. *Lesions in the Terminal or Small Arteries.*—The change in the arterioles is characterized by proliferation of the endothelium and subendothelial tissues, fragmentation of the elastica, and alterations in the media. There has been much dispute as to the primary alteration in the muscle layer, some holding that there is evidence of a distinct hypertrophy, which others fail to recognize. Whether or not there be an initial increase in the muscle layer of the arteriole, there is, sooner or later, if the condition persists, a degenerative change, hyaline in tendency, with loss of elasticity, thickening of the intimal and subintimal tissues, and narrowing of the lumen, and hence increased peripheral resistance.

Lesions in the Larger Arterial Trunks.—In the second conspicuous alteration of arteriosclerosis there develops in the larger arteries a succession of changes greatly influencing the elasticity of these structures. Councilman recognizes at least three divisions of this type of arterial change. In the nodular form a cellular infiltration occurs around the vasovasorum, as originally pointed out by Martin, extending into the media and subintimal layers. Fragmentation of the elastica with efforts at production of new elastic tissue occurs. Later necrotic and degenerative changes weaken the wall and, as pointed out by Thoma, endothelial proliferation tends to restore the smooth lumen. The cells forming the node undergo hyaline and fatty degeneration, giving rise to a mass of cellular detritus constituting the so-called atheromatous abscess. Should the overlying endothelium give way an atheromatous ulcer is formed; these areas are particularly prone to develop around smaller branches given off by relatively large trunks, and lessen the blood-carrying capacity of the affected branches.

The nodules, seen on the vascular surface of the larger arteries, are elevated, yellowish, and often soft from degenerative changes, and, later, are infiltrated by calcareous material, becoming rigid so that they break when bent.

Often associated with this nodular form is a diffuse arteriosclerosis, which may also occur independently. The affected vessels are dilated, thin-walled, with irregular elevations at points, taking on more or less fully the character of the nodules already described. Sometimes the intima is almost unchanged.

FIG. 75



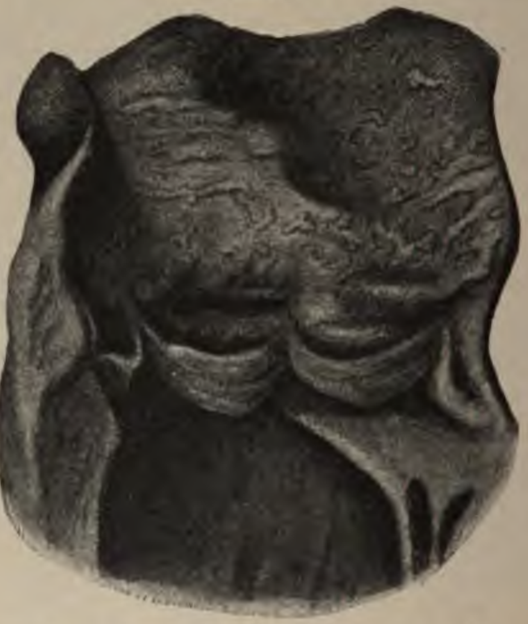
Left coronary artery. Advanced arteriosclerosis, from a case of fatal angina pectoris. Magnified 30 diameters. But little of the adventitia is shown. The media is thinned and at points encroached upon, but the most conspicuous change is in the intima, beneath the endothelial layer of which there has been extensive proliferation and leukocytic accumulation, most marked in the upper segment, greatly altering the lumen of the vessel, lessening its carrying capacity, and rendering it practically inelastic. There was a marked fibroid myocarditis in the area supplied by the vessel.

The senile arteriosclerosis so classified by Councilman would appear to represent that late stage of the nodular type in which so-called atheromatous ulcers and abscesses form with calcareous scales, giving rise to rigid "pipe-stem" or "slate-pencil" vessels that can be rolled under the fingers as tortuous, inelastic, rigid tubes. The blood-carrying capacity of such vessels is materially diminished, and the resistance offered to the circulation proportionately increased.

These briefly described alterations in the arteries may be associated with similar, though usually much less marked, changes in the veins (phlebosclerosis), the combined arterial and venous lesions constituting what Thoma has called angiosclerosis.

The thoracic aorta is the large vessel of all others which usually presents the greatest atheromatous change. Its entire inner surface may be so roughened that it no longer presents any of the appearances seen in health. In other cases, in which the process has not gone so far, we find patches, or plaques, of atheromatous change all over its lining. These patches may cover areas of softening or areas of calcification, and on them thrombi may form. They are particularly prone to appear about the origin of branch vessels, and this is the reason that fatal disease of the coronary arteries so often occurs as part of the aortic changes.

Fig. 76



Atheromatous plaques on the lining of the aorta. (Graupner and Zimmermann.)

In vessels of the intermediate class and in the aorta these changes may so weaken the resistance of the vessel wall that it yields to pressure and an aneurysm develops.

When changes occur in the small vessels their intima becomes thickened by an outgrowth of the endothelial cells, and the connective-tissue cells in the media also proliferate, so that the vessel becomes fibroid, the elastic coat being rendered rigid and the calibre of the vessel diminished. If this process proceeds far it causes an obliterative endarteritis.

The secondary effects of these vascular changes have already been largely considered when discussing the causes of cardiac hypertrophy and myocardial degeneration. The heart, if its own tissues are not invaded, undergoes hypertrophy to enable it to pump blood through rigid, unyielding vessels, and finally, when the pressure becomes too high, develops a leak at the mitral valve to relieve pressure, or breaks down and fails, suddenly or gradually, under the strain thrown upon it. The hypertrophy chiefly affects the left ventricle, because it is upon this part that the strain falls.

Symptoms.—If the heart is examined the apex will be found displaced a little downward and to the left, and palpation will show that the impulse against the chest wall is forcible if hypertrophy is present.

A symptom of equally great importance is the accentuation of the aortic second sound at the second right costal cartilage, and, indeed, wherever it is heard elsewhere in the chest. If the radial or temporal arteries are palpated they are found thickened and corded, often elongated and tortuous and so rigid it is difficult to extinguish the pulse by pressure. This high arterial tension is one of the most important clinical conditions that can be estimated by the physician.

A patient presenting these signs and symptoms may continue in apparently excellent health for several years, but as life progresses the cardiovascular changes also progress, and cardiac failure, attacks of angina pectoris, or renal disease ensue. Much depends in these cases upon how widespread the lesions are and where they are most developed. If the coronary arteries are the parts chiefly affected, anginoid attacks soon come on. If the cerebral arteries are calcareous apoplexy ends existence, or, if the stroke be mild, it were better for the patient if death ensued. Not rarely attacks of vertigo, of fleeting monoplegia or hemiplegia, and aphasia may take place as the result of the arteriocapillary fibrosis, without being due necessarily to rupture of a cerebral vessel. If, again, the renal vessels are involved, then the general manifestations of chronic contracted kidney are produced.

Among the complications may be named cerebral and pulmonary embolus, and gangrene of the extremities from the same cause.

Treatment.—The treatment consists in the use of the iodides, whether there be a history of syphilis or not, in the administration of nitroglycerin to lower arterial tension and relieve the heart of labor, and in the use of gentle exercise and warm bathing in moderation to flush the capillaries with blood. After the circulation begins to fail in the advanced stages of the disease strychnine and digitalis may be urgently required. Alcohol in any form must be avoided as if it were poison. Highly seasoned dishes are also to be avoided. If the heart is tired, strophanthus is to be given to support this organ. High altitudes are dangerous for such patients. Great muscular and mental strain are dangerous because the increased arterial pressure may rupture a vessel or weary the heart.

As chronic interstitial nephritis is nearly always present the treatment for that state should be instituted if any sign of renal disease can be discovered. (See Chronic Interstitial Nephritis.)

ANEURYSM.

Definition.—An aneurysm is a localized dilatation of an artery and depends upon a weakening of its wall so that it is unable to withstand the pressure of the blood. This dilatation may involve the entire circumference of the vessel, forming a cylindrical or fusiform aneurysm, or it may chiefly affect only a part of the circumference, forming a sacculated aneurysm. The walls of the aneurysm are composed of the thinned coats of the vessel, but as the sac develops some of these may in part disappear.

The term "*dissecting aneurysm*" is applied to that form in which the blood escapes through the intima and forces a passage for itself into the middle area of the vessel wall, between the media and the adventitia. By "*false aneurysm*" is meant a state in which all the coats of the vessel give way so that the blood escapes into the surrounding tissues, where a pulsating sac usually forms, owing to the development of fibrous tissue around it. An "*aneurysmal varix,*" or "varicose aneurysm," is one in which the artery communicates with a vein through an abnormal opening, so that the vein and its neighboring veins are distended with pulsating blood. An "*embolic aneurysm*" is one in which a vessel is plugged by an embolus and then undergoes dilatation in its proximal part. "*Mycotic aneurysm*" is often multiple and occurs as a result of the infection of the vessel by micro-organisms, as in ulcerative endocarditis.

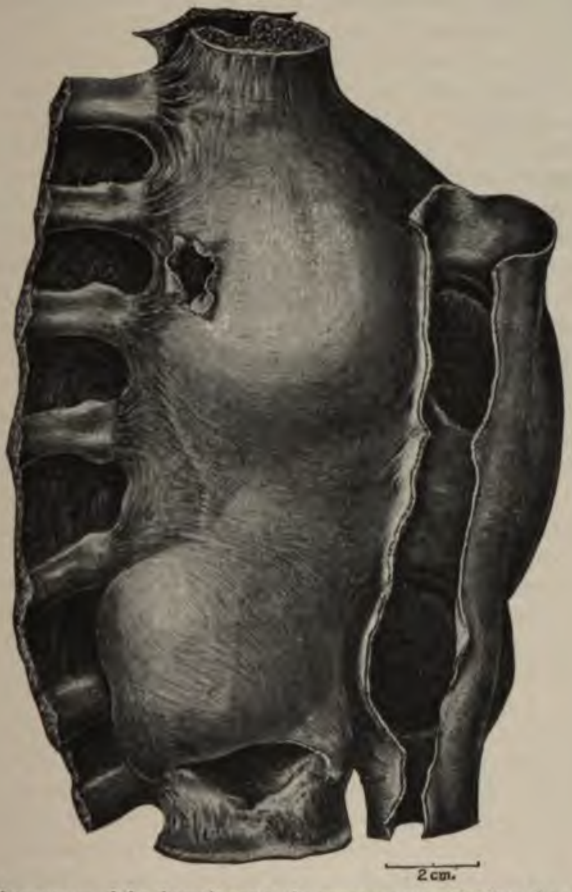
Etiology.—Aneurysm is due to arterial degenerative changes whereby the normal elasticity of the vessel is impaired and its lining membrane injured. (See Arteriosclerosis.) The primary causes of aneurysm are, therefore, identical with those of ordinary arterial disease, and consist in syphilis, alcoholism, and excessive toil or sudden strain. Thus syphilis, for example, weakens the vessel and a strain causes it to give way. There are also cases, not so commonly met with, in which congenital defects seem to exist in the vessel walls. Thus, I saw a few years ago a young man of about twenty-eight years, who developed a popliteal aneurysm and then a thoracic aneurysm and finally died of cerebral aneurysm, but who at no time suffered from syphilis or from strain. It may also arise from injury, as when a vessel is damaged by a stab wound or by a bullet, and these injuries may result in the development of the aneurysm many years after the injury, and only when the arterial changes of advancing age still further weaken the area which was damaged. In other cases the acute infectious diseases, without acusing general or widespread arterial change, may produce localized vascular inflammation and necrosis.

Pathology and Morbid Anatomy.—In *fusiform aneurysm* the wall of the vessel dilates in its full circumference, but certain spots give way more rapidly than others, so that the surface of the dilated vessel is slightly nodular or uneven. As it increases in size its walls become thinner, but if an inflammatory process is set up in the surrounding tissues the actual thickness of the wall may be increased, and finally a marked deposit of lime salts may take place.

In *sacculated aneurysm* the dilatation may originate in at least two ways.

In one, the entire vessel having become weakened, dilates, and the middle coat atrophies. This process of atrophy becomes further advanced in one area than in another, and here bulging rapidly progresses. In other cases the low-grade inflammatory process results in degeneration of the tissues lying under the intima, thereby greatly weakening the sustaining lamina, which eventually yields, so that the fibrous sheath of the vessel may be all that is left of the arterial wall. In either case, however, a process of compensation or repair may develop and the sac become filled with a clot which is usually laminated and remarkably tough. Such aneurysmal sacs often grow to an enormous size, becoming as large as a child's head.

FIG. 77



Double sacculated aneurysm of the thoracic aorta, the upper sac rupturing into the pleura. On the right is the aorta, in which can be seen the two oval openings communicating with the aneurysmal sacs. The inferior margin of the lower sac has been pushed upward, showing the erosion of the body of the vertebra, which, above the point shown, has exposed the spinal canal, but had not, in this case, compressed the cord.

Frequency.—The relative frequency of aneurysm as compared to other diseases is not of much interest, and there are no very large statistics which deal with this point. There is, however, interesting information at hand in regard to the relative frequency of the most frequent and important forms of aneurysm. Thus, at St. Bartholomew's Hospital, Browne found that in thirty years there were 468 cases of aortic aneurysm, 80 of popliteal aneurysm, 21 of femoral aneurysm, 14 of subclavian aneurysm, 8 of carotid aneurysm, and 6 of external iliac aneurysm.

Aneurysm of the Thoracic Aorta.—Not only is aortic aneurysm the most common lesion, but it is, by reason of the importance of this vessel and of the tissues about it, capable of causing very characteristic and also very obscure symptoms, both by disturbing the circulation and by pressure on neighboring organs.

For convenience of study the aorta is usually divided into three parts: the ascending, the transverse, and the descending. Each of these may be the seat of an aneurysm, but the ascending portion is most frequently affected. Some years ago one of my assistants, Holder, and myself studied the statistics derived from 953 cases of aortic aneurysm, and obtained the following results. No less than 570 of these were cases of aneurysm of the ascending portion of the arch. Of these, 544 were sacculated and 466 occurred in males and 78 in females. The remaining 26 cases were fusiform, and all of these 26 cases occurred in males. These statistics emphasize very forcibly the far greater frequency of sacculated aneurysm than the fusiform variety. When we consider that of nearly 1000 cases analyzed, aneurysm of the ascending aorta occurred no less than 570 times, while aneurysm of the transverse portion occurred only 104 times, and of the descending portion 110 times, the great difference in the relative frequency of the lesion in different parts of the aorta is also marked.

Of the 466 cases of sacculated aneurysm occurring in males, it is interesting to note that the great majority of them occurred in persons between thirty-five and forty-five years of age, that the next greatest frequency was in persons between twenty-five and thirty-five, then between forty-five and fifty-five.

When we come to the consideration of aneurysms involving the second or transverse portion of the aorta we find, once again, that the most common age for the development of this lesion is between thirty-five and forty-five; for out of 88 males suffering from this lesion 37 were between these ages, 21 between forty-five and fifty-five, 14 between twenty-five and thirty-five, 10 between fifty-five and seventy, and 2 between fifteen and twenty-five. The same facts as to age also hold true for aneurysm of the descending arch. It is evident, therefore, that aneurysm is not a disease of old age, but of the middle period of life. As Coats has well expressed it, "aneurysm occurs when the period of greatest bodily vigor overlaps the period of occurrence of atheroma."

Symptoms.—Aneurysm of the aortic arch not infrequently lasts for some time before producing any symptoms, and is then spoken of as a "latent aneurysm." In other instances it causes symptoms almost as soon as it develops, and the difference in the promptness with which the signs appear depend largely upon the site of the growth and the parts pressed upon.

When the *convex surface of the ascending* arch is involved we find the patient presenting *engorgement of the veins of the head, neck,* and *arm* on the *right side,* and the *voice* is often *altered* or *lost* from the pressure upon the recurrent laryngeal nerve of the right side. The *pupil* of the *right eye* may be *dilated,* due to irritation of the sympathetic; or it may be *contracted* because the ciliospinal nerves are paralyzed by pressure. There is often *severe pain* due to pressure, and attacks of anginoid pain may be present.

The physical signs are dulness on percussion over the second right interspace, a bruit, or roaring sound, produced by the passage of blood through the sac, and perhaps bulging of the first, second, or third interspace on the right side. If the hand is placed over the area of bulging, a distinct, expansile, heaving movement is felt. Some displacement and hypertrophy of the heart is often present, and, if the sac is a large one, the apex beat of the heart may be far below and outside the normal spot near the nipple. If the sac develops on the concave part of the arch, then the downward displacement of the heart is still greater, for obvious reasons.

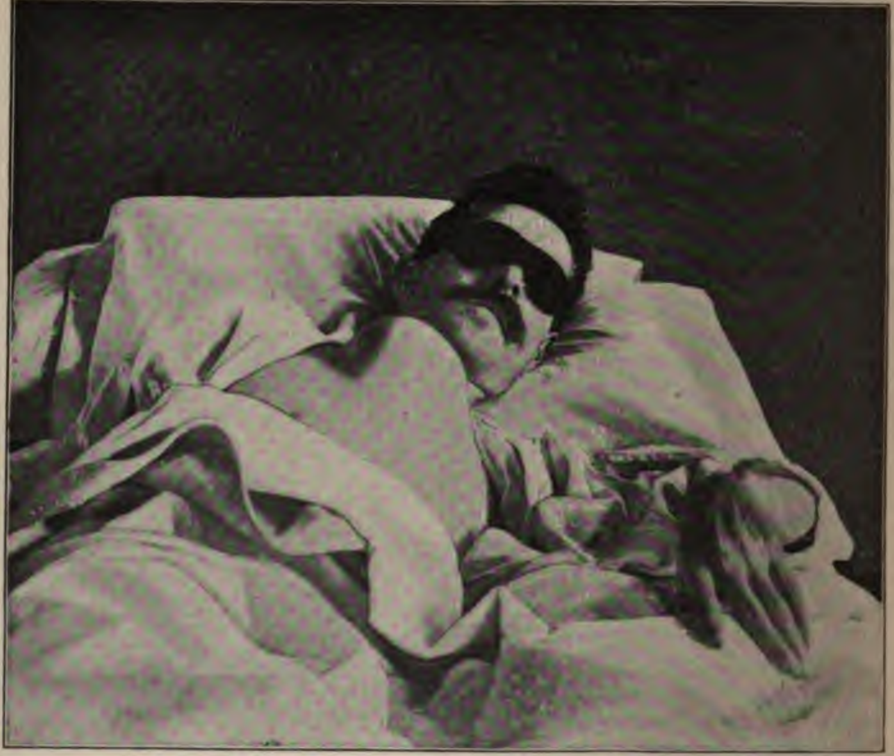
Hypertrophy of the heart is by no means a constant sequence to aneurysm. Not rarely the heart is not increased in size at all, but its apex may be displaced and the impulse transmitted to the chest wall more markedly than normal because of the pressure produced by the aneurysmal sac.

When we come to the consideration of the direction in which sacculated aneurysms of the ascending arch most commonly rupture, or, in other words, when we study the neighboring tissues into which the blood forces its way when the wall of the aneurysm bursts, we find that the vast majority rupture into the pericardium. Thus, out of 289 cases in which death was stated to have been due to rupture in males, 75 ruptured into the pericardium and 58 into the pulmonary artery; 23 ruptured into the right auricle, 3 of these taking place some time before death; 23 ruptured externally; 14 ruptured into the superior vena cava; 11 into the œsophagus; 9 into the left auricle; 8 into the right ventricle; 8 into the trachea; 6 into the left ventricle; 6 into the left pleura, and 5 into the right lung; 3 burst in the posterior mediastinum and 1 burst simultaneously into the trachea and œsophagus; in 20 others no statement was made as to the direction of the rupture. It is, however, a fact that death is much more commonly due to pressure symptoms than to rupture.

Aneurysm of the *transverse portion* of the arch usually causes a *ringing, brassy cough, dysphagia, expansile pulsation* in the suprasternal notch, and *dulness on percussion* on the first and second left intercostal spaces. Its pressure on the innominate vein may cause congestion of the left side of the face and neck. *Dyspnœa* from tracheal pressure may be present, and there may be *aphonia* from paralysis of the left vocal cord, arising from pressure on the left recurrent laryngeal nerve. If the growth is so situated that it passes from the left bronchus it may cause bronchiectasis and even bronchial suppuration by preventing drainage. Again, if the sac be a large one, it may involve the innominate artery, the left carotid, and even the subclavian, and in this manner the radial pulse may be absent on one side. Even the pulse in the arteries of the trunk and lower extremities may be greatly lessened in vigor. The *bruit* may be loud and angry, but if the laminated clot be large it is often absent. The *aortic second sound* is usually *accentuated* or *ringing* in character unless aortic regurgitation is present.

When the *descending arch* is affected it often occurs that the aneurysm extends posteriorly, and the bruit and pulsation are found in the back, between the scapula and the spinal column on the left side. In these cases severe pain due to pressure on the intercostal nerves is often present, and the pressure on the vertebræ may cause erosion and even paraplegia by destroying the spinal cord.

FIG. 78



Aneurysm of the ascending and transverse part of the aortic arch, with erosion of the chest wall.

Aneurysms of the ascending and transverse portion of the aorta often produce an extraordinary degree of erosion, and so pass through the wall of the chest by causing the absorption of the bony tissues, and by pushing the fragments of the ribs to one side. This is well shown in the accompanying cut. The surface of this tumor is often shining from distention of the skin, it is discolored by blood, and the surface may weep bloody serum for days as the end approaches.

Under the name tracheal tugging a symptom of aortic aneurysm, which consists in the transmission of a tugging sensation to the trachea, has been described by Oliver and studied by MacDonnell. To make this test, the patient stands erect with his head slightly tipped backward, so as to stretch the tissues of the front of the neck. The cricoid cartilage is now grasped by the thumb and finger and drawn toward the chin, when if aneurysm is present a tugging sensation will sometimes be felt with each beat of the heart. Sewall has shown that this sign is present in cases which have adhesions in the left pleura, and in some healthy persons when they take a deep inspiration.

Occasionally in aortic aneurysm incurvation of the finger-nails and clubbing of the finger-tips on one side may be present.

Blood-spitting is due to the formation of an erosion of the mucosa at the spot in the bronchial tube where the tumor causes pressure. Such a cause produces only a slight blood stain of the sputum. When the blood passes by a process of leakage through the wall of the sac and escapes into a bronchus it may be in considerable amount, and death may be due to a free hemorrhage of this sort.

Very rarely there develops in the chest, as the result of aortic aneurysm, an adhesion between the sac and the superior vena cava, so that on the development of ulceration a communication between the two vessels is established, forming on a large scale an arteriovenous aneurysm. The most exhaustive study of this state has been made by Pepper and Griffith, and was reported to the Association of American Physicians in 1890. They could find only 28 cases in literature in addition to the one they observed. Less commonly the aneurysm communicates with the pulmonary artery.

Diagnosis.—The symptoms of aneurysm, on which the diagnosis must be chiefly based, have already been mentioned. They may be briefly named as follows: The presence of bruit, expansile pulsation, pressure symptoms, dulness on percussion over the second and third interspace anteriorly on either side, unilateral sweating, and mydriasis or myosis, and thoracic pain. Swellings due to aneurysm nearly always are expansile, but care must be taken that swellings which pulsate by reason of transmitted impulse are not mistaken for a true dilated vessel.

Nothing is more difficult to diagnosticate correctly than the early manifestations of aortic aneurysm. Scarcely a physician of experience can look back and not recall cases in which its early signs completely misled him. The inconstant pain in the chest is often thought to be rheumatic or neuralgic. In other instances dyspnœic seizures are thought to be asthmatic, or attacks of severe cardiac pain are considered to be due to true angina pectoris. The persistence of symptoms like these, despite treatment, the age of the patient, the degenerated state of the palpable arteries, the sounds of the heart, and the history of syphilis, of alcoholism, and of strain or blow or wounds, are all at least capable of arousing suspicion of the real condition. The pain of aneurysm is often dull, gnawing, and constant, but in some cases pain may be absent. Occasionally an unsuspected aneurysm may cause an attack of stridor or paroxysmal dyspnœa, resembling somewhat a laryngeal crisis in locomotor ataxia. This should excite suspicion of aneurysm causing pressure on the recurrent laryngeal nerves. A hemorrhage from the lungs in the absence of tuberculosis should also be regarded as significant. This hæmoptysis may be frothy and mucoid, or rusty, like that of pneumonia, or prune-juice in hue.

A very valuable aid in the diagnosis of thoracic aneurysm is the use of the x-rays, either the fluoroscope being used or x-ray pictures being taken. The advantage of the fluoroscope is the fact that the physician can see the expansile movement of the mass.

Prognosis.—The prognosis of aortic aneurysm is, in the vast majority of cases, inevitably fatal, but in some cases life is preserved for many years if nature succeeds, by the deposition of laminated clot, in walling off the sac. An old scrub-woman has presented herself to my clinic during the past thirteen years, each season, with a massive aneurysm of the aorta which eroded the sternum years ago, and which has not grown since to any extent. She works hard for her living and has little discomfort. Her case is the exception that proves the rule, however. If the growth is of any size, life rarely lasts more than a few months. Even when it is small a rupture may occur. As already stated, death from aortic aneurysm is usually due to pressure on adjacent parts, and not most commonly to rupture, as is generally thought.

Treatment.—Aneurysm of the peripheral arteries is best treated by compression and ligation, and the methods to be employed will be found discussed in works devoted to surgery.

In aortic aneurysm there are two plans of treatment which may be instituted, namely, medicinal and dietetic, on the one hand, and operative on the other.

The medicinal treatment depends to some extent upon the underlying cause of the aneurysm. If it be due to syphilis, in the sense that this disease is chiefly responsible for the vascular degeneration, it is hardly necessary to state that the iodides in full doses are advisable, not that they can cure the aneurysm in the sense of regenerating an old vessel, but that they may, by their specific influence, arrest the degenerative influence in the vessel wall, and so delay the progress of the malady. Even if there is no history of syphilis in the case, the iodides are often of value in that they seem to arrest in some unknown manner degenerative changes in the vessels. They should be given in sufficiently large dose to produce some evidence of iodism, but not in sufficient dose to seriously disorder digestion.

The second point in the treatment of the case is the institution of the greatest degree of rest which is compatible with comfortable existence. The patient should be placed in bed and required to use a bedpan in order that he may not disturb his circulatory equilibrium by getting up. He should also be given small doses of the bromides, if necessary, to overcome nervous irritation and restlessness, and if the action of his heart is exceedingly tumultuous and fails to become more quiet, by rest in bed, I have known small doses of aconite, such as 2 minims of the tincture, three or four times a day, to be advantageous. In other cases veratrum viride is useful in the same dose. Such a plan of rest treatment is useless unless it is carried out for weeks, and sufficient time must be allowed for a laminated clot to form in the aneurysm and reinforce its walls.

The so-called Tufnell treatment of aneurysm consists in a more rigorous method than that just described. The patient is not only put at absolute rest, but he is also given considerable quantities of iodide of potassium and as low a diet as is compatible with existence. Indeed, the treatment may be called the starvation plan of treatment, for it is the deliberate purpose, when this plan is instituted, to lower the activity of the circulation by the depression which is associated with semi-starvation. I have seen this plan instituted in a few cases only, and I have never seen good results from it. Surely, no advantage can accrue except by diminishing the activity of the circulation, and this can be obtained by the use of aconite or veratrum viride.

The use of digitalis in these cases is not advisable because, while the drug steadies the heart, it increases arterial tension and so tends to increase the pressure upon the aneurysmal sac

The operative treatment of thoracic aneurysm is only possible when the tumor is of the sacculated type. The fusiform type of aneurysm contraindicates its employment. A large number of operative procedures have been suggested, but there is only one which has at the present time received general recognition by the profession, namely, the so-called Corradi method, in which there is introduced into the aneurysmal sac several feet of fine gold wire which has been previously twisted about a glass spool, both the spool and the wire being carefully sterilized by boiling before they are used. After the skin over the sac has been carefully sterilized, the greatest gentleness being used lest it be damaged, a hollow needle, which is insulated by being coated with porcelain, is pushed into the sac, and then through it is passed from ten to thirty feet of wire, according to the size of the growth. A larger number of feet have been used, but ten or fifteen feet will be sufficient in the vast majority of cases. As soon as all the wire, save about six inches has passed into the sac, the external end of the wire is made fast to an electrode which is attached to the positive pole of a galvanic battery. A large, wet, clay electrode attached to the negative pole is placed under the back to complete the circuit and by means of a "current controller" the electricity is gently turned on. At first about 5 milliampères are used; at the end of five minutes the current is raised to 10 milliampères, and after this the current is increased every five minutes by 5 milliampères until about 50 milliampères are employed. A higher number of milliampères have been used, but with increasing experience I am confident that they are unnecessary and perhaps harmful. As the result of this method of procedure it not infrequently happens that by the end of the first twenty minutes or half-hour the sac is found to be somewhat more firm than before, and that pulsation in it has diminished owing to the fact that the acid reaction produced by electrolysis about the gold wire has resulted in the formation of a clot, which, as time goes by, becomes more and more firm and solid. At the end of from thirty minutes to an hour the wire is disconnected from the battery, its external end is pushed underneath the skin, and the external wound is closed by collodion. Absolute quiet must be maintained for ten days or two weeks after the operation, in order that the clot may become thoroughly consolidated.

I have now performed this operation thirteen times, and my experience has been that in no instance did the patient suffer much pain. In several instances the patient has stated during the operation that he was much improved, and in every instance he has voluntarily expressed his pleasure at the subsequent improvement. Unfortunately, the condition of the aorta is such that when we close one bulging spot in this way, it is not long before another area gives way under pressure, and so another aneurysm is formed. In all of my cases this accident has ultimately occurred. The greatest duration of life after any one of the operations which I have performed has been a little over six months. But in one case operated on by Stewart some years ago the patient lived in comparative health for a period of three years, and then died from pneumonia following an alcoholic debauch, although at the time of operation the aneurysm could be seen as large as a fist outside the chest wall. Considering the slight pain of the operation, it is indicated in many cases, if only for the temporary relief which it gives.

Aneurysm of the Abdominal Aorta.—Aneurysm of the abdominal aorta usually occurs near the diaphragm, and it is far more rare than aneurysm of the thoracic aorta. Out of 325 cases of aortic aneurysm collected at Guy's Hospital in forty-six years, 54 involved the abdominal aorta. Of these 63 per cent. occurred between the ages of twenty-one and forty years and 77 per cent. between twenty-one and fifty years; over 90 per cent. occurred in men. The growth usually arises from the neighborhood of the cœliac axis. Like that in the thorax, its sacculated form is more common than its fusiform type. It usually projects forward, but it may extend backward and cause erosion of the vertebræ, followed by pressure on the spinal cord. *Pain* is usually a constant symptom, which is often referred to the region of the heart or to the back. On inspecting the abdomen *distinct pulsation* may be seen at once, but care must be taken that the transmitted pulsation of the aorta in a state of health is not mistaken for true expansion. These two states can be separated by careful palpation. In some instances the aorta can be clearly felt through the belly wall; in other cases a morbid growth in the stomach or in the omentum can be felt, but although it pulsates it is not expansile. If the patient is placed in the knee-elbow position so that the growth falls away from the aorta, the diagnosis may be readily made. *Auscultation* may reveal a *bruit* if aneurysm is present, but if a stethoscope is used it is easy by pressure on the aorta to narrow its lumen and cause a humming sound, which is not a sign of aneurysm. Again, in hysterical persons a very marked pulsation of the aorta may be complained of by the patient and felt by the physician. I saw not long since an hysterical male who had rhythmical contraction of his abdominal rectus muscles synchronous with his pulse. It was only possible to diagnose his case by giving him ether to the point of relaxation.

A larger proportion of these cases end by rupture than is the case in aneurysm of the thoracic aorta. Thus, out of these 54 cases no less than 43 died from rupture.

Aneurysm of the abdominal aorta may rupture into the retroperitoneal tissues, through the diaphragm into the pleural spaces, or into the general peritoneal cavity, or more rarely into an intraperitoneal viscus, as, for example, the stomach. Rarely death ensues from the vessel becoming closed by a thrombus.

Even more rare than aneurysm of the abdominal aorta is aneurysm of its branches. Cases have been recorded in which a blow upon the belly has caused *aneurysm of the hepatic artery*, and in a case seen by the writer it followed ulceration produced by a malignant growth of the gall-bladder. That this condition is very rare is shown by the fact that up to 1898 only 26 cases had been recorded. The symptoms are usually mistaken for hepatic colic, and this error is all the more easy because jaundice from pressure is often present. In most of the recorded cases the antemortem diagnosis has been "gallstones" or "duodenal ulcer."

Aneurysm of the splenic artery is rarer than aneurysm of the hepatic artery, and causes symptoms like those of gastric ulcer.

Aneurysm of the superior mesenteric artery is also rare. Rolleston has collected 20 cases. Embolism is the usual cause, and injury may be responsible for it. This growth may cause jaundice by pressure on the gall-duct, as in a case reported by J. A. Wilson; or it may press on the renal arteries and cause uræmia, as in a case recorded by Burney Yeo. Aneurysm of the inferior mesenteric artery is practically unknown.

Aneurysm of the renal artery is also rare, although small multiple sacs are sometimes seen. If the sac be very large hæmaturia may occur, or wasting of the kidney may ensue. Sometimes by the sudden rupture of an aneurysm of this vessel the retroperitoneal space has been filled with blood. In the surgical clinic of the Jefferson College Hospital, my colleague, Dr. W. W. Keen, recently cut down on a kidney because of severe renal symptoms, and found a large aneurysm of the artery. He was forced to remove the aneurysm and the kidney.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

STOMATITIS.

Definition.—As its name implies, stomatitis is an inflammation of the mouth. Of the many forms that have been described three are important: catarrhal stomatitis, aphthous stomatitis, and ulcerative stomatitis. All these forms of stomatitis usually occur in childhood. The catarrhal and aphthous forms are more common in early infancy, but the ulcerative type is practically never met with in children who have not as yet gotten teeth, and is more common in those past puberty than are the other forms.

Catarrhal Stomatitis.—In catarrhal stomatitis there is hyperæmia of the mucous membrane of the tongue and cheeks, with an increase in secretion on the part of the mucous and salivary glands. It arises from injury, as by some foreign body being taken into the mouth which acts as a mechanical irritant, or by hot or irritating liquids. These are, however, only predisposing causes. Difficult dentition, or the use of a rubber nursing nipple which is dirty, are more common factors. It also occurs as one of the manifestations of the acute eruptive diseases, as in scarlet fever and measles, and it may be a symptom of some metallic poisoning, as mercury, lead, or arsenic.

Symptoms.—The symptoms of catarrhal stomatitis are *intense hyperæmia* of the mucous membrane of the mouth with some *swelling* which is particularly visible on the gums. If the finger be placed in the mouth a sense of increased heat is felt. The child evidently suffers a good deal of *pain*. When given the breast or bottle it eagerly seizes it because of hunger and thirst, and then, as the nipple touches the tender mucous membrane, gives a cry of pain and disappointment. Cool water is usually taken with avidity if given from a cup. The *flow of saliva is so free* that constant dribbling on the chin is present or the excess of saliva is swallowed and disturbs digestion. If the mouth be very carefully examined it may be that tiny blisters at the opening of the mucous glands will be seen and the papilla of the tongue will be found enlarged, swollen, and unduly red in hue. Some *digestive disturbance* and *diarrhœa* are nearly always present. Whether these symptoms are the result of the condition of the mouth, or whether the state of the mouth is secondary to disturbed digestion, is often difficult to determine.

Prognosis.—Recovery from catarrhal stomatitis is usually rapid, the condition rarely lasting for more than a few days after the disordered digestion is corrected and proper cleanliness of the mouth is obtained.

Aphthous Stomatitis.—The aphthous form of stomatitis, sometimes called follicular or vesicular stomatitis or canker, may be considered as a still further development of the catarrhal form. In this condition we not only have a diffuse hyperæmia of the mucous membrane of the mouth, but in addition small spots appear which look as if the superficial epithelium had been snipped off with curved scissors. These spots are, of course, exquisitely sensitive. All the symptoms of the catarrhal form are much exaggerated, and in addition to more marked digestive disturbance the patient often has systemic disturbance, as is shown by some fever and general wretchedness. Nutrition is interfered with materially only by the inability to take food.

Ulcerative Stomatitis.—The ulcerative form of stomatitis, sometimes called fetid stomatitis, or putrid sore mouth, is a much more serious type, but it is often so mild that the dividing line between it and the aphthous form is not readily made. When well developed, the mucous membrane is seen to be studded by small ulcers which may coalesce, forming rather large areas of superficial necrosis. If the child is badly nourished, or suffering from some malady which impairs its general nutrition, ulcerative stomatitis may become a very serious affection, causing *great suffering*, interfering seriously with proper feeding and rapidly undermining the strength of the patient. The ulcers are situated chiefly along the edges of the gums which recede from the teeth, or on the margins of the tongue, on the buccal membrane, and even on the lips. The *breath* is often *very foul* and the corners of the mouth become excoriated from the salivary flow. When the patient is a sufferer from scurvy and bad hygienic surroundings, it may be an important factor in preventing recovery.

In many cases the condition arises from carious teeth and occasionally the disease occurs epidemically. On the other hand, I have repeatedly seen mild ulcerative stomatitis occur in otherwise healthy young girls past puberty, in whom none of these causes were apparent.

In severe cases the ulceration may be very deep and may even cause loosening of the teeth or superficial necrosis of the lower jaw.

Treatment.—The treatment of all these forms of stomatitis may be considered simultaneously. All of them are to be treated by careful attention to cleanliness of the mouth itself and of all objects entering the mouth, by careful regulation of the bowels and the food, and by a mouth-wash of chlorate of potash and myrrh in the following formula:

℞.—Potassii chlorat.	gr. xxx.
Tinct. myrrhæ	♏ x.
Elix. calisayæ	f℥iij.—M

Sig.—Dilute one tablespoonful with two of water, and use as a mouth-wash

When very young children are treated this solution may be applied to the mucous membrane by means of a swab tied to a small stick or pencil. These measures are usually efficient in the catarrhal and aphthous type.

In the ulcerative form it may be necessary in addition to touch each ulcer with a solid stick of nitrate of silver or with a solution of this drug of the strength of 60 grains to the ounce. This is painful, but efficacious. Only a few spots should be touched at each sitting. When the teeth are not carious peroxide of hydrogen may be used locally. The system should be well supported by nutritious foods such as cold consomme or cold chicken-jelly, by ordinary foods if they can be taken into the mouth, and, if anæmia is present, by the use of iron and quinine in moderate doses.

When for any reason chlorate of potash cannot be applied locally the physician may give the drug internally in the dose of 2 grains every three hours, since, as it is eliminated by the salivary glands, the saliva bathes the diseased mucous membrane with the drug. This plan of treatment is contraindicated if any renal irritation or marked gastric disturbance is present.

In scorbutic cases fresh vegetables, fruit, and beef-juice are absolutely needful in the treatment of the patient.

There still remains to be discussed in this connection three forms of sore mouth which do not, strictly speaking, fall under any of the headings just given, since in all of them definite specific causes have been isolated, namely, so-called thrush or parasitic stomatitis, gangrenous stomatitis, and mercurial stomatitis.

Thrush.—Thrush, or parasitic stomatitis (Soor or Muguet), is due to the presence in the mouth of a parasite variously named *Saccharomyces albicans, Monilia candida,* or *Oidium albicans.* This parasite has been classed with yeasts, and grows with branching filaments at the ends of which egg-shaped, torula cells are produced. Thrush is distinctly associated with impaired health of the mucous membrane, and in children, in whom it is most commonly met with, it is due, as a rule, to the use of dirty nursing nipples or nursing bottles, or to general impairment of health. When the latter cause is present, thrush may appear in the mouth of adults, and for this reason it sometimes aids in increasing the miseries of those whose vitality is impaired by tuberculosis, diabetes, and by prolonged exhausting fevers. It is also seen in children suffering from marasmus. The parasite can be readily conveyed from one person to another by utensils.

Symptoms.—The subjective symptoms of thrush consist in the same *discomfort in the mouth* which is met with in aphthous stomatitis. The objective symptoms are, however, to be carefully separated from that state. Instead of denudation or depression of the surface of the mucous membrane there is seen on the tongue small particles or specks in the form of *tiny pearly white spots,* which are raised and may gradually coalesce and seem to from a membrane that is usually easily removed, although its removal may leave a bleeding surface. From the tongue the growth may extend to the entire mucous membrane of the mouth and the soft and hard palate. Very rarely it even spreads to the pharynx and œsophagus, and even into the stomach and small intestines. Holt states that the fungus has been found in the lungs of babies suffering from bronchopneumonia. Thrush is separated from aphthous stomatitis by the fact that the profuse salivation of the latter is replaced by great dryness of the mouth, and by the aid of a microscopic examination of the growth.

Prognosis.—The prognosis depends upon the health of the patient. In the robust the condition may last but a few days, but in the feeble and impoverished it may persist for weeks. It does not materially influence the general health except by interfering with the taking of nourishment.

Treatment.—The treatment consists in cleanliness, good feeding, the use of a mouth-wash of borax in the strength of 10 grains to the ounce, or of a mouth-wash of permanganate of potash, 1 grain in 8 ounces of water, or by diluting peroxide of hydrogen, 1 part in 5 of water. Any underlying disorder of nutrition should be carefully removed if it be possible. All sweets and syrups should be carefully avoided.

Gangrenous Stomatitis, Cancrum Oris, or Noma.—Noma is a term applied to all forms of severe ulceration of a localized character attacking mucous membranes, but the state may be present without the mucous membrane being broken, the tissues of the cheek being chiefly affected. The condition occurs so rarely in those who are past puberty that it may be said to be a disease of early childhood. It affects the buccal mucous membrane and cheek so constantly that the word "noma" has come to mean a malignant and sometimes a perforating gangrenous process involving the cheek, although, strictly speaking, noma may affect the ear, the vagina, the buttock, the nose, or the external genitalia.

In the vast majority of cases noma results in the death of the patient, not so much because it is in itself a fatal disease as that it is a terminal infection; that is, one which develops only in a child or adult whose vital resistance is so lowered by disease or poor nutrition that the pathological process known as noma is possible.

The disease is rarely seen in private practice, but has its greatest frequency in institutions for poor children, where because of mismanagement, or of the wretched state of the child on admission, it readily falls a victim to infections of all kinds.

Noma more commonly follows measles than any of the other eruptive diseases, but it also sometimes complicates or follows scarlet fever, typhoid fever, or whooping-cough. It is practically never a primary lesion at the point of development, but begins in a solution of continuity such as an ulcer due to a carious tooth or as a sequence of ulcerative stomatitis. In all probability it is not due to an infection by any single micro-organism, but to several organisms which may be associated. Cases have recently been recorded in which the ulceration seemed to be due to the bacillus of diphtheria, but mixed infection is the rule. An attempt to establish specificity for any single organism has been futile.

Not only has the ulcerative process a tendency to rapidly become deep and so to perforate the cheek, but it lacks the sharp line of demarcation marking the wall often built by nature to prevent the spread of gangrene. That is to say, the ulcer is bounded by an extending area of necrosis, often branching and discolored, which spreads from day to day, with no apparent effort on the part of the system to limit its progress. The soft parts melt into the fetid ulcer, and cartilage and bone undergo necrosis. When recovery does occur, which is exceedingly rare, a line of demarcation forms, and the extension of the disease is arrested in this way; the ulcer clears up and repair slowly takes place.

Symptoms.—The local symptoms of noma are a *foul breath,* a state of localized ulceration with *deep induration* of the tissues near by, and the speedy development in the centre of the ulcer, of a dirty-looking *slough* of necrotic tissue. The side of the *face* is usually much *swollen* and *distorted,* and when felt by the fingers the tissues feel brawny and hard. If the ulceration extends to the gums, the teeth become loosened. So great may be the destructive process that the teeth and alveoli may be seen through the perforated cheek.

The systemic symptoms are not characteristic of any specific state, but are those of *profound systemic poisoning, depression,* and *exhaustion.* The *pulse* is *rapid* and *feeble,* the *appearance* of the patient markedly *septic* and *cachectic,* and the *temperature* mildly *febrile.* The height of the temperature depends, however, to a considerable degree, upon the vitality of the patient. When the sufferer is profoundly exhausted and the vital state is very low marked febrile movement does not occur.

Treatment.—From the description just given it is evident that the treatment of noma, to be successful, must depend upon its early institution and thorough character. It must also consist in the use of such nutritious food and such medicines as will serve to support the strength of the patient. The local treatment consists in the early and complete cauterization of the part affected by the electrocautery or its excision by the knife, so that the necrotic mass is at once destroyed and removed. The physician must not limit the operation to the dead tissues, but extend the excision to the living tissues as well in order that none of the infected tissue may remain. The local process may be temporarily treated by swabbing the part with peroxide of hydrogen or by using permanganate of potash. In cases in which the streptococcus or the bacillus of diphtheria are present, the serum therapy needed for these specific infections should be used.

ECZEMA OF THE TONGUE.

Under this distinctly erroneous term is described a condition in which there is a superficial overgrowth and desquamation of the epithelium covering the tongue. As a rule, the centre of each spot of desquamation begins to heal while the periphery is still spreading, so that the appearance of the inflammatory zone is distinctly circinate. Its irregular outline has given it the name of "geographical tongue." In other instances the appearance of the tongue is that of a worm-eaten leaf. In some patients it produces no discomfort whatever. In others the patient may have some itching and tingling, and on examining the tongue is surprised to find the curious outlines which have been described. Not infrequently nervous patients are wont to consider that it is an evidence of syphilis, or perhaps of a malignant growth. A modified form of this condition is very frequently seen in children as a result of a catarrhal condition of the stomach and bowels.

Treatment.—In adults it is best treated by the local application of nitrate of silver, 20 grains to the ounce. In children the correction of the gastrointestinal disorder usually results in a normal growth of epithelium, so that the condition is relieved. As a rule, such children require some simple bitter such as tincture of gentian with 5 or 10 grains of bicarbonate of soda.

LEUKOPLAKIA BUCCALIS.

Leukoplakia buccalis is sometimes called smoker's tongue, ichthyosis lingualis, and buccal psoriasis. It is characterized by the development of white spots of considerable size on the mucous membrane of the mouth and tongue, which are due to cellular infiltration of the subepithelial connective tissue and a thickening of the epithelium. When the spots occur on the edge of the tongue and are indented by the teeth they sometimes look like the scars or puckerings which are seen on the edges of the tongue in cases of advanced syphilis. Occasionally these areas may be slightly ulcerated, and in some cases are thought to be the seat of epitheliomatous degeneration. In the majority of instances, however, they are benign, and after removal of the cause require no treatment unless the surface is ulcerated, in which case they may be touched with nitrate of silver and the patient directed to avoid taking hot, irritating substances into the mouth, and particularly to avoid smoking or chewing. Leukoplakia is sometimes looked upon as a precancerous condition, and this is probably true in the sense that the state if permitted to continue may lead to cancer.

MUCOUS PATCHES.

Mucous patches are opaque, white, flattened swellings on the mucous membrane of the mouth and lips, and are characteristic of secondary syphilis. (See Syphilis.) From them the virus of syphilis is readily communicated. Not infrequently their surface is somewhat ulcerated.

Treatment.—The treatment, of course, consists in the active employment of mercury internally and the use of nitrate of silver locally.

DISEASES OF THE SALIVARY GLANDS

FUNCTIONAL DISORDERS OF THE SALIVARY GLANDS.

Ptyalism.—Ptyalism, or salivation, occurs as the result of poisoning by mercury or the iodides. It is also produced by such drugs as jaborandi, and occasionally occurs because of irritation of the mucous membrane of the mouth by the development of stomatitis in one of its several forms. Rarely a form of idiopathic ptyalism is met with in young children. Under these conditions the salivation is probably the result of a neurosis.

When due to the influence of a drug, the condition is to be arrested by stopping the use of that substance and aiding in its elimination by the employment of sodium bicarbonate if the iodides have been taken, and mild saline purgatives if mercury has been used. A mouth-wash containing 10 grains of chlorate of potassium and 2 drachms of fluid extract of rhus glabra in an ounce of water will be useful to improve the condition of the mucous membrane and arrest the flow of saliva. Sometimes moderate doses of atropine are useful. In other cases, 10 to 15 grain doses of camphoric acid given thrice a day may be used.

DRY MOUTH.

Dry mouth, or *xerostomia*, is frequently met with in all fevers, but sometimes occurs as an independent condition. Under these circumstances the tongue is seen to be red and dry, with lessened superficial epithelium, so that it is smooth and shiny. It is said to be most frequent in women after great nervous excitement or in those suffering from hysteria.

Treatment.—Temporary relief from dryness of the mouth may be produced by washing it with a mixture of 1 part of glycerin to 2 of water, to which has been added a little lemon-juice.

INFLAMMATION OF THE SALIVARY GLANDS.

The most important and most common inflammation of the salivary glands is the swelling of the parotid gland in mumps, which has already been considered. The next most frequent cause of inflammation of the salivary glands is a septic condition of the mouth in the course of one of the prolonged adynamic fevers, such as typhoid fever, and in persons who are suffering from cerebral softening. In these cases it would seem that infection may pass through the salivary duct, and so cause inflammation of the gland itself. Occasionally a similar accident occurs in pyæmia, pneumonia, syphilis, and scarlet fever. In some of these diseases the infection undoubtedly enters the glands by way of the bloodvessels and lymphatics. Pyogenic infection of the parotid, whether hæmal, lymphatic, or by a duct, produces a suppurative interstitial parotitis, or parotid abscess, also called "parotid bubo." A curious form of inflammation of the parotid gland with stenosis of Steno's duct is sometimes seen in cases of sulphuric acid poisoning. While it is possible for the sublingual and submaxillary glands to be involved, the parotid is the gland which nearly always suffers.

The inflammation may be treated in its early stages by cold compresses, by the application of leeches, and by the administration of circulatory sedatives like aconite, provided the patient is not already depressed by disease. So far as possible the treatment should also be addressed to the relief of the underlying cause of the condition. If the gland is suffering from a subacute inflammation, mercurial ointment, iodine ointment, or ichthyol ointment may be thoroughly rubbed into the skin over it, unless perchance the gland is swollen as the result of mercurial or iodide influence. When an abscess forms it must not be forgotten that it should be opened promptly, but with great care. Not infrequently a parotid abscess is so closely associated with an important bloodvessel, or indeed with the external carotid artery, that a careless incision may produce disaster. Such an abscess should always be opened by careful dissection.

Chronic indurative or sclerosing parotitis is a well-known pathological condition the clinical features of which are still obscure. It has been observed in diabetes with and without pancreatic disease, but the exact relationship, if any, is not known.

Ludvig's Angina.—This condition, sometimes called *Angina Ludovici*, is a cellulitis of the neck which begins as an inflammation of the floor of the mouth, usually in the area of the submaxillary gland. As a rule it begins on one side and then spreads to the opposite side. In malignant cases it rapidly involves not only the floor of the mouth, but the tissues about the root of the tongue and under the angles of the jaw, so causing œdema of the glottis. In some cases these tissues slough, forming what is known as *cynanche gangrænosa*. In still others, an abscess points externally.

Symptoms.—The symptoms are brawny swelling, great pain, rigors, fevers, and, if the infection is severe, general septicæmia and death.

Treatment.—In the very early stages the use of cold and of leeches to the skin under the jaw may be of advantage. After the inflammation is well developed, drainage should be established by the surgeon.

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Definition.—Acute pharyngitis is an acute catarrhal inflammation of the mucous membrane lining the pharynx in which there is hyperæmia and congestion, with some infiltration of the submucous tissues, and, later, an increased secretion of mucus.

Etiology.—Acute pharyngitis is caused, as a rule, by simultaneous exposure to cold and infectious dust. Cold and damp air first impair the vital resistance of the pharyngeal mucous membrane, and then dust, laden with microorganisms, falling upon it speedily produces infection. There can be no doubt that systemic conditions also favor the development of this state.

Aside from the fact that lowered vitality always permits infection to take place readily, there can be no doubt that the excessive use of alcohol, tobacco, or rich foods, or the presence of a torpid liver, or a catarrh of the nose, mouth, or stomach aid materially in permitting the condition to arise. Pharyngitis also arises as a result of lithæmic states. Sometimes infection seems to come from a chronic tonsillitis or is an extension from the nares. The condition is particularly prone to arise in those who work for a number of hours a day in imperfectly ventilated rooms. Sometimes irritating fumes and dust seem capable of producing this condition.

Pathology.—After a preliminary dry stage the engorgement of the blood-vessels of the mucous membrane and the inflammation of the mucous glands results in the pouring out of considerable quantities of mucus, marked epithelial desquamation, and in severe cases some fibrin. If pyogenic organisms are present the secretion may be distinctly mucopurulent. In some instances the swelling of the submucous tissues is very marked. In others, although severe, it may be superficial. In very rare cases the exudate may be so fibrinous as to form a false membrane which is not always due to the presence of the bacillus of diphtheria. Not rarely in such cases the uvula and the tonsils are also involved.

Symptoms.—Acute pharyngitis usually comes on suddenly, with the symptoms of what is popularly called "*sore throat*," so that the patient feels that the *mucous membrane* is *swollen* and *sore*, and there is some *pain* on swallowing. On inspection it will be found that the posterior wall of the pharynx and neighboring parts are dry and red, with the capillaries injected. After secretion is established the parts are thoroughly moistened by serum and mucus. In some instances, if the infiltration of the submucous tissues is marked, the patient may complain of a *sense of constriction* in the throat, and at times a good deal of pain may extend along the Eustachian tube into the ear. If the pain is not too great, the patient may continually clear his throat in an effort to relieve the irritation. Constitutional symptoms are usually mild, but the *tongue is coated* and the patient may be somewhat depressed.

Prognosis.—Recovery always ensues unless some unforeseen complication arises.

Treatment.—The treatment consists, if the patient is seen in the early stages, in the application of a cold compress to the neck below the angle of the jaw. This compress is made by dipping cloths in ice-water, wringing them out, and then binding them against the part. They should not be allowed to become warm and so produce the relaxing effects of a poultice. Internally, if the patient is an adult, he may be given from 1 to 5 drops of the tincture of aconite with a drachm of sweet spirit of nitre in a glassful of hot lemonade. Before taking this he should be put to bed in order that when perspiration develops he will not catch cold. Small pieces of ice may be held in the mouth, but, as a rule, better results will be obtained if the patient gargles with as hot water as he can bear. This water may be fortified by adding to it an equal quantity of the distilled extract of witch-hazel. If the bowels are at all constipated, saline purgatives, such as citrate of magnesia, should be used to unload them. If there is any rheumatic or gouty tendency, the patient will do best if, in addition to the purgatives, he is given 20 grains of bicarbonate of potash in large draughts of water every four or five hours;

or, instead, 10 grains of salol may be given every three hours until 40 grains have been used.

Local treatment aside from the use of the gargle is usually unnecessary. If the condition is due to a gouty tendency, the use of any one of the oily substances commonly employed in atomizers produces increased discomfort, in the writer's experience. If it is not due to this cause, some relief may be obtained by spraying the parts with 3 drops each of oil of sandal-wood and oil of sassafras in an ounce of liquid albolene. In other cases, when the oils cause discomfort, my colleague, Dr. Kyle, strongly recommends applying hydrochloric acid, in the proportion of 5 to 10 drops of the dilute acid in an ounce of water, for the purpose of contracting the dilated bloodvessels.

The salicylates are useful if lithæmia is present.

When the second stage is reached 5 to 10 grains of chloride of ammonium may be given in equal parts of fluid extract of licorice and water four or five times a day. Or, instead, 10 grains of benzoate of ammonium may be given in capsule four times a day.

ULCERATIVE OR PHLEGMONOUS PHARYNGITIS.

Etiology.—This condition, sometimes called ulcerated sore throat, or phlegmonous pharyngitis, is due to an infection of the mucous membrane of the throat by micro-organisms. It is not uncommonly seen in physicians and nurses who are attending children suffering from scarlet fever and diphtheria. Many years ago it was frequently met with in medical students who were dissecting cadavers which had been imperfectly preserved. This form of pharyngitis is exceedingly painful in its early stages, and is characterized by changes much like those just described in acute catarrhal pharyngitis, except that a superficial necrosis of the mucous membrane rapidly occurs, so that in a few hours small, irregular ulcers may be seen upon the soft palate, the half arches, and the pharyngeal wall. If the infection is severe, so that the submucous tissues are involved, it becomes a phlegmonous pharyngitis.

Symptoms.—A patient with infectious pharyngitis usually complains of much *pain in the throat* and in the *muscles of the neck.* This is greatly increased when he attempts to swallow. There may be *slight febrile movement* and depression.

Treatment.—The treatment consists in spraying the inflamed mucous membrane with a normal salt solution, and following this by a gargle or spray of 1 per cent. solution of carbolic acid and water or albolene. This in turn is followed by a spray of menthol, 4 grains to the ounce. Cold compresses applied under the jaw are advantageous. If one or two ulcers are particularly active, they may be touched with nitrate of silver. Usually it is advisable to give the patient a moderate purgative dose of calomel and to follow it by a saline purge, such as a Seidlitz powder.

CROUPOUS PHARYNGITIS.

Etiology.—Croupous pharyngitis occurs in two forms as diphtheria, which has already been described, and as a simple membranous pharyngitis, which commonly is due to infection by the pneumococcus or *Streptococcus pyogenes*. The chief difference between this form of pharyngitis and diphtheria is that the mucous membrane is not deeply involved, that true ulceration never occurs, and the Klebs-Loeffler bacillus is absent. The inflammation is, however, of such a character that a false membrane develops with desquamation of the epithelium. If the membrane is removed, the tissues beneath may bleed, very much as they do in diphtheria. Although there is usually a slight chill and some fever, the degree of systemic disturbance is by no means as marked as in the well-developed case of true diphtheria.

Treatment.—The treatment consists in washing the infected parts thoroughly by a spray of normal salt solution, and following this by a solution of hydrogen peroxide and water half and half, and this in turn by gargling and spraying with distilled extract of witch-hazel. Usually this treatment is sufficient. If the condition persists, Loeffler's solution may be applied locally. Antitoxin is to be used if diphtheria is present. (See Diphtheria.)

CHRONIC PHARYNGITIS.

Etiology.—A condition of chronic inflammation of the pharyngeal mucous membrane is frequently met with in certain climates, particularly that of the Atlantic seaboard. It is also found in persons who continually use the voice, and so it has obtained the name of "auctioneer's" or "clergymen's" sore throat. It is also met with in persons who use tobacco to excess, and sometimes in those who take too much alcohol. Obstructions in the nasal passages seem distinctly to predispose to this state.

Pathology.—The pathological condition consists in a thickening of the mucous membrane of the pharynx and an increase in the connective tissues of the mucous membrane itself, and of the submucous tissues. This may result in a secondary atrophy of the glands in the mucous membrane.

Symptoms.—The symptoms consist in a thickening of the pharyngeal secretions and irritation of the mucous membrane, so that the patient is continually attempting to clear the throat, which often feels dry and harsh. The cough is spasmodic, unproductive in its result, and is made much worse by exposure to cold and dust.

Treatment.—The treatment consists in a regulation of the digestive system, in giving tone to the circulation if it is feeble, in rest for the nervous system if the patient is overworked, and in the internal administration of benzoate of ammonium in 10 grain doses several times a day. Before going to bed at night the patient should gargle his throat with hot water or with hot salt solution, and if the bloodvessels are much dilated a spray of dilute hydrochloric acid, 10 drops to the ounce of water, should be used.

FOLLICULAR PHARYNGITIS.

Etiology.—Under the name of follicular pharyngitis a closely related condition to that just described exists, in which an annoying cough is persistent, and in which a considerable number of enlarged follicles, surrounded by an injected mucous membrane, can be seen on the posterior wall of the pharynx.

Treatment.—The treatment is of the same character as that just described for chronic pharyngitis. Occasionally it is necessary to cauterize the follicles. An excellent application is tincture of iodine 1 part in 2 parts of glycerin.

DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

Definition.—As its name implies, this disease consists in an acute inflammation of the tonsils, accompanied by great swelling of their tissues, and an associated pharyngitis. It occurs in two forms, the follicular and diffuse. The follicular form is distinctly infectious.

Etiology.—Acute tonsillitis is the result of an infection by pathogenic micro-organisms, which are practically always present in the crypts of the tonsils, but do not penetrate the mucous membrane until its permeability is increased by congestion, or by general causes affecting perhaps the vital resistance of the entire body. In many instances the suppurating form arises because the organisms attempt to enter the general system by way of the tonsils, and the suppurative process is the result of an effort to prevent such an entrance. The streptococcus is a cause in some cases. In other cases the organism which Poynton and Payne think is the cause of acute articular rheumatism is responsible, and the bacillus of diphtheria is also a common factor.

Follicular tonsillitis is more common in the period of life from five to twenty years than at any other time, and is rare in infancy. Some individuals suffer from frequent attacks until they reach forty or fifty years of age. After this time of life it is very rare.

Although follicular tonsillitis is rare in adults, the suppurating form is frequently met with in this class of patients. Persons who have a lymphatic temperament are far more susceptible than persons of the wiry type, and it is particularly prone to occur in those who, because of obstruction of the nasal passages, are "mouth breathers," or who suffer from subacute or chronic hypertrophic catarrh of the nasopharynx. One attack distinctly predisposes to another. My experience leads me to believe that it is distinctly infectious, for I have repeatedly seen healthy persons develop the malady after being exposed to the breath of those who were ill with it. Another predisposing cause is the breathing of vitiated air, and air that is contaminated by sewer gas or smoke. How much these influences act directly as sources of infection and how much as agents which, by diminishing vital resistance, make infection possible, is difficult to determine.

Pathology and Morbid Anatomy.—In acute follicular tonsillitis there is an inflammatory swelling of the parenchyma of the gland. The mucous membrane covering the gland is intensely hyperæmic and may even show vesicles on its surface. Each follicle exudes a cheesy-looking mass, and these masses dot the surface of the tonsil or coalesce and produce a tonsillar coating, which at first glance closely resembles the false membrane of diphtheria. If some of this material is examined microscopically it is found to consist of dead epithelial cells, micro-organisms, and pus cells. In addition to these superficial changes there is hyperæmia of the tonsillar capillaries, and proliferation of the lymphoid cells in the deeper tissues of the glands. In the more deeply situated and intense inflammations of the gland, sometimes called quinsy, there is a necrosis of the tissues, suppuration takes place, and the pus escapes from the tonsillar abscess, either by the aid of the surgeon's knife or by rupture of the abscess wall. In any of these conditions the passage of bacteria or their toxins into the lymphatics may produce glandular enlargement in the neck.

Symptoms.—A patient suffering from the earliest stages of acute tonsillitis may first feel *soreness of the throat,* with a sense of local swelling or constriction, or the systemic signs of the infection may first be manifested. Creeping, *chilly sensations,* or even a true rigor, may develop, and there is very frequently an amount of *aching* and *pain in the limbs* which is extraordinarily severe, so that the patient complains most bitterly, not only of this symptom, but of the degree of illness, so that he fears a serious malady. *Violent headache* is often a prominent symptom, and the *temperature* soon becomes *very high,* mounting to 103° or 104° or even 105° in a few hours. Rarely *nausea* and *vomiting* may occur. An examination of the throat will show the presence of distinct swellings of the tonsils, which not rarely extend as far across the fauces as the uvula, and even press against one another. These swellings are intensely congested and frequently covered with exudate, and are often very foul in appearance. The *breath* of the patient is exceedingly *foul,* and unless ventilation is very good the odor may fill the room. Owing to the swelling of the tonsils and adjacent glands and stiffness of the muscles of the sides of the neck, an examination of the patient's throat may be very painful.

As general systemic infection often enters the body by way of the tonsils it is wise to be on the watch for signs of *endocarditis.* Doubtless the association of rheumatism with tonsillitis by many practitioners is due more to the development of septic arthritis from the entrance of pathogenic germs by these pathways than to any real relationship between acute rheumatism and tonsillitis. I have seen several cases of severe ulcerative endocarditis and acute arthritis follow an acute tonsillitis. It is a noteworthy fact that acute follicular tonsillitis is practically always bilateral, while the deeper form, sometimes called quinsy, is often unilateral.

In the suppurating form of the disease the systemic manifestations are often less severe than in the follicular types just described, but the local pain is often very severe, and opening the mouth may be very painful. The tonsil is often enormously enlarged, but is rarely dotted with follicular spots. Instead it may be smooth and shining in appearance. The inflammation often extends to the uvula, which may be so swollen and elongated as to cause great distress.

Treatment.—The treatment of both forms of tonsillitis is largely identical. To the surface of the tonsil, in the first twenty-four hours of the inflammation, there is nothing better to arrest the process and relieve pain than pure guaiacol applied by a cotton applicator. This often causes great pain for the moment, but it is remarkably efficacious. Externally, over the gland, a small ice-bag is a valuable application. It should be kept constantly applied for several days. Internally in the very early stages the use of biniodide of mercury is very useful in the dose of $\frac{1}{500}$ of a grain every half-hour till $\frac{1}{50}$ grain has been taken.

After the stage of onset is past the best internal treatment is 10 to 20 minims of tincture of iron chloride well diluted with water every three or four hours and potassium bicarbonate, or citrate, in copious draughts of water to flush the kidneys and diminish the backache.

Many practitioners rely largely on salol or salicin at this time, giving them in full doses. They are efficacious, but they increase headache, disorder the digestion, and may irritate the kidneys, which are prone to irritation in this disease.

Some cases get relief from gargling with very hot water or by holding ice in the mouth.

When the tonsils are chronically enlarged and repeated attacks of tonsillitis occur they should be removed between attacks by the tonsillotome. The application of caustics like nitrate of silver often makes them larger than before.

In the suppurating form the pus should be evacuated as soon as it is formed, the tonsils being punctured by a bistoury or a tenotome, the tip of which is exposed after being run through a small cork so as to guard it and prevent any movement of the patient from causing the physician to injure an important bloodvessel.

In all cases of follicular tonsillitis cultures of the secretions from the throat should be examined for the Klebs-Loeffler bacillus, for in all cases before making a diagnosis of follicular tonsillitis the physician should carefully exclude diphtheria. (See Diphtheria.) When children are in the household the patient should be carefully isolated.

CHRONIC HYPERTROPHIC TONSILLITIS.

Definition.—Chronic hypertrophic tonsillitis is a condition of overgrowth, or hyperplasia of the tonsils, which affects all parts of both tonsils and usually involves the so-called pharyngeal tonsil. In some instances the lymphoid texture of the glands is chiefly affected, while in others the connective tissue undergoes the greater part of the overgrowth. In the one instance the enlarged tonsils present themselves as projecting masses, soft in texture. In the other they are remarkably hard and cut with a resistance almost cartilaginous in character. In some instances there is overgrowth of the tonsils without the adenoid of the pharynx being involved. Nearly always patients with this affection suffer from associated nasal catarrh, often from secondary middle-ear disease, and they present a peculiar expression of stupidity or lack of intelligence.

Symptoms.—Aside from organic disease of the great viscera there is no chronic malady which produces such extraordinary changes in the physical appearance, growth, and mental development as does this one. The obstruction to free nasal breathing results in *mouth breathing*, and this in turn causes the child to hold the mouth open in a silly manner, which detracts from its facial appearance. So, too, the lack of free respiration results in a failure of physical development, so that the *chest* is often *poorly developed*, and even the entire body is dwarfed. Finally, this same cause produces *restless nights* and so causes loss of physical rest, which may be emphasized by attacks of *spasmodic croup* or *night screaming*. *Constant cough* at night on lying down is also often a troublesome symptom and is due to undue dryness of the nasopharynx or to tickling of the uvula by the edges of the projecting tonsils.

After the disease has lasted for years the child is often stupid, morose, and apathetic to a degree, and the open mouth, stunted nose, and heavy eyes make a diagnosis of the tonsillar state easy. When the child is stripped the chest is often found to be barrel-shaped or the patient is pigeon-breasted, or it presents the *Trichterbrust* of the Germans, or the so-called funnel chest of English writers, in which there is a deep depression of the lower part of the breast-bone. These thoracic changes are due to two chief causes: first, the general impairment of nutrition produces malnutrition of the thoracic walls as in rickets, and, second, in the effort at sufficient respiration the chest walls undergo faulty development. The *breath* is often *quite fetid*, due to retained secretions and particles of food in the crypts of the tonsils, and if the tonsils be pressed upon a surprisingly large amount of material can be expelled from their cavities.

The nasopharyngeal spaces of such children are first-rate culture fields for the growth of bacteria of all sorts and for acute infections, such as diphtheria and scarlet fever.

Treatment.—If we except the effects produced by the use of thyroid gland in cretinism it is not possible to find any state in which the physician can cause such a complete metamorphosis in his patient as to health of mind and body as in this malady. The removal of the enlarged tonsils by tonsillotomy and the scraping away or curetting of the pharyngeal adenoid results in free and easy breathing and in an extraordinary change in growth and spirits. Children who have been stunted in mind and body for years gain, it may be, thirty pounds in a few months, become rosy and bright-looking, and are able for the first time to keep up with their fellows in school and in sport.

The use of cod-liver oil and syrup of the iodide of iron after the operation is a great aid to speedy recovery.

Physicians and parents should regard it as a duty to see that children are relieved from this trouble, which threatens health and mental power, and which exposes the patient as a fair mark to infectious disease. Chronic middle-ear disease and deafness often ensue if these growths are not removed.

While it is true that operation often gives permanent relief, it sometimes has to be repeated, as the tissues redevelop after removal of the primary hypertrophy.

As an anæsthetic ether is far safer than chloroform during the performance of the operation of tonsillotomy or curetting the pharyngeal tonsil.

DISEASES OF THE ŒSOPHAGUS.

ŒSOPHAGITIS.

An acute inflammation of the œsophagus often ensues after the ingestion of irritant poisons, such as concentrated lye, ammonia-water, carbolic acid, and similar substances. Under these conditions it is but a part of the general inflammation of the gastrointestinal tract which all irritant poisons induce, and has little interest except from a toxicological standpoint and the strictures that commonly follow. When œsophagitis is due to disease it may arise from an extension of a diphtheria from the pharynx or from an extension of the inflammation in the pharynx in cases of scarlet fever. So, too, in certain cases of typhoid fever the œsophagus may undergo inflammatory changes, and these may progress to such an extent that ulceration ensues. (See Typhoid Fever.) Sometimes, too, the œsophagus is involved in cases of aphthous stomatitis and in thrush.

A membranous œsophagitis occurs, but it is rare. It may follow the swallowing of escharotics and is occasionally seen after prolonged alcoholic excesses. A cast of the entire œsophagus may be expelled.

The mucous membrane in ordinary inflammations of the œsophagus is reddened and hyperæmic. There may be some pain beneath the breastbone, which is increased by the swallowing of food or drink, but in the milder types no symptoms are present, and unless ulceration is followed by cicatrization, neither the physician nor the patient may be aware of the fact that the œsophagus has suffered from an inflammatory process.

Should symptoms of pain and discomfort exist, they may be relieved by the use of demulcent drinks, of which perhaps the best is emulsion of sweet almonds, or milk with arrowroot. Sometimes swallowing small pieces of ice gives relief.

In cases of heart disease and cirrhosis of the liver a form of chronic œsophagitis sometimes develops, in which the mucous membrane suffers from a chronic catarrh and the smaller bloodvessels become varicose and rupture, causing the vomiting of blood.

ORGANIC STRICTURE OF THE ŒSOPHAGUS.

As has already been intimated, stricture of the œsophagus usually follows the healing of an ulcer which is produced by the ingestion of some corrosive substance, or by ulceration developing in the course of typhoid fever or syphilis. Stricture may be cylindrical or annular, symmetrical or asymmetrical, single or multiple. The usual sites are behind the cricoid cartilage, opposite the bifurcation of the trachea, and at the *hiatus œsophagei.* Occasionally inflammation of a lymphatic gland, the pressure of a tumor in the mediastinum, or of an aneurysm, causes a narrowing of the gullet, and, more rarely still, a polypoid tumor may grow from the mucous membrane. Malignant stricture will be discussed later.

Symptoms.—The symptoms consist in *difficulty in swallowing* food, the patient stating that it lodges part way down to the stomach, and that if any considerable quantities are taken regurgitation takes place, the regurgitated materials containing no trace of gastric juice or the products of digestion. If an œsophageal bougie is passed it is found to be arrested at the level of the stricture, and it may be impossible to push it past this point.

Treatment.—The treatment consists in the use of a small bougie to dilate the stricture, followed by the employment of larger, graduated bougies. If the stricture is so tight that food cannot pass and the life of the patient is endangered by inanition, and if the use of a bougie fails to overcome the obstruction, surgical interference will be necessary. Sometimes stricture of the œsophagus is a congenital condition, in which case the child rarely lives, and little can be done for its relief.

DILATATION OF THE ŒSOPHAGUS.

Etiology.—Dilatation of the œsophagus occurs in two forms, namely, the diffuse and the localized. In the diffuse form the entire tube is dilated, and there may be some overgrowth of the muscular fibres, which has occurred in an endeavor on the part of the œsophagus to force food past an obstruction which exists near the cardiac orifice of the stomach.

When a localized dilatation occurs it takes the form of a diverticulum, which may be divided into two types, namely, "pressure diverticula" and "traction diverticula." The pressure diverticula are very rare and are found usually at the junction of the pharynx and the œsophagus, where the muscular fibres are weakest. Their origin is supposed to depend upon pressure upon this point of the gullet in deglutition, and they are thought to develop in those who are in the habit of bolting their food. The diverticulum arising from this cause is lined with mucous membrane, its submucous tissues are thickened, and the muscular coat is atrophied so that the mucous coat bulges through it as a hernia-like projection. The lesion always occurs in the posterior or posterolateral wall of the œsophagus.

Traction diverticula occur more frequently than the type just mentioned, but are very seldom recognized during life. They are thought to be due to contraction of tissues which have become attached to the œsophagus by inflammatory adhesions, as in cases in which inflammation of the bronchial lymph glands has taken place. This lesion is usually found in the anterior wall of the gullet, and a number of diverticula may be present in a single case.

Atonic dilatation of the œsophagus is occasionally observed; the dilated tube may be fusiform or flask-shaped, the part of the organ corresponding to the neck of the flask being upward. Such dilatation occurs independently of organic disease below, and has been attributed to spasm of the cardia. The dilated œsophagus may be extremely capacious, holding a pint or more of fluid.

Symptoms.—The symptoms of diffuse dilatation of the œsophagus due to stenosis are those produced by stricture of the gullet, and consist chiefly in *difficulty in swallowing.* It is a condition rarely recognized during life. The small diverticula due to traction by adhesions are also rarely recognized, unless one of them becomes so large that it forms a pocket in which food accumulates, until by reason of its decomposition or fermentation it produces so much irritation and inflammation that the stomach, œsophagus, and all the muscles of the chest and abdomen endeavor to expel it by a process akin to that of vomiting. Sometimes a diverticulum of this character is capable of holding a very considerable quantity of fluid.

Diagnosis.—The diagnosis of a diverticulum is reached by the use of a stomach tube, which is passed as far as it will go and then used as it would be in lavage, or as a stomach pump would be employed. Under these circumstances milk or other liquids which have been swallowed are brought up when the tube has not been passed far enough to make it possible that the liquids are obtained from the stomach. Such large diverticula are usually of the traction type. A diverticulum or dilatation may be discovered by filling the cavity with a mixture of bismuth and syrup of acacia and using the x-rays.

Treatment.—The treatment consists in the use of a stomach tube for the purpose of feeding the patient, provided the physician or the patient is successful in passing the tube past the diverticulum. In those cases in which the diverticulum is so large that it prevents the ingestion of food, by pressure on the œsophagus, and so interferes with the patient's nutrition, the only resort is a surgical operation and rectal alimentation.

SPASM OF THE ŒSOPHAGUS.

This affection, sometimes called "*œsophagismus,*" is rarely met with except in persons suffering from hysteria or insanity. It is sometimes seen in hydrophobia, and it is this difficulty in swallowing water, rather than an actual dread of water, which has given that disease its name; for with the thought of swallowing, at the sight of water, the spasm develops. The difficulty in swallowing usually points to the presence of spasm or stricture, and if there is no history of the ingestion of an irritant some time before, or other causes which would be likely to produce ulceration and stricture, the strong possibility of spasm is to be considered. Usually in spasm it is much easier to pass an œsophageal bougie than it is in cases of stricture. But occasionally the spasm is sufficiently tight to oppose the passage of the bougie, and to give to the hand guiding the bougie the sensation which is produced by the end of the instrument coming in contact with a true organic obstruction.

Treatment.—The treatment consists in the passage of a bougie once or twice a day; in the administration of nervous sedatives if the patient is in a condition of nervous excitation; of stimulants, if she is in a condition of profound depression, and of measures devoted to the improvement of the general health, both mental and physical, if it is impaired. As the spasm may be due to a fissure, ulcer, or other tender spot the food should not be bulky, but preferably liquid and never taken in large amounts.

CANCER OF THE ŒSOPHAGUS.

Cancer of the œsophagus usually occurs in the form of squamous epithelioma, and affects men more frequently than women. Occasionally a medullary cancer is found in this region. The growth soon undergoes ulceration, and not infrequently the entire circumference of the tube may be involved, and in this manner a stenosis may be developed with dilatation of the œsophagus above the point of growth. The disease is quite rare, for of 7290 cases of cancer collected by Williams but 6 per cent. were primary in the œsophagus. Tanchon in 9118 cases of cancer found only 13 in the œsophagus. Cases have been recorded as early in life as nineteen years, but most patients are past forty. The disease is about equally divided as to its occurrence in different levels of the tube. Thus, if the statistics of Kraus, von Hacker, and Newmann are added together, making 1477 cases, we find that 582 were near the cardia, 453 near the middle third, and 440 at the upper third.

Symptoms.—The symptoms, as in cases of ordinary stenosis due to stricture, consist in *difficult deglutition* and not infrequently *considerable pain*, both at the time of swallowing and when the œsophagus is at rest. The *food* is often *regurgitated* almost as soon as it leaves the pharynx, and sometimes the effort at regurgitation is followed by the appearance of some blood and mucus. *Blood* and *mucus* also very frequently appear if a bougie is used to pass the stricture. The age of the patient, the absence of a history of the ingestion of a corrosive poison, the emaciation and weakness, and the presence of primary or secondary growths elsewhere render the diagnosis possible. Sometimes an aneurysm of the thoracic aorta by pressing on the œsophagus may produce somewhat similar symptoms, but under these circumstances the physical signs of aneurysm may be found.

Prognosis.—The prognosis in œsophageal cancer is, of course, exceedingly bad. Death comes from exhaustion and starvation, from pneumonia due to the inhalation of septic materials, or to ulceration of a large blood-vessel. Sometimes the lymphatic glands of the neck are secondarily involved. Occasionally the physician is chagrined at the autopsy to find that an unsuspected œsophageal cancer has been present for weeks without presenting symptoms, and without his having recognized its existence.

Treatment.—Medicinal measures are of course fruitless except for the relief of pain. Surgical measures are of little value, since the patient is usually so exhausted by the time that the diagnosis is confirmed and he is willing to resort to an operation, that life is prolonged but little by the operative interference. Exner has recently reported a case in which improvement followed the use of radium in a tube attached to a bougie.

DISEASES OF THE STOMACH.

ACUTE GASTRIC CATARRH.

Definition.—Acute gastric catarrh, or acute catarrhal gastritis, as its name indicates, is a state in which the mucous membrane of the stomach becomes hyperæmic and then swollen, with lessened secretion, followed by excessive production of mucus and reduction in the quantity of digestive ferments. The term acute gastritis is sometimes employed to describe this state. This is unfortunate because a true inflammation of all the coats of the stomach is not present except when irritating foods have been taken in excess, or when some irritant poison has been swallowed.

Etiology.—The causes of acute gastric catarrh are very various. In children it is often a sequel to taking cold, with some resulting interference with the activity of the liver. It also follows the excessive use of sweets in this class of patients. In older persons it is nearly always due to the takingof an excessive amount of indigestible food or overloading the viscus with ordinary foodstuffs, particularly if alcoholic drinks and highly seasoned articles have been swallowed.

Symptoms.—The symptoms of acute gastric catarrh in children are quite different from those met with in adults in most instances. The child has *nausea* and *vomiting*, and some *epigastric discomfort*. *Fever*, varying from 100° to 102°, often develops and persists for several days. The *bowels* may be *constipated* or *several loose movements* may occur daily. The appearance of the tongue in this class of cases is very characteristic. It is evenly coated by a thin white fur which is dotted by many tiny red spots where the enlarged papillæ exist. The *tongue* is also somewhat *drier than normal* and the *breath* slightly *foul* and *hot*. The urine is usually decreased in amount and high colored.

In adults acute gastric catarrh presents somewhat different symptoms. There is often some *pain* or *tenderness* on pressure *in the epigastrium* and a loss of appetite amounting to disgust for food. *Nausea* may be quite persistent and *vomiting* may occur.

In both children and adults the attacks may last for from one to four days.

Diagnosis.—When the physician is called to see a child who is suffering from moderate fever, a somewhat coated tongue and epigastric distress, he must not be hasty in stating that the case is one of acute gastric catarrh, because many of the acute infectious diseases begin by moderate gastric disorder and a coating of the tongue. Whenever the gastric symptoms persist for more than four or five days, enteric fever should be suspected as being the cause of the illness, but care must be taken that the common error of calling mild typhoid fever "gastric fever" is not made. Uncomplicated acute gastric catarrh is so rare in adults, who have not abused alcohol, that great care should be taken before the physician is satisfied with this diagnosis.

Treatment.—The treatment consists in unloading the portal and hepatic circulation, which is nearly always disordered, by the use of small doses of calomel, giving ¼ of a grain every half-hour for eight doses, and following this in five hours by a Seidlitz powder or some other mild and cooling saline purge. If the stomach is very irritable the Seidlitz powders should be divided into fourths and taken at fifteen-minute intervals. After this treatment has acted the patient should receive small doses of bismuth subnitrate and oxalate of cerium. For a child 2 grains of the former and 1 of the latter may be given every hour for five or six doses. Adults may take 5 grains of the bismuth at a dose.

The rest of the treatment consists in the use of small quantities of liquid food, such as whey or barley-water, or scalded toast. Small quantities of milk and lime-water may be used. In some instances total abstinence from food for twelve or twenty-four hours gives the best results. Rest in bed is also very advantageous for both children and adults.

ACUTE TOXIC GASTRITIS.

Etiology.—Acute toxic gastritis is produced, as its name implies, by the entrance into the stomach of any poison which is capable of acting as a severe irritant or as a corrosive. Some of these irritants act with only sufficient severity to produce hyperæmia and moderate superficial inflammation, but others corrode the stomach, and may even perforate it. Most of the metallic poisons, such as mercuric chloride and mercuric iodide; oxalic acid, carbolic acid, and the ordinary mineral acids, such as sulphuric, hydrochloric, and nitric acids, belong to the class of severe corroding irritants. Other substances, such as phosphorus and chlorate of potassium, not only act as irritants, but produce fatty degeneration. Arsenic differs in its action on the stomach from that of the mineral acids in that it does not coagulate albumin and is not corrosive. It causes, however, a peculiar exfoliation of the mucous membrane lining the stomach and bowels, partly by its local effect induced by direct contact, and partly by the elimination of the arsenic by the gastric walls, whereby the mucous membrane is shed. Among the vegetable substances which may cause gastritis may be mentioned croton oil, elaterium, and castor-oil beans.

Morbid Anatomy.—As the result of the violent inflammation of the stomach produced by these drugs death may ensue, or, if the patient survives the acute stage, extensive exfoliation of the superficial part of the mucosa occurs, or ulcers may form which ultimately cause death. Free and interstitial hemorrhages occur. In other instances there is so much destruction of the peptic tubules that the gastric mucosa is largely destroyed and cicatricial contractions ensue.

Symptoms.—The symptoms of toxic gastritis are *violent burning pain* in the *stomach*, *thirst*, and *vomiting*. The vomited matters may be stained with blood, and usually contain an excess of mucus and often flakes of the mucosa itself. Sometimes *diarrhœa* develops. The *pulse* is rapid, the patient's *expression is anxious*, and death may occur within a few hours in collapse.

Diagnosis.—While it is not possible to make a positive diagnosis as to the character of the poison which has been taken by the appearance of the stomach at autopsy, certain poisons nevertheless produce definite lesions which may be considered almost peculiar in themselves. When sulphuric acid is taken, the parts are blackened; nitric acid makes them yellow. Most of the alkalies stain them brown and the silver salts cause intense redness followed by a brown and black hue on exposure to light. It is also stated that oxalic acid causes a peculiar gelatinous appearance of the gastric mucous membrane and that ammonia may cause a suppurative condition.

Treatment.—The treatment, of course, depends upon the character of the poison. In all instances, except when phosphorus is taken, olive oil may be given to protect the stomach; morphine should be used hypodermically to relieve pain and irritation, and external heat should be applied to combat shock. When one of the acids has been taken, any alkali such as lime-water, plaster from the walls suspended in water, very dilute ammonia, or even soap and water may be used to antidote the acid except in the case of oxalic acid, when only lime or chalk should be used; for while calcium oxalate is insoluble, the oxalates of potassium and sodium are not insoluble, and are practically as poisonous as the oxalic acid itself. When lead in one of its soluble forms, or carbolic acid is taken as a poison the antidote is any soluble sulphate, of which magnesium sulphate and sodium sulphate are the best.

The after-treatment of acute toxic gastritis consists in the use of large doses of bismuth subnitrate, and in the employment of rectal feeding, so that the stomach may be put at rest for as many days as possible.

PHLEGMONOUS GASTRITIS.

Definition.—The term "phlegmonous gastritis" is applied to a condition in which the inflammation is of such a character that it proceeds to suppuration, the chief part of the suppurative process developing in the submucous tissues, and sometimes extending to the muscular coat of the stomach and even to the peritoneum.

Etiology.—The disease is exceedingly rare, and has appeared with about equal frequency at all ages from ten to eighty years. In some instances it is primary, the seat of the infection beginning in an ulcer or growth, or more rarely from some direct traumatism. The secondary cases are those in which the process develops during the course of one of the acute infections, such as typhoid fever, puerperal fever, pyæmia, and variola. Fifty cases have been gathered by Jacoby and 85 by Leith, but these figures of course include many of the same instances of the disease. Phlegmonous gastritis is of two kinds: a diffuse form with a rapid course, profound systemic disturbances, and speedy death, and a circumscribed form in which the symptoms are much less severe; the patient living, it may be, for weeks, but usually dying as the result of the disease.

The pyloric portion of the stomach is chiefly affected. Its walls are greatly thickened, and the submucous tissues are riddled with pus, having undergone almost complete necrosis. Not infrequently many perforations of the mucous membrane occur, so that the internal surface of the stomach has a sieve-like appearance.

Several cases of suppurative gastritis have been reported in the United States, and they are remarkable enough to demand notice. In Kinnicutt's case the disease followed an alcoholic debauch. In Hemmeter's case the patient had vomited blood, was treated for gastric ulcer, and did not die until two months after the onset of severe symptoms. The infection had here occurred through a healed ulcer. In Smith's case violent epigastric pain with collapse and death in two and a half days took place, the entire submucosa being infiltrated with pus. In Loomis' case abdominal pain, bilious vomiting, and a feeble pulse with delirium preceded death, which occurred at the end of four days. In Hun's case the gastric walls were fully one-half inch in thickness and the tissues between the peritoneum and the mucous membrane were purulent.

In the circumscribed form of phlegmonous gastritis the abscess may be single, or multiple abscesses may be present.

Symptoms.—The symptoms in the diffuse form consist in the sudden development of *violent epigastric pain*, followed by *faintness* and *collapse*, with incessant vomiting. After the vomiting has continued for some time, the vomited matter may be stained with bile.

When the disease is secondary to malignant growth, it is stated that vomiting does not often develop. Notwithstanding the severity of the process, *pus is never found in the vomit* in the acute cases. The temperature varies from 103° to 105°. The *pulse is rapid* and *feeble*, and becomes more and more so until death occurs in *collapse*, preceded by a stage of apathy.

Diagnosis.—There is practically no array of diagnostic symptoms in cases of this disease save the onset of violent epigastric pain.

The pus may escape into the general peritoneal cavity, the patient dying with symptoms of perforation and collapse. The most extraordinary case of this character so far recorded is the one reported by Callow, in which the patient vomited a pint of pus, pus appeared in the stools, and there were seven pints of it in the peritoneal cavity. All this pus was traced to a large abscess cavity in the wall of the stomach which had, however, not been accompanied by pain during its formation.

Treatment.—When it is possible to make a diagnosis prompt operative interference is strongly indicated. So far, this has not been attempted early enough in any case to give satisfactory results. The parts should be drained and a gastroenterostomy performed. (For an excellent paper on this subject see Moynihan in the *Medical Chronicle* for November, 1903.)

DIPHTHERITIC GASTRITIS.

Inflammation of the stomach with the formation of a false membrane due to the Klebs-Loeffler bacillus is very rarely encountered. It nearly always follows diphtheria in the upper air-passages. Still more rarely in cases of septicæmia, scarlet fever, and smallpox a false membrane may develop in the stomach. The condition is of interest solely from a pathological standpoint.

MYCOTIC GASTRITIS.

Inflammation of the stomach due to the growth of specific micro-organisms upon its mucous membrane is very rare. The thrush fungus, *Oidium albicans*, has been found in certain cases. Anthrax of the stomach has also followed anthrax of the mouth. The yeast fungus is not infrequently present, but rarely produces severe inflammation of the stomach. Cases which are in the nature of medical curiosities have been reported in which acute inflammation of the stomach was due to the action of maggots deposited about the mouth by the common fly, or swallowed with various articles of food. In all such instances the condition of the stomach has been profoundly depressed, as otherwise the ability of this organ to destroy invading micro-organisms and parasites prevents it from becoming infected.

CHRONIC GASTRITIS.

Definition.—By chronic gastritis is meant a state in which the gastric mucous membrane suffers from prolonged and persistent inflammation of a low grade, or repeated acute or subacute attacks, which result in more or less well-marked pathological changes. The term "chronic gastric catarrh" is often used as a synonym to describe this condition.

Etiology.—The causes of chronic gastritis are very numerous. In the majority of cases they consist in the frequent entrance into the stomach of irritating foods or drinks, as in alcoholics, or in persons given to the excessive use of highly seasoned foods. In these cases a very large part of the disorder in the gastric mucosa is also dependent upon the engorged portal circulation and the hepatic torpor which such dietary indiscretions induce. Cardiac diseases which lead to hepatic congestion very frequently induce gastric catarrh. Alcohol, bad food, or badly chewed food, hepatic cirrhosis, and congestion of the liver are, therefore, the chief causes of this condition.

Pathology and Morbid Anatomy.—The changes in the stomach in chronic gastritis may be divided into two classes: In the first there is a proliferation of the connective-tissue cells and formation of new tissue, which, like similar forms of overgrowth elsewhere, results in atrophy or degenerative changes in the gastric glands. In other words, the lesions are not limited to the superficial portion of the mucous membrane, but extend well down into the deeper layers. As the contraction of the overgrown connective tissue proceeds, it may cause the projection on the surface of the inner wall of the stomach of wart-like masses, so that broad, raised patches of mucous membrane are discernible, or even polypoid formations appear (gastritis polyposa). The entire gastric mucosa may be contracted or plicated.

In other cases the lesions appear to be more of a degenerative type; the sclerosis or fibrosis is inconspicuous, and the epithelial, or granular, atrophy is most marked. In other cases cystic alteration, with little fibrous hyperplasia, may be the dominant alteration. In the pyloric end of the stomach the contraction which follows the overgrowth of connective tissue may produce stenosis, a condition which is emphasized in some cases by the inflammatory process extending to the muscular layer of the stomach, by which means still greater thickening takes place. The closure of the pyloric orifice is, therefore, due to a true hyperplasia. The cause of the nipple-like projections found in the gastric mucous membrane in some of these cases is unknown. It may be due to the constricting influence of connective tissue formed between the tubules, or it may arise from overgrowth of the submucous coat of the stomach.

The *second type* of chronic gastritis is that characterized, not by overgrowth of connective tissue, as has just been described, but by wasting or atrophy of the glands. In some cases, however, there is at first some hyperplasia of connective tissue, and this is followed by atrophic changes. The mucous membrane becomes thin and smooth and is often pigmented, while the epithelial cells lining the gastric tubules suffer from atrophic, fatty or necrotic changes. The deeper tissues in some cases escape, but in others they also undergo wasting, so that even the submucous coat and the muscular coat atrophy. To this condition has been given the unfortunate, but etymologically correct, name of *phthisis ventriculi*. In such a case it is quite possible for gastric dilatation to develop.

Atrophic gastritis is much more rare than the hyperplastic type, and is often, if not always, associated with another grave condition, pernicious anæmia. Finally, in very rare instances, cases of atrophic gastritis may develop into ulceration of the gastric mucosa, the ulcers being small, round or irregular in shape, and rarely penetrating very deeply (erosive gastritis). They are found chiefly near the pylorus and may bleed very freely.

Symptoms.—The symptoms of chronic gastritis consist in *loss of appetite*, *impairment of the sense of taste*, and *nausea*, which is particularly prone to be present in the morning and may often amount to actual vomiting—the "*morning vomiting of the drunkard*." The vomited matters are but partly digested and are often mixed with much mucus. Most cases frequently *belch* *up gas*, and with it a mouthful of acid fluid may be brought up which scalds the pharynx. Hydrochloric acid may be lacking in the gastric contents, but in its place an excess of butyric and acetic acids is often present, particularly if the stomach is feeble and is unable to expel its contents into the bowel with sufficient promptness. Lactic acid is also present in some cases. It is the presence of these acids that causes heartburn, or pyrosis. In some cases, however, an excess, or at least a normal amount, of hydrochloric acid is secreted.

The *tongue* is moderately *coated*, the *bowels* are prone to *constipation*, and the general nutrition is slightly impaired, partly because of poor digestion, but chiefly because of the fact that the patient has cut off from his diet list one article after another, with the thought that it "disagrees" with him. There is not, however, an impairment of nutrition sufficient to cause great loss of weight in many cases, because the digestive function of the duodenum is not always impaired. If the liver is diseased, a very considerable loss of weight is usually present. *Digestion* is, of course, as is clear from a consideration of the state of the gastric mucous membrane, *greatly delayed* and *very imperfect*, and as a result the patient becomes inert, low-spirited, and vitally depressed, so that he presents the clinical picture of what is commonly called by the laity "a confirmed dyspeptic."

If the cause of the gastric disorder is alcoholic cirrhosis of the liver, the symptoms of cirrhosis are associated with those of gastric catarrh.

A constant, unproductive cough is often present without any lesions being found in the lungs

When the atrophic form of chronic gastritis is present, a very profound degree of anæmia is often developed, as already stated.

Diagnosis.—The separation of chronic gastritis from gastric cancer is by no means easy in many instances, for in many cases of cancer gastritis is also present. The presence of a mass, of considerable pain, of coffee-ground vomit, and an absence of HCl in the gastric fluids would point to the diagnosis of cancer; but as pain is not always present in cancer, as ulcers of the stomach may be present in chronic catarrh, giving rise to bloody vomit, and as HCl is often diminished or absent in this state, these signs are not entirely reliable. Lactic acid is not commonly present in large amount in chronic gastritis, but it is usually present in excess in cancer.

Prognosis.—The prognosis in a case of chronic gastritis must be given guardedly, for while one patient may speedily recover under proper treatment, other patients remain ill for long periods, even with the most skilful treatment. Much depends, too, upon the course of the disease and upon the general health of the patient. While, on the one hand, the malady does not cause death, on the other, complete recovery may seem impossible.

Treatment.—Theoretically, chronic gastritis may be prevented by avoiding the use of irritating and indigestible foods and alcoholic drinks, but, practically, patients are not seen until after the condition has been developed by the various causes which have been enumerated. After the condition has developed the treatment must be devoted to the removal of the habits which act as causes, to the relief of the conditions which exist in other portions of the body, and to the cure of the symptoms already present in the stomach.

In cases in which errors in diet exist these must be rectified, and if alcohol is used it must be stopped. If an examination of the heart shows that it is feeble, and that the gastric condition is due to an impaired circulation, rest and the use of moderate doses of digitalis must be resorted to, but it must not be forgotten that digitalis in full doses is capable of causing gastric distress. Usually it is necessary, in order to get the best results, to administer, every few days, small doses of blue mass, which not only unloads the liver, but seems to increase the efficiency of the digitalis.

The local treatment of the stomach consists in the employment of lavage, by means of which the excessive quantities of mucus and undigested food are removed. Emetics should not be employed, as they are too violent and apt to increase the inflammatory process. Not infrequently the mucus which is secreted is so thick and tenacious that there is some difficulty in removing it from the stomach. Under these circumstances, various medicinal substances may be added to the water which is employed for the lavage. A salt solution may be used, composed of ½ ounce of sodium chloride and 1 ounce of sodium bicarbonate, placed in a quart of warm water. After the stomach has been thoroughly cleansed, it may be washed a second time with boric acid, 1:100; salicylic acid, 1:1000; chloroform-water, 1:200; hydrochloric acid, 1:200. When chloroform-water is used, great care should be taken that the chloroform is thoroughly mixed with water and that the mixture is then allowed to stand for a sufficient time to permit of the separation of any excess of chloroform.

Lavage should be carried out, as a rule, not oftener than twice in twenty-four hours; in many cases once every alternate day is often enough. The best time to perform lavage is usually in the evening at about 9 o'clock, so that the stomach may have complete rest for the next ten hours. In those cases, however, in which the taking of food in the morning produces great distress, it is often advantageous to use lavage on first arising, in order that mucus may be removed.

In regard to *drugs*, it may be said that the one which has the greatest reputation is the nitrate of silver given in pills containing ¼ grain, or in solution in the dose of from ¼ to 1 grain to 2 drachms of one of the aromatic waters, as cinnamon-water or peppermint-water. Another drug which has a high reputation is the subnitrate of bismuth, which should be given in large doses, about 1 drachm twice or thrice a day. Both of these forms of treatment possess the disadvantage that they are constipating, and therefore the patient usually has to take a small dose of one of the mild laxative saline waters on first arising in the morning.

For the relief of loss of appetite and for absence of hydrochloric acid, the various simple bitters, such as cinchona, quassia, and cardamom, may be given. Of the compound tincture of cardamom, 1 or 2 drachms may be given once or twice a day with meals. If, in addition, it is believed that the stomach lacks motive power, strychnine may also be used, and the fluid extract of condurango may be given in the dose of a drachm three times a day. If digestion is delayed because of a lack of hydrochloric acid, 5 drops of this dilute acid may be given with each meal, combined with a good essence of pepsin. The administration of nitrate of silver one hour before meals usually diminishes pyrosis, or heartburn, but, if it does not, magnesium carbonate or bicarbonate of sodium may be used for this purpose. These alkalies frequently diminish pain by decreasing acidity.

The *diet* should consist of easily digested foods, and it is to be remembered that small meals given five or six times a day are better than large meals given three times a day. Chicken, beef, and mutton broths, free from fat and fortified by the addition of barley or rice, are exceedingly useful. If solids are taken, the patient must be instructed to chew both the meats and starches thoroughly. Often it is advisable to have the meat made tender by pounding it, or by cooking it in such a way that its fibres are readily dissolved by the gastric juice.

The digestion of starches, like baked potatoes, toasted bread, Zweiback, and pulled bread, should be aided by the use of taka-diastase or pancreatin. Often a capsule containing both of these digestive ferments will be advantageous in its effect.

Milk may be given to those with whom it agrees. In some instances, when it cannot be taken pure, it can be digested readily if diluted with some sparkling water, particularly Vichy water. In other instances the addition of a small quantity of salt aids in its digestion, and in still others lime-water may be given with it.

The question as to the use of the light wines by a patient suffering from chronic gastritis is debatable. If any fermentation is present, they must not be used. If patients are accustomed to drinking wine with each meal, it may be advisable to permit small quantities, particularly with luncheon and dinner. Champagnes are usually distinctly harmful.

GASTRIC DILATATION.

Definition.—By dilatation of the stomach is meant a condition in which this viscus loses its propulsive power to a greater or less degree and also undergoes a certain amount of dilatation, so that its capacity is increased. It is sometimes called " gastric ectasy," or " gastrectasis."

Etiology.—While it is true that dilatation is the state which impresses itself most forcibly upon the clinician when a patient is examined who is suffering from this malady, it is also a fact that the dilatation is always the result of some primary difficulty in expelling the contents of the stomach into the duodenum. In some instances this is due to stenosis of the pylorus produced by a thickening, as in chronic gastric catarrh; in others it may be due to what is called hypertrophic stenosis of the pylorus, and in still others the obstruction may be offered by a tumor at this point or by a cicatrix or other form of stricture. Rarely the pylorus becomes so glued to nearby tissues that it is held abnormally high, and is so fixed that it is almost impossible for the stomach to force its contents past the orifice.

A second cause of difficulty in emptying the stomach exists in a weakness, congenital or acquired, which so impairs the motor power of the viscus that it is too feeble to empty itself. Neither obstruction nor inherent weakness of the muscle fibres in the gastric wall are necessarily associated with dilatation, but it can be readily understood that these causes may so result. It is conceivable that in the obstructive cases the stomach may undergo some hypertrophy, and this takes place in a considerable number of cases as a primary result of the obstruction. The constant endeavor of the stomach to empty itself, however, ultimately causes fibroid changes in the muscle fibres from fatigue, and this condition is emphasized by impaired nutrition of the stomach, and perhaps by impaired nerve supply as well. Finally, it is undoubtedly true that, in some persons at least, the repeated distention of the stomach by large amounts of food and drink may cause permanent dilatation, particularly if these materials be of such a character that they produce chronic gastritis, and so impair the tone of the gastric walls. Workmen in breweries who partake of large amounts of beer, and diabetics who eat and drink to excess because of their disease, often suffer from gastrectasia.

Dilatation of the stomach is usually a disease of middle age or of adult life, but cases are not uncommon in children. The youngest case I ever saw was in a child of eighteen months. The dilatation due to obstruction is the type in which the greatest enlargement of the stomach develops.

Pathology and Morbid Anatomy.—The size of the stomach may be greatly increased, so that the average capacity of a quart (1000 c.c.) increased to even four quarts. Under these circumstances, the lower border of the stomach extends far below the normal level. Its walls are decreased in thickness, there is atrophy of the lining mucous membrane, and the muscular fibres are even more wasted, so that many of them disappear and are replaced by connective tissue. When primary atrophy of the muscularis has been present, it not rarely happens that an excess of muscle fibres are found in the pyloric region, although advanced secondary wasting has occurred elsewhere. In some cases of dilatation the gastric walls do not become thin, but may appear thicker than normal, because of an overgrowth of connective tissue which supplants the muscular layer of the organ.

Gastric dilatation does not always result in an equally well-developed increase in size. In some instances the cardiac orifice and the pylorus are near one another, so that the great curvature hangs like a plumber's trap; in other cases cicatrices distort it and even cause an hour-glass form, with a dilatation on either side.

Symptoms.—The symptoms of gastric dilatation are usually considered by the patient to be those of "chronic dyspepsia." There is usually loss of appetite, a sense of *gastric discomfort* and weight, or a feeling of *dragging down* in the abdomen and a good deal of *belching of gas*, which is often accompanied by some particles of food mixed with fluid. The sense of *distention* and *distress* gradually increases until it is almost insupportable, and then the viscus finding the burden too great, unloads itself by an attack of vomiting, in which the patient is surprised to find *articles of food ingested*, perhaps, *several days before*. Both the physician and the patient are, not rarely, amazed at the quantity expelled, for the volume shows that it represents the ingested fluids and solids of several days. Such an attack of vomiting, in which the quantity expelled is far in excess of the amount recently swallowed, is a very important diagnostic point. These attacks of vomiting usually occur at night. When the dilatation is severe, so that the stomach cannot completely empty itself, the relief given by vomiting is only partial, and perhaps no relief follows.

The *bowels are constipated*, and the *stools* when passed are *scanty*, because so much of the food ingested is not passed on into the duodenum. The *urine* is also *scanty*. Not rarely it is decreased to one-third the normal quantity.

Many of the symptoms are due to stasis of the food in the stomach, fermentation, and the absorption of toxic materials from bacterial growth.

In cases in which bile appears in the urine, the cause of the dilatation probably does not depend upon gastric dilatation alone, but upon some obstruction in the duodenum, which dams back the food in the pyloric orifice and so forces the stomach to undergo distention.

The *physical signs* of gastric dilatation are as follows: On *inspection* in some of these cases, it may be possible to outline the stomach if it is distended with food and gas. This determination of its area and limitation is, however, much better accomplished by percussion after the stomach has been emptied by the use of the stomach tube and then has been distended by gas. This distention may be produced either by giving the halves of a Seidlitz powder separately (or by the use of 30 grains of tartaric acid in one-half glass of water and 2 drachms of sodium bicarbonate in another half-glass of water), or by introducing a stomach tube, attaching a Davidson syringe to it and then pumping air into the stomach until it is distended. This latter plan is probably the safer of the two if ulcer is supposed to be present, but if the patient is not accustomed to the use of the tube its presence causes so much retching and gastric unrest that it is usually impossible to make a satisfactory examination of the true area of gastric tympany.

The tympanitic note produced by the *percussion* of a stomach so distended very clearly outlines it in many cases. If there is doubt as to the presence of gas in the colon, which may cause tympany, the large intestine should be filled with fluid, by the injection of a large clyster, when the areas of gastric tympany and intestinal flatness on percussion can be readily defined. In other cases the stomach may be filled with fluid, and, if need be, the bowel filled with air to develop the same outlines.

It has been held by some clinicians that the use of carbonic acid for the purpose of dilating the stomach for diagnostic purposes is dangerous, but when we consider the hundreds of instances in which it has been used in every part of the world without evil effect, we must conclude that it rarely does harm. Behrend has recently reported 3 cases, however, in which death followed its use, the patient in one instance suffering from a profuse hemorrhage, another patient bringing up froth and blood, and the third, which did not die for five days, suffering from great distress and prostration. Wharton and Musser have reported a perforation of the stomach after drinking a glass of carbonated water. All of these cases were complicated by gastric ulcer.

Auscultation of the epigastrium may reveal splashing, or succussion, in many cases of gastric dilatation, but this sign should never be regarded of very great import, for not rarely the same sound is produced by fluid in the bowel.

Some years ago Einhorn invented the electric light method of diagnosticating dilatation (*gastrodiaphany*). This consists in filling the stomach with water and then passing a small electric light into the stomach, the patient and physician being in a dark room. If the abdominal wall be not too thick, the area of light can be readily outlined and the size of the stomach determined. The efficiency of this method of determining the size of the stomach can be much increased by the use of fluorescent media. The best of these is developed by the use of two solutions. One solution consists of bicarbonate of sodium, 40 grains, dissolved in 1 pint of distilled water. The second is composed of the same ingredients plus 2 drachms of glycerin and ¼ of a grain of fluorescin to the pint. Before the test is used the patient should take 2 grains of quinine three times in a day. He then swallows ½ pint of solution No. 1 and ½ pint of solution No. 2, when, on the introduction of the gastrodiaphane, the illumination of the stomach becomes very marked.

Another means of diagnosis is the use of the x-rays after the patient has received a large dose of bismuth subnitrate (2 to 4 ounces). By this means the area of the stomach can be determined. The bismuth must always be washed out after the test to prevent poisoning.

Another aid is Turck's gastric sound, the end of which can be felt through the abdominal wall, if it is thin, as it is moved about in the stomach.

The use of drugs, which are dissolved only in the intestine, to test the motor power of the stomach is of some diagnostic value. Salol, for example, is given in the dose of 15 or 20 grains, and the urine tested after five hours for salicyluric acid by means of the perchloride of iron test, which consists in adding tincture of iron chloride to the urine, when, if this acid is present, a purple color is obtained. When dilatation is present there may be no response for twenty-four hours.

Finally, as a means of determining that the digestive power of the stomach is greatly impaired, a test meal should be used after the stomach has been cleaned by lavage. If dilatation is present, the digestive process will always be very slow and imperfect.

Cases of gastric dilatation sometimes develop a state called *gastric tetany*, in which tetanic spasms develop in the extremities. This is preceded by a sensation of formication, or numbness, associated with drowsiness. It has ensued, as a rule, upon the employment of lavage. Following the sensory symptoms the patient is seized with violent vomiting, and after or during this attack of emesis the muscles of the thumb and fingers contract, so that the thumb is drawn into the palm of the hand and the fingers are flexed. The wrist is also strongly flexed, but it may be extended. The forearm is flexed on the arm, and the biceps is hard and tense. These positions may not be maintained, but be changed into extension. Both sides are involved, but one side usually suffers more than the other. When the legs are affected the toes are flexed and the knees bent. The facial muscles may be in spasm and the patient may have explosive speech, as if in a shouting delirium. If the affected arm be pressed upon over the course of its vessels or nerves, the attacks may be reproduced (Trousseau's sign); if the point of exit of the facial nerve be tapped, facial spasm develops (Chvostek's sign); if electricity is used it is found that the muscles are excessively irritable (Erb's sign). The cramp-like contractions are painful. In severe cases death may occur from exhaustion. Out of 101 cases reported by European clinicians, no less than 75 died.

Although the employment of the stomach tube has induced attacks of gastric tetany in some cases, attacks not due to this cause are to be prevented by frequent and thorough lavage, and are to be combated, when present, by nerve sedatives such as morphine or hyoscine hypodermically. Their occurrence in a mild form urges upon the physician the need of operation for the gastric state.

Gastric tetany occurs in cases of dilatation more frequently than in cases of ulcer.

Diagnosis.—As a rule, the diagnosis of gastric dilatation, in its well-developed stage, is not difficult. Care must always be exercised, however, that dilatation and gastroptosis are not confused, for in both affections the lower border of the stomach may be found far below the normal level, particularly if it is distended with liquid or gas. The use of any of the methods of percussion and palpation, or the other means of diagnosis just described, will speedily separate the one state from the other, and the relatively limited capacity of the stomach in ptosis and its large capacity in dilatation will be another factor in deciding upon the real state which is present.

Treatment.—The treatment of gastric dilatation is not promising unless the patient is seen and his condition recognized in the early stages of the disease. At this time, and later on as well, his diet should be most carefully regulated. He should be instructed to avoid all fatty articles of food which may give rise to lactic and butyric acid fermentation, and should also avoid the use of sweet materials, which may also undergo fermentative changes. The food which he takes should be thoroughly masticated and insalivated, it being remembered that the saliva is an important digestive juice, and that much may be done in aiding the digestion by thoroughly moistening the food with this secretion. The patient must also be warned not to eat a large amount of food at any one time, but rather to subsist on four or five small meals a day. He should also be instructed not to take large quantities of liquids with his meals.

As to the articles of diet, he may have beef, mutton, chicken, or other simple varieties of meats, broiled or roasted, but not fried. Potatoes should only be taken when baked, and then in moderation. Zweiback, or soda biscuits which have been once more cooked by pouring scalding water over them, may be taken in moderation. The digestion of the starches should always be aided by the simultaneous ingestion of a capsule containing 2 grains of taka-diastase and 2 grains of pancreatin. If the diet is largely a meat diet, digestion should be aided by the use of hydrochloric acid and pepsin, 5 to 20 drops of the dilute acid and 2 teaspoonfuls of a good essence of pepsin being used. Often it is wise to add to this mixture 10 drops of the tincture of nux vomica, or $\frac{1}{20}$ of a grain of strychnine, for its effect as a bitter tonic and for the purpose of improving, if possible, the activity of the stomach.

In no case should the patient be allowed to accumulate fluid and food in the stomach for more than twenty-four hours. In other words, we should not wait until nature relieves the stomach by an attack of vomiting.

As in chronic gastric catarrh, lavage should be performed once in every twenty-four hours, preferably at night before going to bed, and in all cases it should be remembered that it is not only futile but harmful to introduce food into a stomach which is already partly filled with fermenting materials, and which is therefore incapable of dealing with new food, which if taken simply adds to the decomposing mass already present.

In many of these cases it is advisable, after emptying the stomach of its contents, to wash it out with one of the solutions named in the article on the Treatment of Chronic Gastric Catarrh.

Emptying the stomach by means of the stomach tube and forbidding the use of excessive quantities of food are not only advantageous in that they permit digestion, poor as it may be, to proceed, but also do good in that they prevent the stomach from being overloaded and distended, and so further dilated, by its contents. It seems hardly necessary to add that beer, sweet wines, and champagnes should be absolutely forbidden for such patients.

The medicinal treatment of gastrectasy consists in the use of nux vomica or strychnine in full doses combined with physostigma, the object being to improve the tone of the muscular fibres of the stomach. A pill composed of ½ grain of extract of nux vomica and ⅙ grain of extract of physostigma may be given three or four times a day with advantage. Gastrointestinal antiseptics are usually not particularly useful. From 2 to 5 minims of creosote or guaiacol may be given in capsule one or two hours after eating, with the hope that they will stop fermentation. In other instances guaiacol carbonate may be given in the dose of 2 to 5 grains in capsule. In still other instances naphthol may be given in the dose of 2 to 5 grains in capsule three times a day, or benzonaphthol may be used in the dose of 10 grains, in the same manner.

Many physicians of experience resort to the use of faradic electricity in these cases, introducing a stomach tube containing the positive electrode and applying the negative electrode to some point on the surface of the body.

In cases in which the dilatation is so severe and the symptoms so distressing that none of the measures so far suggested give adequate relief, the question of operative interference must be considered. Under these circumstances, the question as to the cause of the dilatation becomes an important factor. If it is dependent upon pyloric stenosis, a gastroenterostomy or pylorectomy is indicated. But if, on the other hand, it does not depend upon this cause, but upon inherent atony and failure of the gastric walls, pylorectomy is, of course, of little value, and a gastroenterostomy is indicated. Before proceeding to operation, however, it must be remembered that there is some difference between an operative recovery and benefit to the patient. Rarely in these cases the patient survives the operation and makes a surgical recovery, but abdominal discomfort persists, either because of the presence of adhesions, irritation of nerve fibres, or other causes which it is difficult to determine, and which may be dependent upon the altered course of food from the stomach to the bowel.

Acute Gastrectasis.—Under the names acute gastric dilatation, gastro-intestinal paralysis, atonic gastrectasis, toxic gastrectasis, and paralytic dilatation of the stomach, there occurs an acute, often rapidly fatal, dilatation of the stomach alone or of the stomach and intestines.

Etiology.—Some cases are apparently causeless, and even at autopsy no cause may be demonstrable. Others occur in the course of acute infectious processes, some of which are systemic, such as scarlet fever and typhoid fever; in others the lesion is some distance from the affected viscus, as in pneumonia and meningitis, while still others depend for their development upon infectious processes in the neighborhood of the stomach or intestine, conspicuous among which may be mentioned peritonitis. The condition occasionally follows surgical anæsthesia, and it has been suggested that swallowing of mucus saturated with the anæsthetic may be the cause in some cases. It has been attributed to acute pyloric obstruction, as by foreign bodies or spasm, but that this is not always the cause is shown by the reported instances in which dilatation extended through the pylorus and first and second parts of the duodenum or even into the ileum. It has been thought to depend upon obstruction of the duodenum by the superior mesenteric vessels. It sometimes follows operation involving the peritoneum, and may commence after labor. Reynier strongly urges the influence of the nervous system in the production of acute gastrointestinal paralyses.

Morbid Anatomy.—At autopsy the stomach is large, thin, and flaccid; it may extend almost to the pubes. It contains gas and fluid; the latter may be thick and viscid, but it is usually thin, watery, greenish or occasionally brownish in color, and frequently contains flocculi. The gastric mucosa may weep blood and the vessels be widely distended.

Symptoms.—These usually come on rapidly. In operative cases they may be delayed twenty-four to forty-eight hours. There is *marked abdominal distention* amounting to *actual ballooning;* the dilated organ occupies the middle and upper left areas of the abdomen, and may be outlined through the abdominal wall. *Peristaltic waves* are rarely recognizable. *Vomiting* is nearly always present. In the few reported cases in which vomiting has been absent, it has been suggested that the associated relaxation in the abdominal wall has rendered emesis impossible. The vomited fluid is thin, watery, greenish or brownish. *Profound depression* or symptoms bordering on *collapse* quickly appear. The *pulse is small, rapid,* and *weak;* the *respirations* shallow and frequent; the *temperature,* in the absence of complications, is usually low and may be subnormal. *Thirst is intense,* and, on account of suppressed absorption and prompt vomiting, is unrelieved by drinks. The *urine is scanty* or even *suppressed.*

Diagnosis.—The acuteness of the symptoms and rapidly progressing collapse differentiate the condition from chronic dilatation. The vomiting is more incessant and the pain less than in volvulus of the stomach, which in some respects it closely resembles. The gastrorrhœa that accompanies it and the character of the vomit is unlike acute indigestion, and, ordinarily there is no expulsion of fragments of the mucosa as in true toxic gastritis The relaxed abdominal wall is quite unlike the rigid wall of peritonitis.

Prognosis.—The mortality is high; in Herff's series of 34 cases, 29 died. In the so-called reflex group—those unassociated with any intra-abdominal lesion—prompt treatment promises some relief.

Treatment.—Its prevention after operation may be accomplished by lavage immediately at the end of anæsthesia, and if vomiting appear and persist, lavage should be repeated. As it is probable that the condition is of toxic origin, free lavage should repeatedly be practised, and the stomach kept empty by the frequent use of the stomach tube. Water and food had best be given by enema. Strychnine and atropine have been suggested. As early as possible saline purgatives should be administered in small, but often repeated, doses. As relapses are possible as late as the third day, feeding, and even the administration of fluids, must be begun most cautiously in cases fortunate enough to survive.

GASTRIC ULCER.

Definition.—Ulcer of the stomach, often called peptic ulcer, or *ulcus ventriculi*, is due to necrosis of a part of the mucous membrane of this organ, so that an exposure of the submucous tissue is present.

Etiology.—Almost ever since the processes of gastric digestion have been known, animated discussions have arisen as to why the stomach is not digested by its own juices and a large number of explanations have been offered, many of which have been anything but satisfactory. At present the conditions which result in gastric ulcer are known to be closely connected with the inability of the gastric mucosa to resist the action of the gastric juice. If, by any cause, the vital resistance of the mucous membrane is impaired, at the point of greatest impairment an ulcer may be developed. In very rare instances an injury to the surface of the abdomen may extend, or be transmitted, deeply enough to cause a lesion in the gastric wall; but it is more common for injuries to occur by internal agents, as by the use of certain articles of food which may interfere with the circulation in the wall of the stomach, as, for example, boiled tea, taken very hot, which contains an excess of tannic acid. So, too, an embolus or thrombus in a branch of a gastric artery may deprive an area of its blood supply, and subsequent digestion remove the dead tissue and so form an ulcer. Another predisposing cause of ulcer is the secretion of superacid juice or of an excess of ordinary juice, and finally, in some cases, a local necrosis of the tissues is produced by the entrance of infecting micro-organisms, which, however, cannot enter the mucosa if normal vital resistance is maintained.

Frequency.—The frequency of gastric ulcer in some parts of the world is far greater than in others. Even between England and the United States the difference is extraordinary. Out of 59,762 medical cases in the London hospitals, there were 1649 cases of gastric ulcer; while out of 75,612 medical cases in hospitals in different cities in the United States, there were only 446 cases. According to these figures the morbidity of gastric ulcer is more than four times as great in England as it is in the United States. Since these figures were compiled Howard has confirmed them by others.

In regard to the relationship of age and ulcer statistics vary slightly, but those of Welch are still to be considered the most competent. He found that the largest number of cases of ulcer occurred between twenty and thirty years of age, and Lebert also found that seven-tenths of 252 cases were between twenty and forty years of age. A case of ulcer in an infant only thirty hours old has, however, been recorded by Goodhart. Cutler, in an exhaustive search in literature, found only 24 cases under ten years of age with autopsy and 2 without autopsy, and has added 3 more which occurred in the Massachusetts General Hospital; 29 in all.

Women suffer from ulcer far more frequently than men. This is shown by all statistics and is illustrated by the following figures: Of 1548 cases of gastric ulcer collected from the official reports of hospitals in the United States and England, 1273 occurred in women and 275 in men. Of 1699 cases examined postmortem and studied by Welch, 1020 were in women and 679 in men. Cantlie states that out of 20,586 cases in Montreal there were 85 cases of gastric ulcer, and of these 82 were women. The average age was twenty-seven and a half years.

Other etiological factors of interest are occupation and associated disease. Thus, seamstresses and servant girls are singularly prone to ulcer, as are also tailors and shoemakers. Such persons are usually chlorotic or anæmic. So, too, ulcer is sometimes a complication of tuberculosis, and it may be in itself tuberculous.

Gastric ulcer may be divided into four classes: In the *first* the lesion is very mild, the mucous membrane being eroded in such a manner that its superficial epithelium is destroyed. All authors do not agree, however, that these erosions are a form of peptic ulcer. The *second type* is characterized by an ulcerative process which penetrates more deeply, so that the submucous tissues are affected. The *third* invades the submucous, muscular, and even the peritoneal coat, and may cause perforation. The *fourth type* is that in which as a result of cicatrization and contraction scars and deformities develop, which produce serious consequences.

Pathology and Morbid Anatomy.—Gastric ulcer is usually single, but cases are not very rare in which the ulcers are numerous. When acute it forms rapidly and presents a peculiar punched-out appearance. In the usual chronic form the edges are more shelving, indurated, and not so sharply defined. The size of the ulcer varies from a small spot scarcely larger than a pinhead to an enormous excavation covering nearly two-thirds of the gastric surface. These large ulcers are, however, very rarely met with.

The depth to which the ulcerative process extends is also variable. The mucous membrane nearly always suffers most, but the tissues beneath it are affected as well, and the destructive process may, as just stated, extend as far as the peritoneal coat. Undermined ulcers are extremely rare. Around the edge of the ulcer there is usually marked hyperæmia, and the surrounding tissues, especially in chronic ulcers, are often infiltrated by formative cells or by the development of connective tissue. Usually the rest of the stomach exhibits more or less marked chronic gastritis.

Ulcer of the stomach is usually found on the posterior wall of the viscus near the pylorus and on the lesser curvature (75 per cent.), probably because this is the part of the stomach which carries out the grinding process and urges the food into the duodenum, and therefore is exposed to injury and abrasion. Armstrong has, however, analyzed 240 cases of gastric ulcer and found the anterior wall affected in 125 cases, the posterior wall in only 32.

If the healing process is not rapid enough to arrest the ulcerative process, the wall of the stomach may be perforated and so produce severe abdominal symptoms. More commonly, however, as the inflammatory process approaches the surface of the stomach it causes this viscus to become glued to a neighboring organ, and so it happens that the floor of the ulcer may be formed by an adjacent viscus. In this way neighboring organs may be involved in the inflammatory and septic process, and not rarely subphrenic abscess is due to this cause. Sometimes a perforation takes place into the colon or duodenum, and cases have been recorded in which the pericardium and pleura have been involved in this manner. The liver is also sometimes infected. Fenwick, in an analysis of 127 cases, found the stomach adherent to the pancreas in 49, to the liver in 33, and to the liver and pancreas in 10.

Fig. 79

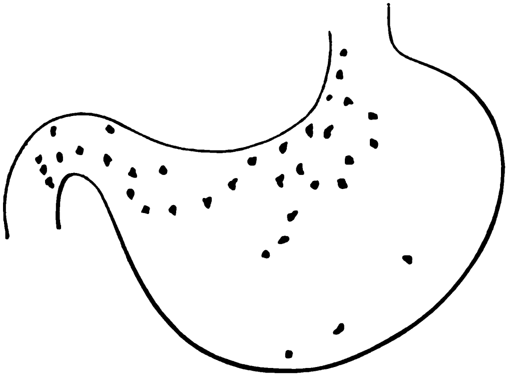


Diagram showing the situation of ulcers of the stomach on the lesser curvature and near the pylorus. (Modified from English.)

Ulcers on the anterior surface of the stomach are less common, but more prone to perforation into the peritoneum than those situated posteriorly. It is held that the anterior wall is more movable than the posterior, and hence time for adhesion to apposed tissue is less.

In many cases, a tendency to healing asserts itself and gradually, the exposed tissues are healed by the formation of a cicatrix which may cause considerable puckering as it develops.

If the ulcer has been near the pylorus this may cause pyloric stenosis, or if it be near the middle of the stomach and the ulcer has been extensive an hour-glass contraction may result. (See Hour-glass Stomach.)

Symptoms.—Ulcer of the stomach, at least in its milder forms, may exist for years without its presence being suspected, the patient suffering from a train of moderate gastric symptoms, generally described as dyspeptic. In most cases, however, it makes its presence known by symptoms which sooner or later send the patient to her physician for relief. The symptoms now complained of are *discomfort* and *pain after eating*, with a *constant gnawing* between meals when the stomach is empty. Not infrequently this gastric distress is relieved by taking some food, and then increases as an excess of gastric juice is poured out to digest the food. The pain, when characteristic, is peculiar in its distribution, for it radiates from the epigastrium back to the shoulder-blade, or to a spot between the shoulder blade and the spine. Head has also shown that in gastric ulcer there is an area of cutaneous hyperæsthesia in a small triangular spot in the left epigastrium. This is demonstrable by a light touch, and not on deep palpation. At times the pain is exceedingly severe, and it may require active medication because of its intensity. Oftentimes the patient attempts to find relief by lying on the stomach or placing a pillow against it, but as a rule the epigastrium is so tender that any pressure on the part of the physician makes the patient wince. Careful palpation may, however, reveal an area of thickening or induration, if the ulcer is a chronic one. Associated with these symptoms there is often *vomiting* of very acid fluid, and an examination of the gastric contents will show an *excess of hydrochloric acid* both as to percentage and actual quantity. *Constipation* is usually *marked*, and the *urinary flow* is *scanty*.

It is important to remember that ulcer of the stomach does not by any means always cause the same train of symptoms. Attention has already been called to the fact that the symptoms may be latent. In other cases there may develop, with great suddenness, a profuse *hæmatemesis* or symptoms of *collapse* from perforation, and one of these accidents may be the first symptom of any importance. In other instances there is a *general failure* of *health*, *marked emaciation*, and a development of *profound anæmia*. In still others *violent neuralgic pains* (gastralgia) are the chief manifestations. In some instances the disease lasts but a few weeks; in others it is prolonged for years.

The symptoms so far described are chiefly those of acute or subacute ulcer. *Chronic ulcer*, on the other hand, may produce none of these symptoms when the patient presents herself for treatment. Beyond a history of *gastric distress*, which may have existed for many years, there may be no pain on pressure and no soreness, in the sense of tenderness. Indeed, the symptoms may be those of gastric dilatation, or of pyloric stenosis. The patient is emaciated by reason of voluntary starvation, to decrease discomfort, and by the loss of food by vomiting. So, too, a cicatrix near the middle of the stomach may produce an hour-glass stomach. This may become evident on distending the stomach with gas or fluid, but it is to be recalled that there is danger that rupture may ensue from this practice. (See Pyloric Stenosis and Dilatation of the Stomach.)

In no other disease, save pernicious anæmia, is there such a notable diminution of red blood cells as takes place in many cases of chronic gastric ulcer. This is due to the more or less constant loss of blood which escapes by the bowel, the loss of which is usually not recognized. When hemorrhage does not occur great anæmia is rare, although the patient may appear pallid.

When *hemorrhage from the stomach* takes place the blood may be vomited or be passed by the bowel. The hemorrhage may follow many weeks of suffering or it may be the first sign that the gastric mucous membrane is diseased. The quantity of blood lost may be very small or so arge as to almost exsanguinate the patient, the variation depending upon the size of the bloodvessel which is eroded. If a large vessel is perforated by a small ulcer it is not difficult to understand why it is that hemorrhage may be the first symptom. In other words, the hemorrhage may be the first symptom of ulcer. On the other hand, not infrequently gastric hemorrhage, particularly if it be from a chronic ulcer, may be so scanty as never to cause bloody vomiting, the small amount of blood escaping with the food into the bowel. Moynihan believes that all ulcers bleed at some time n their existence. (See Diagnosis.)

In studying the question of *perforation* of gastric ulcer it is well to recall that this accident may or may not be preceded by symptoms which will serve as a warning to the physician if not to the patient. There is, in some cases, a progressive increase in discomfort and pain after eating, a greater degree of tenderness or pain over the epigastrium, and more frequent vomiting. When such signs are present the patient must be placed at absolute rest, and if the symptoms do not speedily become modified operation must be considered. On the other hand, the literature on this subject contains cases in which the history of gastric disorder was entirely absent, and the patient was suddenly seized by symptoms of perforation.

When perforation does develop, it is usually in the anterior gastric wall, and from what has been said of the various ways in which perforation of the stomach occurs, it must be evident that the symptoms may vary over a wide range of severity. When no inflammatory adhesions have been formed and the gastric contents escape suddenly into the general peritoneal cavity, the onset is, of course, *startling in its acuteness* and the *pain* is exceedingly *severe*, but the locality of the pain is frequently far removed from the area of the accident. *Vomiting* and *collapse* may soon develop, and *general peritonitis* begins if operative relief is not promptly given.

If the perforation is more gradual the symptoms are less violent and the opening may at first be so small that only a little of the gastric contents escapes into the peritoneal cavity. In those cases in which adhesions have formed before perforation takes place a subphrenic abscess may result. In such instances the perforation is usually on the posterior wall of the stomach.

Sometimes perforation of the stomach may take place without the sharp and decisive symptoms just described.

While the pulse usually is rapid it may not be materially increased in rate.

Again, it is important to remember that after perforation of the stomach there may be a "period of repose," or "fallacious calm," during which time the patient feels less pain and distress, and the pulse approximates its normal speed.

At one time, it will be recalled, a decrease in the area of liver dulness was supposed to be indicative of gastric or intestinal perforation, but we now know that the absence of this sign does not negative perforation. Thus, Pearson found a decrease in the area of liver dulness in 33 per cent. of 140 complained of are *discomfort* and *pain after eating*, with a *constant gnawing* between meals when the stomach is empty. Not infrequently this gastric distress is relieved by taking some food, and then increases as an excess of gastric juice is poured out to digest the food. The pain, when characteristic, is peculiar in its distribution, for it radiates from the epigastrium back to the shoulder-blade, or to a spot between the shoulder blade and the spine. Head has also shown that in gastric ulcer there is an area of cutaneous hyperæsthesia in a small triangular spot in the left epigastrium. This is demonstrable by a light touch, and not on deep palpation. At times the pain is exceedingly severe, and it may require active medication because of its intensity. Oftentimes the patient attempts to find relief by lying on the stomach or placing a pillow against it, but as a rule the epigastrium is so tender that any pressure on the part of the physician makes the patient wince. Careful palpation may, however, reveal an area of thickening or induration, if the ulcer is a chronic one. Associated with these symptoms there is often *vomiting* of very acid fluid, and an examination of the gastric contents will show an *excess of hydrochloric acid* both as to percentage and actual quantity. *Constipation* is usually *marked*, and the *urinary flow* is *scanty*.

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Diagnosis.—Gastric ulcer in some instances is so manifestly present that there is little difficulty in determining the cause of the illness. Care must be taken that the pain of appendicitis, gallstone colic, renal colic, and intense menstrual colic is not taken for that due to perforation. Moynihan speaks of 3 cases operated upon for perforation in which menstruation was the cause of the pain, and states that of 49 cases of perforated duodenal ulcer appendicitis was thought to be the cause of illness in 18. In the gastralgic cases ulcer must be separated from ordinary gastric neuralgia by the recollection of the fact that true gastric neuralgia is very rare, by the additional fact that the pain of neuralgia is not induced by taking food and by the fact that in neuralgia evidences of gastric indigestion are not constantly present, as they are in most cases of ulcer. Severe pains in the stomach due to locomotor ataxia (gastric crises) can usually be excluded by the presence of Argyll-Robertson pupils, absence of knee-jerks, and swaying when the patient stands with the eyes shut.

The irritation produced by gallstones may produce symptoms resembling gastric ulcer, but in these cases the history of gallstone colic will be given and the taking of food has no influence upon the pain. Then, too, the pain in the back due to ulcer is to the left of the middle line near the twelfth dorsal vertebra, whereas that due to gallstone is on the right of the median line and a little lower down. So, too, palpation of the neighborhood of the gall-bladder may reveal an enlargement of this viscus, and jaundice points to cholelithiasis rather than to ulcer.

Cases of chronic ulcer of the stomach with much cicatricial tissue around the ulcer, or at the seat of an ulcer which has healed, may present symptoms almost identical with those of gastric cancer. Pain and obstruction to the passage of food through the pylorus, gastric dilatation due to this latter cause, and emaciation from all these causes may combine to present a clinical picture of gastric cancer, particularly if the physician finds, on palpation, that he can feel a mass or masses in the gastric wall. The comparative youth of the patient in cases of ulcer, the absence of cachexia even if anæmia is marked, and a remembrance that ulcer of the stomach is most common in the female sex helps to make the diagnosis possible. Again, in ulcer the gastric contents show an excess of hydrochloric acid after a test meal, whereas in cancer this acid is usually absent or less than normal. (For the tests of the stomach contents see article on Gastric Cancer.)

Finally it must be recalled that duodenal ulcer may cause symptoms so closely resembling gastric ulcer that a differentiation may be impossible. If hemorrhage occurs, and blood is passed in the stools in considerable quantity, the lesion is probably duodenal. (See Duodenal Ulcer.)

Boas considers the demonstration of minute quantities of blood in the feces to be of great value in the diagnosis of gastric ulcer, and especially in determining whether obscure cases of gastric disease are pure gastralgia or conditions in which the symptoms are produced by ulcerative lesions of the gastric mucosa. It has been proven experimentally that the ingestion of so small a quantity as 3 c.c. of blood gives a positive reaction for blood in the feces, and this fact shows that even minute hemorrhages may give rise to similar positive reactions.

Before performing the test all other sources of hemorrhage, such as the swallowing of blood from wounds in the mouth or from lesions in the respiratory tract must be guarded against, and the absence of hæmaturia, and in women metrorrhagia and menstrual blood, must also be assured. No rare meat, fish, or sausages are allowed for two days before the test is made.

Boas prefers the aloin test as recently recommended by Klunge and Schaer to the older guaiacum test. It is performed as follows: 5 to 10 grams of feces, which, if hard, are to be softened by the addition of a small quantity of water, are mixed with 20 c.c. of ether, 3 to 5 c.c. of glacial acetic acid, and the mixture is well shaken in a reagent glass. Then more ether is added, and also 20 or 30 drops of an old oil of turpentine. If to this mixture there now be added 10 or 15 drops of a solution made by dissolving in 3 to 5 c.c. of 70 per cent. alcohol as much aloin as can be taken up on the tip of a small spatula, a light-red color is soon produced if blood is present. This light-red gradually assumes a cherry-red hue if the mixture is allowed to stand. If no blood is present the mixture remains of a yellow color for from one to two hours, when it changes to rose red.

The possibility of parenchymatous gastric hemorrhage, and of hemorrhage from varicose or atheromatous bloodvessels in the œsophagus or stomach, must of course be taken into consideration before a diagnosis of doubtful cases is made.

In several cases Boas has found blood present in the feces shortly after patients had experienced an attack of pain in the epigastrium. After several days of mild diet it was often found that no blood could be detected in the feces.

Prognosis.—The prognosis of cases of gastric ulcer must, of course, vary greatly with the severity of the lesion, and the time during which it has lasted. In those cases in which superficial erosions are present, the patient probably recovers in the great majority of instances. When actual ulceration is present, the proposition is, of course, a different one. The most divergent views exist as to the prognosis in this type of case. Brinton, in his now somewhat ancient statistics, states that 50 per cent. recover under medical treatment. Tricomi places the percentage at 25, as do also Debove and Ramond. Leube states that 25 per cent. die as a direct effect of the disease. Whatever may be the percentage as to recovery, it cannot be doubted that in many instances it is once an ulcer always an ulcer, in the sense that relapses take place soon after the ulcer seems well. In a collection of 500 cases at the London Hospital, made by Bulstrode, 211 had had ulcer before, 18 per cent. died, and 42 per cent. were not cured on discharge.

Ulcers near the pylorus heal more slowly than those which occur elsewhere. Some gastric ulcers probably become cancerous in later life. Graham has found a history of ulcer in 60 per cent. of 125 cases of cancer of the stomach—a fact of some prognostic importance.

The mortality in cases which suffer from hemorrhage is not high. The direct mortality from this cause is only 2.1 per cent., according to Russell. So far as recovery from the ulcer is concerned when it is severe enough to cause hæmorrhage the outlook is not good—44.7 per cent. continued in ill health and 42.6 per cent. recovered (Russell).

Treatment.—The treatment of gastric ulcer consists in restricting the diet, in the administration of medicines qualified to improve the state of the gastric mucous membrane, and in the institution of rest for the general system as well as for the stomach. Foods which are very hot or very cold, particularly those which are very hot, should be carefully avoided, and hyperacidity is to be counteracted by the use of calcined magnesia or bicarbonate of soda.

℞.—Sodii bicarbonatis,
Magnesiæ ponderosæ,
Calcii carbonat. . . . . āā ℥j.
Ol. menth. piperitæ . . . . ♏x.
Sig.—A heaping teaspoonful in half a glass of water when needed

In most instances it is wise to insist that the patient remain in bed for a period of three or four weeks, during which time the rest cure may be instituted in a modified form, since with the improvement in general health and the cure of anæmia healing of the ulcer progresses more rapidly.

The diet should consist of milk which is predigested by a peptonizing powder either before or immediately after it is taken into the stomach. Under certain circumstances it may be permissible to give the patient very soft milk-toast in small quantities, or to give scalded soda-biscuits digested by means of taka-diastase or pancreatin. When the ingestion of food increases gastric pain and distress it may be necessary to give the patient nothing by the stomach for a period varying from one or two days to two weeks, and to nourish him as far as possible during this period by nutritive enemata, which should consist of four ounces of peptonized milk and one egg, injected three times in the twenty-four hours, the bowels being carefully washed out with normal saline solution before each injection in order that the residue from the previous injection may be removed.

For the purpose of relieving thirst, it is often advantageous, when the stomach is irritable, to give, daily, normal saline solution by hypodermoclysis. In other instances a pint to a quart of normal saline solution may be given high up into the colon.

A number of years ago the late J. M. Da Costa strongly recommended ice-cream as a diet for these cases. Care should be taken that the ice-cream does not contain too much sugar, as this will cause fermentation and distress. Frozen milk flavored with vanilla is better.

The patient suffering from gastric ulcer should receive, in the way of medicine, ¼ of a grain of nitrate of silver with ½ of a grain of extract of hyoscyamus in pill three or four times a day and take them an hour before taking food, in order that these drugs may act upon the stomach, exercising a healing influence, and preventing an excessive secretion of gastric juice. If pain is severe, opium may be substituted for hyoscyamus, but there is some evidence that opium does not decrease, and may sometimes increase, the flow of gastric juice. In other instances chloretone in the dose of from 3 to 5 grains may be given three or four times a day, and it is particularly useful if pain is present. In other cases hyperacidity and pain are best controlled by 10 to 20 grain doses of bromide of strontium or of sodium.

The administration of massive doses of bismuth subnitrate has been strongly recommended, chiefly by Fleiner. He gives 150 to 300 grains, stirred in 5 or 6 ounces of warm water, after the stomach has been carefully cleansed by lavage. Of course a heavy precipitation of bismuth occurs upon the gastric mucous membrane. This plan is more suitable for chronic cases than for acute ones.

In all cases of gastric ulcer it is important that the bowels should be moved every day or two by means of one of the alkaline purgative waters, of which probably Carlsbad water has the greatest reputation. The Carlsbad and Hathorn spring-waters at Saratoga do almost, if not quite as well, as the imported water. The advantage of employing these waters is that they not only unload the bowels, but tend to correct acidity and relieve chronic gastric catarrh by their favorable influence upon the gastrointestinal mucous membrane.

Should hemorrhage from the stomach occur, the patient should be put to bed, and if not too depressed by the bleeding, a small ice-bag may be placed over the epigastrium, and 2 teaspoonfuls of adrenalin chloride (1:1000) mixed with 2 or 3 ounces of water may be given by the mouth, with the hope that the adrenalin chloride will contract the bloodvessels and arrest the hemorrhage. Where adrenalin chloride cannot be obtained, from 5 to 30 drops of Monsel's solution may be given in 2 ounces of water. The use of ergot hypodermically, or by the mouth, in this condition can scarcely be of benefit, as it will raise arterial pressure in other parts of the body and may increase the hemorrhage. The general treatment in these cases is, of course, identical with that of profuse hemorrhage occurring from other parts of the body, and consists in the use of $\frac{1}{4}$ of a grain of morphine hypodermically to allay mental distress, and hypodermoclysis of normal salt solution. Should the hemorrhage be sharp and very profuse, the question of operative interference arises.

The decision as to whether an operation should be performed in gastric ulcer with hemorrhage is a most difficult one to reach. We are rarely tempted to operate in such cases unless the hemorrhage is so severe as to be alarming, and yet its very severity renders the condition of the patient unfavorable to operation.

In deciding whether an operation is needful in a case which has had an attack of gastric hemorrhage, consideration must be given to the character of the ulcer. Even those surgeons who are most radical in advising operative measures in these cases state that in cases of hemorrhage from acute ulcer medicinal measures will usually control that particular bleeding, and as an immediate second hemorrhage is rare operation is not demanded. (See Prognosis.) When the hemorrhage does recur and particularly if it be profuse on recurrence, operation is to be considered carefully, whether the ulcer be acute or chronic. The operation of election is gastroenterostomy, for it has been found impossible to find the bleeding spot in most cases because the bleeding often comes from several spots, and indeed may ooze from a multitude of eroded spots. Needless to say that the gastroenterostomy is not designed to stop a bleeding already in active progress, but is to be performed as soon as possible to prevent further bleedings.

When the ulcer is very chronic—that is, has lasted a long time—its edges may be so indurated and the bloodvessels in its cavity so eroded that little hope of cure by natural processes can be entertained, an operation is wise. It is not necessary to excise the ulcer. A gastroenterostomy gives the stomach rest and the circulation in its walls becomes modified. Not only does such an operation remove the danger of hemorrhage, but it may change the patient from a chronic dyspeptic to a well-nourished, healthy individual. If, however, the ulcer causes suffering or chronic gastric distress, surgical interference should be carefully considered. This holds true with special force, if the existence of a chronic ulcer near the pylorus is causing obstruction.

Operation for the removal of the ulcer, when as yet it has neither undergone perforation nor produced hemorrhage, has been advised, but of course has not been very popular, as few patients care to submit to it, and few physicians have considered it advisable to strongly urge operative interference.

The surgical treatment of gastric ulcer with perforation is now a well-recognized procedure in modern medicine. When perforation occurs operation should be resorted to at once, unless the patient is profoundly shocked, when some delay is advisable in order that she may rally.

The best results are obtained in the cases which are operated on during the first twelve hours after the occurrence of perforation. An analysis of these statistics shows that the mortality under operation has been progressively reduced:

In July, 1899, Tinker added 57 cases to Keen's list of 156, and in 1900 he collected 19 others, which made the total number 232, with a mortality of 48.81 per cent. Later, in the year 1900 Finney made an addition of 36 cases to Tinker's last list, thus bringing the number up to 268, of which the mortality was 48 per cent. Of 163 cases collected since the publication of Finney's paper in 1900, 102 recovered and 61 died—a mortality percentage of 37.04. These figures bring the statistics up to June, 1904.

CANCER OF THE STOMACH.

Gastric carcinoma is one of the most common forms of malignant growth. Many years ago Welch showed that in 31,482 cases of cancer the disease affected the stomach in 21.4 per cent. Startling statistics as to the increasing frequency of cancer have been published within the last four years by several writers, of which one of the leaders has been Roswell Park, of Buffalo. In the United States census for 1890, the deaths from gastric cancer were placed at 2014 as against 304 for cancer of the rectum, and 876 from cancer of the liver; whereas, in the census for 1900, there were 4220 deaths from cancer of the stomach, 574 from cancer of the rectum, and 1784 from cancer of the liver. As a large proportion of cases of cancer of the liver are secondary to growths elsewhere, particularly in the stomach, these facts are of great interest. In the city of Washington the deaths from cancer of the stomach in the decade from 1881 to 1890 were 191, and in the decade from 1891 to 1900, 339, which shows an increase greater in proportion than the growth of the city population. So, too, Templeton, of Dundee, found that from 1877 to 1902 the death rate from gastric and œsophageal cancer increased 12.66 per 10,000.

Etiology.—Gastric cancer is far more common in males than in females. Welch states the proportion to be 5 to 4, but in Osler's cases the proportion was 5 to 1. The fact that gastric ulcer may in some cases seem to be the site for the development of gastric cancer has little real bearing upon the etiology of this disease. While some patients state that they have been subject to gastric disorders for years before the final illness develops, it has been my experience that a very large proportion of patients state that hitherto they have had perfect digestion, and have never known what it was to have gastric distress in previous years. In other words, given a man who complains of grave gastric symptoms and loss of weight, who has not cirrhosis of the liver, or dilatation of the stomach, who has not used alcohol to excess, and who boasts of his good stomach during the first forty years of life, that man will often be found to have gastric cancer. We have no knowledge of the etiology of gastric cancer beyond the facts already named.

Morbid Anatomy.—Gastric cancer is usually primary, but it may be secondary. The most common form is the medullary carcinoma, a form of the spheroidal-cell cancer; the adenocarcinoma, the malignant adenoma of German writers, or cylindrical-cell cancer, is second in point of frequency. The scirrhus type is the third in frequency. Gelatiniform degeneration occurs in the first and second, and when present such tumors are called colloid cancers.

The medullary, or spheroidal-cell type, is the most rapid in its growth, usually tends to ulcerate early, and is followed by metastasis and direct extension to contiguous organs sooner than the other types.

The scirrhus is a denser growth, infiltrating and indurating the submucosa often to some distance, or even all of the organ; under such circumstances, the thick, dense, gastric wall has led to the condition being called "India-rubber bottle stomach," or "leathery stomach."

The colloid growths form tumors of greater size, often extending by contiguity and matting adjacent organs into a solid mass. On section the clear or grayish gelatiniform trembling matrix is found to be enclosed in alveoli, often of macroscopic dimensions.

The pylorus is the part of the stomach usually affected; next to it in frequency is the lesser curvature, but out of Welch's 1300 cases the pylorus was affected 791 times and the lesser curvature but 148.

Mr. Moynihan, of Leeds, has investigated the subject of how malignant growths spread in the gastric wall, and has concluded:

1. That malignant disease of the stomach begins in the majority of instances near the pylorus, just below the lesser curvature.

2. That from this point it spreads most rapidly and most widely in the submucosa.

3. That the rate of growth toward the cardiac orifice is rapid, toward

the duodenal side extremely slow. The duodenal extremity of the viscus is rarely affected extensively.

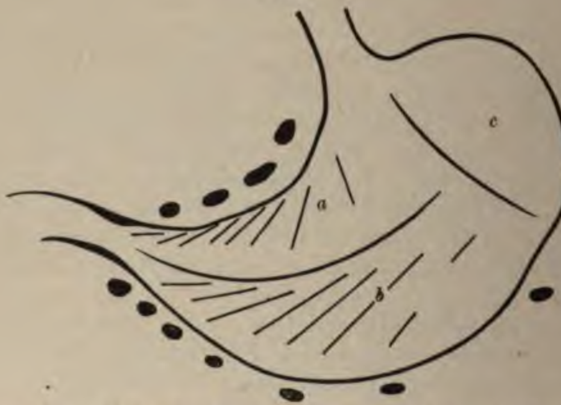
4. That the tendency of the growth is to drift toward the curvatures.

He found that the lymphatic system of the stomach was comparatively simple. There are three chief lymphatic areas of the stomach (Fig. 80).

1. An area along the lesser curvature (*a*) from which the lymphatic vessels pass upward and to the left into the coronary glands. The coronary glands lie along the artery of the same name. At the cœliac axis they become continuous with the glands along the upper border of the pancreas.

2. An area (*b*) along the greater curvature from which the lymphatic vessels pass downward and to the right into the glands lying along the greater curvature. These glands are more numerous near the pylorus, and from here pass to the head of the pancreas and become continuous with the hepatic group of glands which lie along the hepatic artery, and in part along the pyloric artery.

FIG. 80



The lymphatic vessels and glands of the stomach: *a* is the most frequently affected area, *b* is next, and *c* is the "isolated area." (Moynihan.)

3. In addition to these two areas is a third (*c*), for which I (Moynihan) suggested the name "isolated area." This area comprises the greater tuberosity of the stomach, the lower end of the œsophagus, and an area along the greater curvature as far, approximately, as the limit of supply of the left gastroepiploic artery. Its lymphatic vessels pass downward to the hilum of the spleen. The term "isolated area" seems singularly appropriate for this region, for it is very rarely affected by growth spreading upward from the pylorus.

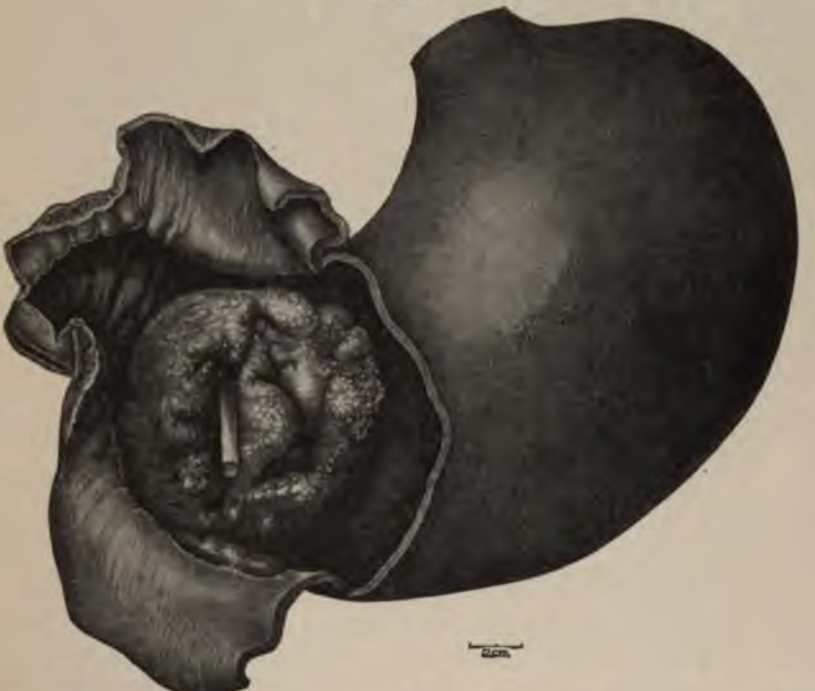
If a cancer of the stomach arising independently of ulcer is examined in its early stages it will usually be found covered with mucous membrane, which later ulcerates. The entire wall of the viscus may soon be involved, but in the colloid form the mucous membrane may not be destroyed.

The effects of cancer of the stomach upon the shape of this viscus depend largely upon the character of the growth, and chiefly upon its situation. The general tendency is for the stomach to be decreased in size, but if the growth obstructs the pylorus the stomach may be greatly dilated. When the tumor is widely diffused the gastric walls are much thickened, and if it be of large size it may displace the pylorus very greatly by its weight.

Perforation of the stomach through the cancerous mass may occur, but this is not a frequent complication.

Symptoms.—The symptoms of gastric cancer may not manifest themselves for a long period after the growth has begun, and even when the general nutrition is impaired the patient may not complain of gastric disorder. It is a mistake to suppose that gastric cancer is usually very painful.

Fig. 81



Stomach. Large, ulcerating, fungoid, cylindrical-cell carcinoma, situated on the posterior wall near the pylorus, which was slightly obstructed by the projecting growth. A glass rod is passed through a perforation near the centre of the floor of the ulcer. The cardiac end of the organ is moderately dilated There was secondary enlargement of the lymph nodes behind the stomach, and metastatic nodules in the liver.

When symptoms are present they may be divided into the *objective* and the *subjective.* The objective symptoms are pallor, which becomes well marked; a *loss of weight,* which is often extraordinary, amounting to a loss of from fifty to seventy pounds in a few months, and with this there is usually a rapid *loss of strength.* The *anæmia* is chiefly the result of a marked decrease of hæmoglobin, although marked reduction and morphological alteration in the erythrocytes may yield a blood picture resembling pernicious anæmia. Sooner or later *leukocytosis* occurs.

The symptoms presented by the patient, in the sense that they are complained of by him, are loss of strength, *gastric distress* and "*dyspepsia*," loss of appetite, *nausea*, and not rarely *vomiting*. Vomiting is a far more constant symptom when the pylorus is obstructed than when it is free, and the matters vomited may indicate a feeble digestive power and be colored like coffee grounds due to exuded and altered blood arising from the ulcerated growth. Sometimes a free *hæmatemesis* develops. Care should be taken that the presence of altered bile in the vomit is not taken for altered blood.

Not rarely the patient complains of constant *gnawing pain* in the stomach, which may or may not be increased by the taking of food. The pain is usually more severe when the disease is in the region of the pylorus. If the skin over the epigastrium is lightly touched, it may be very sensitive. There may be *moderate fever*, *constipation*, and *œdema of the ankles*.

Robson states that pain is present in 86 per cent. of cases, vomiting in 85.3 per cent., and a tumor is palpable in 76.6 per cent. These figures refer to the entire history of the cases recorded, and, as pointed out elsewhere, none of these symptoms may be present during the early stage of the disease, when it is most amenable to surgical treatment. Such percentages are, therefore, of greater value from a statistical than from a diagnostic standpoint.

Diagnosis.—The pallid, cachectic hue of the patient, combined with a history of loss of weight, and with the fact that the patient is usually beyond the fortieth year, should make the physician suspect at least the presence of a malignant growth, and this suspicion becomes stronger as he is able to exclude other causes of anæmia and emaciation, such as, for example, diabetes, Bright's disease, and pernicious anæmia. The presence of cough will usually be a guide to the examination of the lungs for pulmonary tuberculosis. It must not be forgotten that abdominal tuberculosis produces, as a rule, a dry, harsh skin, instead of the peculiar waxy or greasy skin of malignant growth and pernicious anæmia.

In the diagnosis of malignant growth the blood may give considerable information and aid us in separating this condition from pernicious anæmia, which resembles it very closely in some of its objective symptoms. Malignant growth is characterized by a marked decrease in the amount of hæmoglobin and in the hæmoglobin index, a condition the reverse of that in pernicious anæmia. When the case is far advanced there is usually a diminution in the number of red cells, but this diminution is only moderate in early cases. Leukocytosis is usually present in moderate degree, averaging, perhaps, 20,000 to 25,000, and it is much more marked if metastasis, hemorrhage, ulceration, or septic infection occur. The increase in white cells is chiefly in the polymorphonuclear neutrophiles. It is a noteworthy point that these cells may be relatively increased without a distinct leukocytosis being present. Normoblasts and myelocytes are sometimes found in limited numbers when the disease is far advanced.

While it is true that in a few cases of pernicious anæmia there is a marked diminution in the quantity of hydrochloric acid, it is rarely if ever so persistently absent as it is in gastric carcinoma.

Another condition which may give rise to much difficulty in diagnosis is ulcer of the stomach, with thickening and induration around it. This condition is particularly prone to appear in the neighborhood of the pylorus, and it may be impossible by palpation to differentiate ulcer and induration from scirrhous cancer. In other words, every case in which a mass can be felt in the stomach is not one of cancer. In such a case the excess of hydrochloric acid in ulcer and its absence in cancer are valuable factors in diagnosis. Then, too, ulcer is much more frequently present in persons under forty, but cancer is more common in persons over forty.

Inspection of the epigastrium very often gives most valuable information, because it may reveal a bulging and undue pulsation due to impulse from the aorta transmitted by the growth, or the presence of a nodule. A deep breath taken during inspection may reveal distinct movements of the mass. If a Seidlitz powder is taken in two parts, so that the contents of one paper follow the other, the tumor can sometimes be seen to be projected against the abdominal wall by the distended viscus. Palpation may reveal a mass which usually presents an uneven surface, and this may be distinctly nodular.

The position of the mass may be varied by the pressure of food in the stomach. If a mass cannot be felt, immersing the patient in a hot bath may relax the tense belly wall so that the tumor may be demonstrable. Percussion, if it is carefully and gently performed, over the mass may give an impaired note.

Additional signs on palpation consist of a hardening of the gastric walls due to contraction of their muscular fibres, and if the patient is thin the mass may be so movable that it may be pushed high up under the ribs or far down toward the pelvis. The mobility is of great diagnostic value, for if the mass be due to induration about an ulcer the gastric wall will nearly always be glued to adjacent tissues, and therefore be made fast. As a rule, tumor at the pylorus is more readily palpated than one at the middle of the stomach, and a growth at the cardia is rarely to be felt.

Auscultation may reveal constant, direct, or reversed peristalsis if the growth obstructs the pylorus, and all the symptoms of gastric dilatation may be present if the growth be so situated. (See Gastric Dilatation.)

A very useful and reliable method for the purpose of determining the presence of gastric cancer is the determination of the character of the stomach contents.

If the stomach contents are examined after a test meal, there will be an almost complete or total absence of hydrochloric acid and an abnormal amount of lactic acid present. To determine these facts, we resort to the use of a test meal and certain chemical tests, as follows:

Boas' test meal consists in the use of an ordinary breakfast roll weighing about 1 ounce, with 10 ounces of water, or of weak tea, which should contain no milk or sugar. This is allowed to remain in the stomach for one hour, and then is removed by the stomach tube. The quantity of fluid obtained should equal from three-quarters of an ounce to an ounce and a half. It is best to examine this fluid microscopically, and then, after filtering it, to apply the tests for hydrochloric acid and the organic acids. The two common tests for free hydrochloric acid are Günzburg's phloroglucin-vanillin and Boas' resorcin test. Günzburg's reagent is composed of:

Phloroglucin	gr. xxx (2 grams)
Vanillin	gr. xv (1 gram)
Alcohol (absolute)	℥j (30 c.c.)

This solution should be carefully protected from the light by being kept in a dark bottle and should be frequently prepared, as stale solutions are uncertain. One or two drops of this reagent are placed in a porcelain dish or capsule, with an equal quantity of the filtrate obtained from the gastric contents. The dish is then gently heated over an alcohol lamp or Bunsen burner to such a degree that slow evaporation takes place. If free hydrochloric acid is present, a typical rose-red hue develops at the edge of the mixture where it is drying on the dish, or it may be less of a pink and more of a bright red. This test is exceedingly delicate and very certain.

Boas' reagent depends upon the fact that resorcin produces a somewhat similar reaction with free hydrochloric acid. This reagent is composed of:

Resublimed resorcin	gr. lxxv (5 grams)
Cane-sugar	gr. xlv (3 grams)
Alcohol (94 per cent.)	℥iijss (100 c.c.)

A few drops of this reagent are placed in a porcelain dish and an equal quantity of the stomach filtrate added to them. Heat is applied as in the previous test, and if the acid is present a very perceptible red color appears at the edge of the evaporating mixture.

Still another point of diagnostic importance is the examination of the stomach contents by the microscope, which may reveal blood cells and portions of malignant growth. While it is true that the presence of blood may be due to ulcer, the associated pieces of growth are, of course, diagnostic.

Rommelaere asserts that if a patient over forty years of age with chronic gastric disease eliminates less than 180 grains of urea a day, he has cancer. This is probably too dogmatic. A better way of putting it is to say that when a patient with these symptoms eliminates 450 grains a day he has not gastric cancer.

It is important to note that a bacillus called the Oppler-Boas bacillus is present in the stomach contents of cases of gastric cancer with great constancy. Indeed, it is present ninety-nine times in a hundred. This bacillus is a very long, non-motile organism, which has the power of converting sugar into lactic acid, and lactic acid is present in large amount in this disease.

Prognosis.—The prognosis is only hopeful in inverse ratio with the size of the growth, the ability of the surgeon to remove it, and the general state of the patient. Even surgery can offer only temporary relief, for in the majority of cases recidivity takes place. Fenwick asserts that if the potassium sulphocyanide disappears from the saliva, the patient dies within a month.

Duration.—This varies greatly. In some instances death comes in a few weeks after the disease is recognized. In others it is deferred for months, particularly if the growth be scirrhous and involves the pylorus, when a gastroenterostomy, by relieving obstruction and permitting nourishment, may prolong life for a long period. My colleague, Dr. Keen, performed gastroenterostomy in a case under my care in which the growth was so great that excision was impossible. The patient was greatly emaciated and more than sixty years of age, yet he gained nearly thirty pounds in six months, and lived in comfort for two years and a half after the operation.

Treatment.—The medicinal treatment of gastric cancer consists first in the administration of anodynes if there is much pain. These anodynes may consist of small doses of morphine or codeine or cannabis indica. Much of the distress due to so-called dyspepsia can be relieved by the use of 5 to 10 drops of dilute hydrochloric acid, with 1 or 2 drachms of the fluid extract of condurango, given immediately after or with each meal. If starchy foods are taken, taka-diastase may be used.

The use of a stomach tube for washing out the stomach is usually inadvisable, as it may produce a perforation if the growth is soft or the ulceration deep.

Should vomiting occur and blood be in the vomit, the directions for the treatment of hæmatemesis in the article on Gastric Ulcer should be followed. Many patients with gastric cancer can be much improved and their lives prolonged by operation, but it is essential that the growth shall be limited to the stomach and that it does not involve neighboring parts. For this reason it is of vital importance that the diagnosis of the disease shall be made at the earliest possible moment. Indeed, it may be said that every patient who develops persistent gastric symptoms after the age of forty should be regarded as a possible case of gastric cancer until repeated studies of the gastric contents have excluded this disease. In the opinion of the writer, every case of gastric cancer in its early stages should at least be subjected to exploratory operation, since by this means it may be discovered that the diseased area may be excised. The danger of exploratory operation, as compared to the certainty of death if the growth is not interfered with, is not to be considered.

Kronlein has shown that the average duration of life after patients come under observation is about nine months. If they submit to operation it is more than twelve months. The duration of life was greater in those treated by gastrectomy than in those treated by gastroenterostomy.

HYPERTROPHIC STENOSIS OF THE PYLORUS.

Definition.—This condition, as its name implies, is one in which there is thickening and overgrowth of the muscular fibres in the pyloric portion of the stomach, with spasm and consequent obstruction to the free passage of its contents into the bowel.

Overgrowth of the tissues about the pylorus may be divided into *three types*. In one the gastric walls become thickened by an overgrowth of connective tissue, which not only results in an increased diameter of the part, but also in a diminution of the size of the entire stomach. A second form has been described by French authors, which is associated with sclerotic hypertrophic changes in the other abdominal viscera, such as the liver, pancreas, and kidneys. The third form is that which is known as congenital hypertrophic stenosis, which has no etiological relationship to the two forms just described. All three of these forms are quite rare, the last being most frequent.

Etiology.—We have little knowledge of the causes of hypertrophic stenosis of the pylorus. In some instances it has been thought that the overgrowth is the result of some congenital defect, or, in other words, that it is a primary condition, but in others it has seemed to be certainly secondary. It is probable that in the hypertrophic stenosis of adults the underlying cause is chronic gastritis. When it occurs in adults hypertrophic stenosis is usually a disease of middle life.

Morbid Anatomy.—In that form of this disease which arises in middle life and which develops as a result of chronic gastritis, notwithstanding the non-existence of the obstruction at the pyloric opening, dilatation of the stomach as a secondary condition seems to be rare, but this depends somewhat upon how diffuse the overgrowth of connective tissue happens to be. When it is limited strictly to the pyloric region, dilatation ensues because of the obstruction. When it is more diffuse, the stomach may be diminished in size. Very rarely does the overgrowth of connective tissue develop to such an extent as to make it possible to discover any mass by careful palpation.

When the condition is due to some congenital defect, the pathological condition is somewhat different. It is found that the organ is larger than normal and that its walls are thickened by overgrowth of its muscular fibres. At the pylorus these muscular fibres have undergone great hypertrophy, so that this part of the stomach feels like a solid mass between the fingers, and on section it is found to be dense and firm, the mucous membrane lining the part being thrown into folds which lie in the direction of the long axis of the organ. Sometimes one of these folds is so much larger than others that it aids in producing obstruction and seems to form a large part of the overgrowth. Moynihan has well said that such a stomach may appear and feel much like the bladder and prostate when they have been removed by dissection. If the duodenum is opened, the pyloric orifice may resemble that of the cervix uteri when it is seen through the vagina. The thickening of the muscular fibre is not always limited to the pyloric area, but sometimes extends into the duodenum. The longitudinal muscular layers are not greatly increased in size, and, although there is a general hypertrophy of the muscular element throughout the entire organ, it is often so slight at the cardiac end of the stomach that it is scarcely noticeable.

Symptoms.—The symptoms of stenosis of the pylorus in adults consist in a *sensation of fulness, pressure,* and *pain* in the stomach. There are also evidences of motor insufficiency, and when the obstruction to the pylorus becomes marked, *vomiting* may come on to relieve the stomach of materials which cannot escape into the intestine. So, too, the patient may *lose flesh* as the result of interference with the digestion and with the retention of proper quantities of food. The *hydrochloric acid* of the gastric contents is usually *diminished,* probably because of the chronic gastritis which precedes the disease.

Diagnosis.—It may be exceedingly difficult to differentiate hypertrophic stenosis of the pylorus in an adult from gastric cancer occurring in this portion of the stomach. In the article on Gastric Cancer it was pointed out that many of these patients give a history of perfect digestion and no gastric distress until the cancer develops; but in hypertrophic stenosis there is usually a history of many years of discomfort, with a constant endeavor to find food which would not cause indigestion. The absence of cachexia, although the patient may be anæmic, also points toward hypertrophic stenosis. On the other hand, if the liver or gall-bladder seems to be affected, the condition is almost certainly carcinomatous. In some instances it is impossible to make a differential diagnosis without operation, and even then it may require a microscopic examination to determine that the thickening is not malignant. So far as probabilities are concerned, it may be stated that the presence of obstruction at the pylorus in a person at or past middle life is very much more likely to be carcinoma than hypertrophic stenosis, since the former condition is quite common and the latter condition is very rare.

Difficulty may also be experienced in differentiating hypertrophic stenosis from cicatricial contraction due to the remains of an old gastric ulcer. The physical signs and symptoms present at the moment of examination may give us no information, but the past history of the case may aid us materially. Thus, when gastric ulcer has been present, there may be a history of hemorrhage and severe pain, which is absent in cases of stenosis.

Congenital Pyloric Stenosis.—As already stated, the condition of pyloric stenosis, when not due to ulcer or cancer, is met with in the majority of cases in very young children, and in such is undoubtedly congenital. In such instances the child is born apparently healthy, and after two or three days, or several weeks, of life is seized with *sudden* and *persistent vomiting*, for which no errors in diet can be held responsible. The vomiting is often forcible and gives the child relief for the time being until more food is taken, when it recurs. The time during which the food is retained varies from a few minutes to several hours, and in some instances the mere act of swallowing seems to reflexly produce the motions of vomiting. In some instances the vomiting is sufficiently prolonged to empty the stomach thoroughly; in others a considerable quantity of food may be retained. It is a noteworthy fact that even if the vomiting is severe, *bile* is *never present* in the ejected material, because the closed pylorus prevents it from being drawn from the duodenum. The result of the ejection of the food almost as soon as it is swallowed, combined with the deficient digestive function of the stomach which is nearly always present, is *rapid emaciation*, *exhaustion*, and *death*. Sometimes a period of semi-consciousness ensues, and occasionally the child is seized with a convulsive attack.

The duration of life varies from four to five weeks to six months.

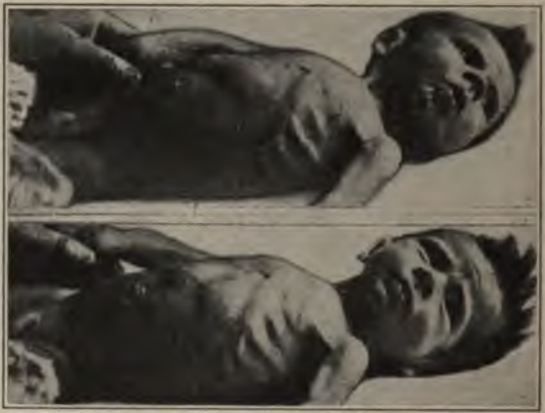
As the disease progresses and emaciation becomes marked, it may be possible to see the outline of the stomach in its forcible contractions if the abdomen is carefully examined in a good light. If deep palpation can be practised, the thickened pyloric portion of the stomach can be felt forming a distinct contrast to the empty and collapsed intestines, which are prevented from containing their usual food and liquid by the obstruction at the pylorus. (See Fig. 82.)

The points which are strongly in favor of hypertrophic stenosis of the pylorus in infancy are the causeless and persistent vomiting, the absence of bile from the vomit, the constipation, the presence of a tumor in the pyloric area, the collapsed intestines and distended stomach, and the fact that gastric digestion is almost completely arrested, and, finally, that these symptoms are present in a young child.

Prognosis.—The prognosis in hypertrophic stenosis is unfavorable as to recovery, and it may be unfavorable as to life if the obstruction is very great. A very few cases are on record in which recovery without operation has occurred.

Treatment.—The treatment consists in the use of lavage and in feeding through a rubber tube, so that the movements of swallowing are not necessary. In some cases it may be possible to prevent the vomiting by gently washing out the stomach before each feeding. When this measure fails, or if the symptoms are exceedingly severe, operative procedure must be resorted to before the child is sufficiently exhausted to contraindicate the operation. The operation should either be pyloroplasty or gastroenterostomy. Moynihan states that anterior gastroenterostomy has been performed 9 times, with 5 recoveries and 4 deaths, but 1 of these deaths was due to acute obstruction caused by a Murphy button. Loreta's operation of pylorodiosis has been performed 9 times, with 7 recoveries.

Fig. 82



Showing the gastric peristalsis. Note constriction of stomach by passing wave. (Ibrahim.)

HOUR-GLASS STOMACH.

Definition.—Hour-glass stomach, sometimes called "bilocular stomach," or "Sanduhrmagen," is a condition in which the stomach is divided into two parts by a contraction which may exist anywhere between the cardiac and pyloric orifices. Very rarely, indeed, a trilocular condition may be present, and, still more rarely, a quadrilocular state may exist.

Etiology.—Hour-glass stomach may be congenital or acquired. There is some difference of opinion as to the relative frequency of these two forms of the condition. Some authors have maintained that all cases are acquired, while others assert that the congenital cases are more common. Fenwick says that about 45 per cent. of the cases which have been so far recorded showed no sign of either ulcer or scar in the stomach, or if an ulcer or scar were present it was manifest that it was more recent than the stricture itself; and again he states that only 1 case of the acquired type has been found in the London Hospital in forty years, whereas several instances of the congenital type were met with during the same period of time. To those who believe in the congenital origin of these cases, Moynihan is strongly opposed. He states that he has examined a number of specimens and searched the literature carefully, and can find no instance in which there is good ground for considering the contraction as congenital. He therefore contradicts those who think that the ulcer is secondary to the contraction.

According to Moynihan, there are three causes of hour-glass contraction: first, perigastric adhesion; second, chronic ulcer; and third, malignant disease. The perigastric adhesions are most commonly due to ulcer of the stomach or to a nearby inflammatory process set up by the presence of gallstone. Chronic ulcer not only produces perigastric adhesions, but it may, in healing, cause much contraction and thickening of the stomach, so that its wall becomes puckered and its calibre decreased. Associated with this contraction, due to the formation of scar tissue, there is also a certain amount of muscular spasm, the circular muscular fibres contracting in such a way as to resemble a sphincter muscle. This spasmodic contraction accounts for the paroxysms of discomfort from which the patient occasionally suffers.

Pathology and Morbid Anatomy.—On examining a stomach for the seat of hour-glass contraction, two sets of thickened muscular fibres, which cross one another, may sometimes be seen. These bundles are usually one-half inch or more in width, and cross one another at the point of contraction, and it is the shortening of these muscular fibres which produces the deformity. That this is not the cause in most instances, however, is shown by the fact that these muscular fibres are frequently absent, or, at least, are not abnormally developed. When the constriction is due to the formation of a cicatrix the narrowed band forming the dividing septum between the two pouches is composed of fibrous tissue; this may be puckered, indicating that it has followed an ulcer.

Symptoms.—The symptoms of hour-glass contractions of the stomach are by no means definite. There are, however, certain physical signs which can be elicited in some cases which are of great importance. These methods are well described by Moynihan in the following words:

"1. If the stomach tube be passed and the stomach washed out with a known quantity of fluid, the loss of a certain quantity will be observed when the return fluid is measured. Thus, if 30 ounces be used, only 24 ounces can be made to return. Woelfler, who called attention to this sign, said that some fluid seemed to disappear 'as though it had flowed through a large hole'—as, indeed, it has, in passing from the cardiac to the pyloric pouch (Woelfler's 'first sign').

"2. If the stomach be washed out until the fluid returns clear, a sudden rush of foul, evil-smelling fluid may occur; or if the stomach be washed clean, the tube withdrawn and passed again in a few minutes, several ounces of dirty, offensive fluid may escape. The fluid has regurgitated through the connecting channel between the pyloric and cardiac pouches (Woelfler's 'second sign').

"3. Paradoxical dilatation. If the stomach be palpated and a succussion splash obtained, the stomach tube passed, and the stomach apparently emptied, palpation will still elicit a distinct splashing sound. This is due to the fact that only the cardiac pouch is drained; the contents of the pyloric pouch remain undisturbed and cause the splashing sound on palpation. For this phenomenon Jaworski has suggested the appropriate name of 'paradoxical dilatation.' Jaboulay has pointed out that if the cardiac loculus be *filled* with water, a splashing sound can still be obtained by palpation over the pyloric pouch. The sign of paradoxical dilatation is best elicited after washing out the stomach in the ordinary manner. When the abdomen is examined at the completion of the washing, and when the stomach has been apparently drained quite dry, a splashing sound is readily obtained, for some of the fluid used has escaped into the pyloric pouch through the connecting channel.

"4. Von Eiselsberg observed in one of his cases that on distending the stomach a bulging of the left side of the epigastrium was produced; after a few moments this gradually subsided, and concomitantly there was a gradual filling up and bulging of the right side.

"5. Von Eiselsberg also called attention to the bubbling, forcing, 'sizzling' sound which can be heard when the stethoscope is applied over the stomach after distention with CO_2. If the two halves of a Seidlitz powder are separately given and the stomach be normal or dilated, no loud sound is heard anywhere except at the pylorus; if a constriction is present in the stomach, a loud, forcible, gushing sound can be easily distinguished at a point two inches or three inches to the left of the middle line.

"6. I (Moynihan) first called attention, two years ago, to a sign which I have since found of great service in establishing a diagnosis of hour-glass stomach. The abdomen is carefully examined and the stomach resonance is percussed. A Seidlitz powder, in two halves, is then administered. On percussing, after about twenty or thirty seconds, an enormous increase in the resonance of the upper part of the stomach can be found, while the lower part remains unaltered. If the pyloric pouch can be felt, or is seen to be clearly demarcated, the diagnosis is inevitable, for the increase in resonance must be in a distended cardiac segment. If the abdomen be watched for a few minutes the pyloric pouch may sometimes be seen gradually to fill and become prominent.

"7. Schmidt-Monard and Eichhorst have both seen a distinct sulcus between the two pouches inflated with CO_2. In one case the two pouches, with a hard—as I thought malignant—mass between them, could readily be seen. When both pouches were distended with CO_2, alternate pressure upon them showed unmistakably that they communicated through a very narrow orifice, for the one could be emptied slowly into the other, and the fluid could be felt to ripple gently through. The diagnosis in such a case is simplicity itself. In another case a distinct notch was seen at the lower border of the inflated stomach.

"8. Ewald has called attention to two signs which he considers of value in establishing a diagnosis. When the stomach is filled with water and examined by gastrodiaphany, the transillumination is seen only in the cardiac pouch; the pyloric pouch remains dark.

"9. The deglutable India-rubber bag of Türck and Hemmeter is passed and distended. The bulging caused thereby is limited to the cardiac pouch, which lies to the left of the middle line.

"The two aids to diagnosis of greatest value are, it will be seen, the washing out of the stomach and its inflation with gas by the administration of a Seidlitz powder in two portions. The fluid used for the washing must be carefully measured before being used; the tube is then passed and the stomach emptied, the contents set aside in a separate dish, and the washing commenced. All the fluid now returning is collected in a separate vessel and carefully measured. The two signs of Ewald are of little importance; a correct diagnosis can always be made without them."

Treatment.—The only treatment for hour-glass contraction which can afford any relief is operative. Thus, a gastroenterostomy may be done from both pouches, or gastroplasty or pyloroplasty may be necessary. For these operations the reader is referred to books on surgery.

GASTRIC NEUROSES.

At the present time several states of the stomach are known to exist which depend upon an altered or perverted nerve supply, and are not connected with any pathological lesion which our methods of examination can detect. Gastric neuroses are not commonly met with as conditions independent of true lesions, and the physician must not rest satisfied with a diagnosis of gastric neurosis until he has exhausted every possible means of discovering an actual morbid change. In some instances the nervous affection of the stomach is a manifestation of disease of the central nervous system; in others it is a sign of perverted nervous function due to neurasthenia or nervous exhaustion, and in still other cases it may be dependent upon growths which, being situated in adjacent tissues, press upon the gastric nerves and so cause pain or spasm. Finally, it is to be remembered that even if the physician can discover no sign of gastric lesion, this does not justify a diagnosis of gastric neurosis, because it not rarely happens that disease elsewhere causes pain which is incorrectly referred by the patient to the region of the stomach. Thus, a child with pericarditis or appendicitis may complain bitterly of epigastric pain.

True gastric neuroses may be divided for study into three classes, viz., *disorders of mobility*, *disorders of sensation*, and *disorders of secretion*, and these in turn are divisible into states of excitation and depression.

Cardiospasm, or cramp of the muscular fibres in the cardiac end of the stomach, is a result, as a rule, of irritation of the gastric mucous membrane by superacid secretion. Occasionally it may develop as the result of distention of the stomach by gas, and in some instances no direct cause for its existence can be discovered save that a state of nervous unrest and instability is present. Cramp of the cardia appears in an acute and fleeting form, and as a chronic condition which causes great distress and may be serious, in that it exhausts the patient. In the former cases pain and spasm seize the patient and then pass away. In the latter it often happens that the patient has difficulty in swallowing and expresses the feeling that the food cannot enter the stomach, but remains in the œsophagus. If the taking of food is persisted in, it speedily accumulates in the œsophagus, and when this tube is distended the patient regurgitates the food undigested and devoid of gastric juice, for it has never entered the stomach. The emaciation which follows this inability to take food may lead to the belief that a gastric carcinoma is present, particularly if the patient is advanced in years. When chronic cardiospasm lasts for a long time, dilatation of the œsophagus may develop, and even a diverticulum may be formed.

Treatment.—The treatment consists in the use of remedies designed to prevent and counteract excessive gastric acidity, the avoidance of all irritating or stimulating forms of food and drink, the use of lavage if there is any evidence of chronic gastric catarrh or of fermentation in the stomach, and in feeding through a stomach tube if there is any difficulty in giving the patient a proper amount of nourishment. Boas states that in some cases solids are taken more readily than liquids. Sedatives, such as chloretone, the bromides and chloral, may be used. Sometimes galvanic electricity gives relief, using for its application an intragastric electrode. In some instances the daily passage of a large-sized gastric or œsophageal bougie produces a cure.

Pylorospasm.—Pylorospasm is nearly always secondary to lesions elsewhere, although a primary spasm may occur. In pylorospasm a contraction wave may be seen in a thin patient endeavoring to urge the gastric contents through the closed pyloric orifice, and if the spasm is persistent the stomach contents will not only be retained, as in gastric dilatation, but they may undergo fermentative changes as well, so that symptoms of chronic gastric catarrh or dilatation may be present. In other cases a reversed peristalsis is set up and vomiting comes on, so that symptoms like those of hypertrophic pyloric stenosis ensue. The *treatment* is identical with that of cardiospasm.

Gastric Hyperperistalsis, called by Kussmaul "peristaltic unrest," is a condition in which the stomach almost incessantly continues to maintain peristaltic movement. As a rule, it is most active after meals, but it may be present when the stomach is empty, and even persist at night during absolute rest. Although marked pain is usually not present, the incessant movement of the stomach causes restlessness and gastric discomfort. Often the wave-like movements of the stomach can be felt through the abdominal wall, and their progress is from left to right. These undulatory movements are not demonstrable, as a rule, unless some gastric dilatation is present and the belly wall fairly thin. Three causes are recognized, viz., excessive acidity producing irritation, great reflex excitability, and, most important of all, stenosis of the pylorus, which obstructs the flow from the stomach.

Treatment.—The treatment consists in the use of sedatives, such as the bromides, chloral, codeine, and hyoscyamus, and in the use of counterirritation over the epigastrium. Not rarely an absolute rest cure, with rectal feeding for a week, may be needful to cause gastric quiet. Causes which produce nervous exhaustion and undigestible foods are to be forbidden and hydrotherapeutic measures should be instituted.

Merycismus.—Merycismus is a neurotic condition in which the patient has the ability at will to regurgitate the food from the stomach into the mouth for the purpose of rechewing it, as is done by ruminants. It is usually met with in neurotic degenerates.

Nervous Eructation.—Nervous eructation is not a very rare affection. The patient is usually very nervous and will often sit for hours "rifting up" gas, which, in many cases, is really swallowed air. In other cases the movements of eructation are performed without any gas being brought up. This condition is commonly seen in hysteria. It is best treated by the rest-cure and the administration of tonics or nervous sedatives, such as the bromides, spirit of chloroform, asafœtida, or chloretone.

Closely related to *nervous eructation* is nervous vomiting.

Hyperæsthesia.—Among the sensory disorders of the stomach is *hyperæsthesia*, in which the taking of food causes great gastric distress, so that the patient refuses to eat enough to maintain nutrition. In hysterical cases the patient may be able to eat what she wishes, yet has pain when other articles of food are given to her.

This condition is to be separated from the hyperæsthesia due to gastric ulcer, since it sometimes develops when this lesion is not present. Sometimes it seems to arise from the abuse of alcohol, coffee, ice, or certain drugs, such as quinine and the salicylates, or tobacco. In other instances it arises from nervous exhaustion due to sexual excess, great mental strain, prolonged lactation, or menorrhagia. So, too, it may develop in the course of chlorosis, and while in the majority of instances this condition in chlorosis points to ulcer, the possibility of no ulcer being present must be considered, in view of our knowledge of the existence of this state.

Symptoms.—The symptoms consist in a *sense of fulness* and distention of the stomach, particularly in the neighborhood of the cardia, with some aching or *burning*, which extends upward under the ribs. *Constipation* is usually present. As the condition advances the disagreeable sensations in the stomach become so severe as to amount to *pain*, and the taking of food usually greatly increases the suffering. When fully developed the patient often suffers from *vomiting*, which may occur after every meal. In ulcer, vomiting usually gives temporary relief, but in hyperæsthesia of the stomach it does not. If the disease persists, there is emaciation due to the pain and constant vomiting. The skin over the epigastrium is usually hyperæsthetic, and the *tenderness on deep palpation* is diffuse and not localized as in ulcer. An examination of the gastric contents usually reveals a normal acidity, but in some cases the acidity may be above or below the normal.

Gastralgia.—Gastralgia, gastrodynia, or gastric neuralgia, may be a cause of much severe suffering, for the patient may be seized by a *paroxysm of pain* which seems as violent as a renal or hepatic colic. This pain is felt not only in the epigastrium, but along the edges of the floating ribs to the spine, and it often recurs with a peculiar periodicity. It is not rare in hysteria and neurasthenia. The gastric crises of locomotor ataxia, the pain of ulcer, and that caused by gas must be carefully excluded before a diagnosis of gastralgia is reached; indeed, a diagnosis of simple gastralgia should always be looked upon with suspicion, because gastric pain is so commonly due to some organic cause. Unlike the pain of ulcer, this form is usually relieved by taking food.

Bulimia.—A neurosis of the stomach characterized by excessive hunger and the ingestion of great quantities of food to alleviate the discomfort is called "Bulimia." It is usually met with in cases of hysteria, in cases of exophthalmic goitre, and in cerebral tumor and epilepsy.

Anorexia Nervosa is a form of neurosis with persistent lack of appetite.

Nervous Disorders of Secretion.—A form of nervous disorder of secretion consists in hypersecretion of gastric juice, producing the ordinary symptoms due to acid stomach. It is often met with in chlorotic girls, and is usually associated with constipation. The excessive secretion may occur in paroxysms or be continuous.

The antithesis of this state is that in which there is an absence of secretion, sometimes called *achylia-gastrica nervosa*. This state of absence of HCl is, of course, common in gastric cancer, and it arises also from atrophy of the gastric tubules, but there are instances in which, apparently because of disordered nerve supply, there is absence of secretion for weeks, months, or even years, yet finally it is perfectly re-established.

Treatment.—The treatment of these disorders of sensation consists in the institution of a rest cure for the rehabilitation of the patient's nervous tone; in the use of hydrotherapeutic measures and electricity designed to bring about the same result, and in the prescription of a mode of life which will avoid nervous worry and strain, provide a sufficient number of hours of sleep and out-door exercise, and prevent the ingestion of articles of food which are difficult to digest or irritating to the stomach. For the prevention or relief of painful or disagreeable sensations, a number of remedies may be employed. Not infrequently a dose of a drachm of spirit of chloroform and a drachm of compound spirit of lavender in a little water will dispel gas and distention and act as a sedative to the stomach. In other instances ½ to 1 grain of menthol may be given in capsule or pill. In still others one of the coal-tar products, as antipyrin, acetanilid, or phenacetin, may be used, and in some instances much relief will be obtained by the use of chloretone in capsule or tablet in 3 to 5 grain doses. Where there is a distinct hysterical element and it is considered desirable to exercise a mental influence, the stomach tube may be passed once or twice a day. If an excessive secretion of hydrochloric acid is present, associated with much nervousness, the bromides may be employed, or nitrate of silver and hyoscyamus may be used.

HEMORRHAGE FROM THE STOMACH.

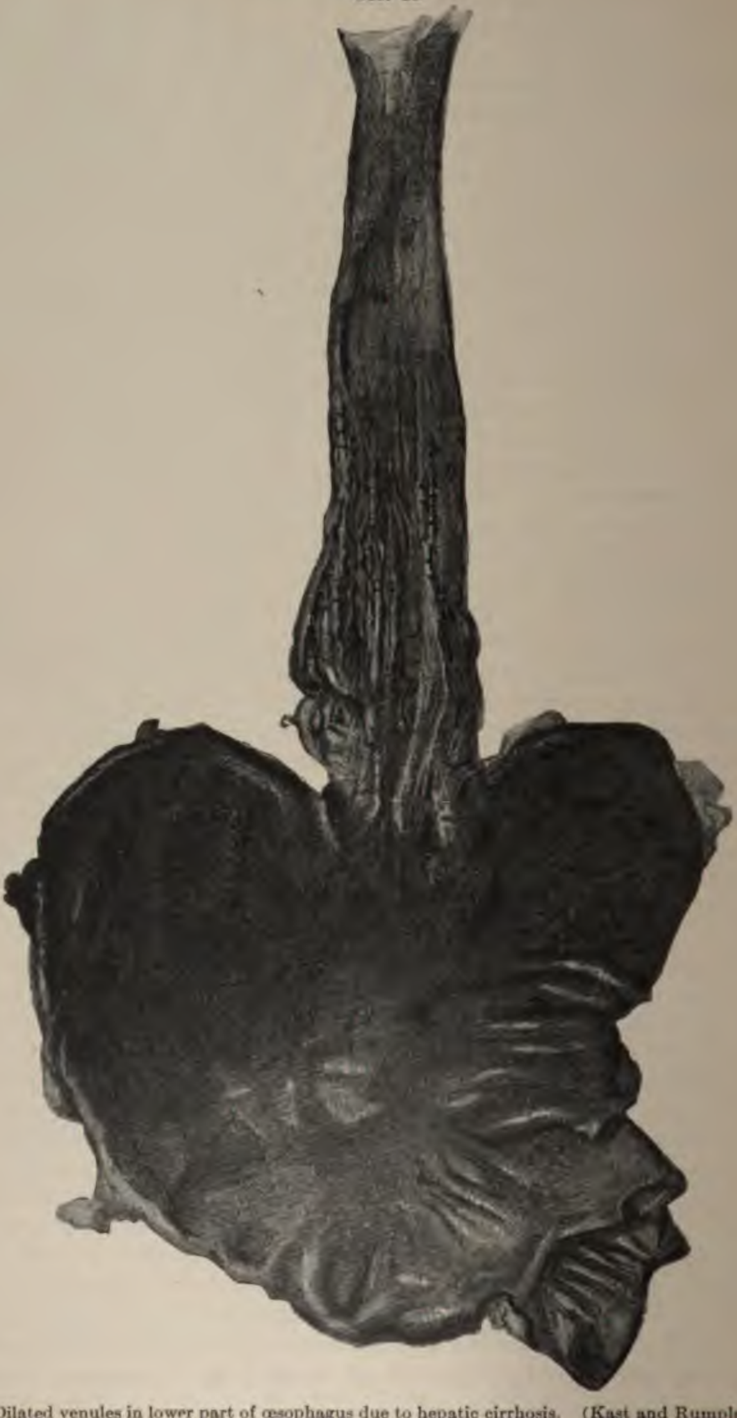
Hemorrhage into the stomach is called *gastrorrhagia*, and when the blood is vomited the condition is one of *hæmatemesis*. It may result from rupture of dilated gastric and œsophageal veins, from ulcer of the stomach, from cancer of the stomach, and from dilatation of the gastric veins in chronic gastric catarrh. It has also been known to follow severe injuries over the epigastrium. Occasionally the vomiting of blood has been due to an aneurysm which has perforated the œsophagus and then drained into the stomach. The physician must also remember that malingerers sometimes swallow blood for the purpose of deceiving their attendants. Sometimes "coffee-ground vomit," due to the presence of altered blood, is met with in cases of gastric cancer, in certain forms of purpura, in hæmophilia, and in persons suffering from such poisons as phosphorus and carbolic acid. Occasionally, too, in cases of exceedingly severe infectious disease, such as yellow fever and smallpox, vomiting of coffee-ground material occurs. By far the most frequent causes of bloody vomiting, however, are cirrhosis of the liver, ulcer, and cancer of the stomach. When due to cirrhosis it is usually met with in males, and when due to ulcer it most commonly occurs in females.

Preble has made a most complete statistical study of gastrointestinal hemorrhage in hepatic cirrhosis, and finds that the great majority of cases occur in the atrophic form, although occasionally hemorrhage takes place in hypertrophic cirrhosis. In one-third of the cases the first hemorrhage is fatal; while in the other two-thirds the hemorrhage occurs at intervals varying from a few years to several years, the longest duration being over a period of eleven years.

In some instances it is possible to make the diagnosis of hepatic cirrhosis, but in other instances the change in the size of the liver is so slow that not for months after the hemorrhage occurs is this organ found to be smaller than normal. In 80 per cent. of the cases, according to Preble, there are varices in the œsophagus, and in more than one-half of these there are evidences of their rupture (Fig. 83). It has also been found that fatal hemorrhages may occur in cases which do not suffer from œsophageal varices. These cases are probably due to the rupture of a large number of capillaries in the alimentary mucous membrane. It is interesting to note that in only 6 per cent. of the cases which showed œsophageal varices was the cirrhosis typical in the sense that the ordinary symptoms of this condition were present. Very profound hemorrhages may come from a very small opening in a bloodvessel, so that at autopsy it may be almost impossible to discover the source of the bleeding.

Aside from the actual vomiting of blood, the symptoms of gastric hemorrhage are those of ordinary hemorrhage, namely, *pallor*, *faintness*, or *actual syncope*, and sometimes death. The vomited blood, if it has been poured out in large quantities, is somewhat venous in color and filled with clots, and if it remains in the stomach any length of time it may become brown or granular in appearance, through the action upon it of the digestive juices. It must always be remembered that the quantity of blood vomited does not necessarily indicate the quantity of blood which has escaped from a bloodvessel, as a very large amount may leak into the stomach before vomiting occurs, and the stomach in vomiting may not completely empty itself. Care must be taken in determining that the blood comes from the stomach, and that the red color is really due to blood. Sometimes a bloody color of the vomit may be due to claret or the juice of various berries. A

FIG. 83



Dilated venules in lower part of œsophagus due to hepatic cirrhosis. (Kast and Rumpler.)

distinction can be made by a microscopic examination, by the history of the ingestion of certain articles of food, and, if need be, by the use of the spectroscope and the various tests which are employed to determine the presence of blood. It must also be borne in mind that persons who suffer from nosebleed, in which the leaking vessel is far back in the nose, may swallow considerable quantities of blood and then vomit it.

Hemorrhage from the lungs, or hæmoptysis, is to be separated from hæmatemesis by the fact that in hemorrhage from the lungs the blood comes up with coughing, and in hemorrhage from the stomach by vomiting, although at times both of these symptoms may be present in each class of cases. The characteristic appearance of a patient well advanced in tuberculosis will be of great diagnostic aid in such cases, and an examination of the chest in the case of hæmoptysis will usually reveal some lesion; whereas, the lungs will be clear in hæmatemesis. In a case of hæmatemesis an examination of the abdomen may reveal an atrophied liver and an enlarged spleen, or some other abdominal state, such as the *caput medusæ*, which will indicate that there is venous stasis in the abdomen. (See Hepatic Cirrhosis.) In hæmoptysis the blood is pink and frothy. In hemorrhage from the stomach it is dark, has little air mixed with it, and is often acid in reaction; whereas, that in hæmoptysis is usually alkaline. In hæmoptysis no dark, tarry stools are present, but they are frequently seen after an attack of hæmatemesis. A day after an attack of hæmoptysis the patient may cough up some thickened, bloody mucus, but there is no difficulty in separating this from the more fluid, dark blood from the stomach. An additional aid in the diagnosis of hæmatemesis is Boas' test given in the article on Gastric Ulcer.

Notwithstanding the profound mental shock and vital depression which often follows a profuse hemorrhage from the stomach, it is worthy of note that death very rarely occurs as the immediate result of this loss of blood, unless the patient is already devitalized by advanced disease or repeated hemorrhages.

For the treatment of bloody vomiting see Treatment of Gastric Ulcer.

CYCLIC VOMITING.

Under the name of cyclic, periodical, or recurrent vomiting, a condition has been rarely met with in which, at certain periods, a child is seized by an attack of *persistent vomiting*, which not only continues while the stomach is being emptied of its normal contents, but persists for many hours afterward, and in some instances ends fatally. The condition in all probability depends upon a form of *autointoxication*. This autointoxication consists, apparently, in a condition of acidiosis. Marfan believed that it was due to an acetonæmia. Edsall, on the other hand, who has studied the disease most carefully, and who has done much work upon the subject of acid intoxication, reports 5 cases in which, at the time of the attacks, this condition existed. The urine between the paroxysms was normal, but immediately before the attack contained diacetic acid and acetone. He found that the immediate administration of large doses of bicarbonate of soda, as much as 2 drachms, This infection may be produced by a large number of organisms, some of which are not pathogenic when the child is in perfect health, and which only become competent to cause disorder or disease when, by some additional cause, the general or local vitality of the patient is reduced. Thus, it not rarely happens that the stools contain myriads of streptococci, staphylococci, the *Bacterium lactis aërogenes*, or the *Bacillus pyocyaneus*, the pathogenic micro-organism which causes green stools, and other bacteria. More important than all, the bacillus of dysentery of Shiga and Flexner is now known to be a cause in a large proportion of cases. (See Cholera Infantum and Dysentery.)

The relationship of specific micro-organisms to the summer diarrhœa of children is well summed up by Pease and Shaw in the following sentences:

"We think it can be fairly concluded that there exists a group of bacilli having, in general, similar characteristics which differentiate them from the typhoid bacillus on the one hand, and the colon bacillus group on the other, which can be said to have an etiological relation to most cases of endemic and epidemic dysentery, whether the same occur in adults or children. That there probably are rare epidemics of this disease in which the clinical course may not conform exactly to the usual type, in which bacteria other than the group of dysentery bacilli are the etiological factors. An example of this kind is the epidemic of dysentery due to the *Bacillus pyocyaneus* infection reported by Lartigau.

"Whether the cases of summer diarrhœa in children not showing symptoms of true dysentery, and not having been more or less closely associated with true cases of the disease, can be considered as uniformly caused by either of these two types of dysentery bacillus is still open to question."

The causes that usually permit these organisms to develop are exposure to cold or excessive heat, so that the bowel is congested and its circulation impaired, and the use of foods which are unsuitable in kind or have become so by infection or chemical change. Winds carrying dust, flies carrying infection, and air carrying gases may all aid in impairing the quality of food. At times the condition develops as a result of an attack of an infectious disease, such as measles. Heat and humidity not only reduce the resistance of the child, but greatly increase the number of micro-organisms in raw foods, especially milk, in which they sometimes number as many as 100,000,000 per cubic centimetre. (Park and Holt.[1])

Pathology and Morbid Anatomy.—The ileocolitis of childhood affects particularly the lower part of the ileum and the colon, the extent depending upon the vital resistance of the child and the virulence of the infection. Even in those cases due to the bacillus of Shiga, the most varied lesions are found. The degree of pathological change varies within wide limits. In some instances there is only a superficial catarrh, with some infiltration of the mucous membrane, while in others the submucous tissues are affected, and in still others areas of mucous membrane may slough. In these severe cases the mucous membrane of the entire alimentary canal may show more or less catarrh.

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If the mucous membrane of the small intestine is examined at autopsy, its villi are found to be soft and swollen, so that the surface of the bowel presents a velvety aspect. In mild cases the hyperæmia is not intense. A universal congestion is present in severe cases, and even punctate extravasations of blood may be seen. The solitary follicles are swollen and protrude above the surface in both the small and large bowel and at the summits of follicles beginning ulceration, which rarely is extensive, is noticeable. Peyer's patches may also be infiltrated, but they are rarely ulcerated. In severe or long-continued cases the solitary glands may ulcerate, but the agminated glands very rarely. An appearance which at first glance may be thought to be an ulcerated Peyer's patch will be found to be due to the running together of several ulcers of solitary follicles.

In severe cases a condition of acute membranous enteritis develops, the lower ileum and colon being covered by a thin false membrane, which can be seen in some cases only with difficulty. This membrane may be of a yellowish-green hue and it lies over a part of the bowel which is greatly thickened by an inflammatory process which involves its deeper coats. Curiously enough, ulceration of the mucous membrane in such an area is unusual.

Symptoms.—The symptoms of acute ileocolitis in childhood vary greatly in their severity and duration. In the *mild catarrhal form* there is a slight *rise of temperature* of from 1° to 2°, with several loose movements of the bowel each day. These stools have a little mucus in each of them and perhaps a few small flecks of undigested food.

If the *condition* is more *severe* there is *pain in the bowels*, with *vomiting*, *high fever*, and the frequent *passage of yellow or greenish stools* containing mucus and considerable amounts of undigested food, and if the condition persists the mucus may become streaked with blood, and *tenesmus* may be severe. There is little flatus and little odor to the passages. The tongue is coated and anorexia is marked.

Because of the fever, vomiting, and diarrhœa, the patient is rapidly prostrated and loses flesh with remarkable speed.

If the course of the colon is palpated through the tumid abdominal wall, some tenderness is usually found. As recovery begins the stools become less frequent, have a more normal color, the quantity of mucus decreases, and the fever falls. If, on the other hand, at the end of a week or two there is no change for the better, the more severe state, in which ulceration of the intestinal lining takes place, is probably present, and death may ensue from exhaustion and depression, with signs of toxæmia.

If recovery takes place it is very slow, a tendency to looseness of the bowels persisting for weeks, mucus being seen in almost every stool, and a relapse being threatened at each change in weather or in the food.

In the *well-developed ulcerated form* the systemic disturbance is profound and often sudden in onset, and its very severity may serve to prevent a *sharp febrile reaction*. The stools are often much fewer per day than in the catarrhal form, but they contain more mucus and less blood. Unlike the stools in the milder type, these passages smell badly. The *belly is distended* and the general loss of flesh is very severe. The *mouth* and tongue are *dry* and *foul*.

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In the *well-developed ulcerated form* the systemic disturbance is profound and often sudden in onset, and its very severity may serve to prevent a *sharp febrile reaction*. The stools are often much fewer per day than in the catarrhal form, but they contain more mucus and less blood. Unlike the stools in the milder type, these passages smell badly. The *belly is distended* and the general loss of flesh is very severe. The *mouth* and tongue are *dry* and *foul*.

In the *membranous form* the stools contain mucus and blood and particles of false membrane, which are easily discerned if the stools are first mixed with water and then strained through a sieve. The degree of prostration and evidence of toxæmia in these cases is very severe, and upon the prolapsed rectal mucous membrane the false membrane may be sometimes seen. At times such cases present at onset, or late in the disease, *severe cerebral symptoms* which may mask the intestinal state.

Diagnosis.—Ileocolitis must be separated from the typhoid fever of infancy. In most cases this is not difficult, because in the typhoid fever of children constipation is often present, and the rose spots may be found. The chief diagnostic points which separate the two affections are that enteric fever is rare and ileocolitis common, that in enteric fever the onset is usually gradual; in ileocolitis it is acute. In one there is an enlarged spleen and the Widal reaction; in the other neither one of these signs is present. If the illness is due to the *Bacillus dysenteriæ*, the agglutination test may reveal that fact. (See Dysentery.)

Prognosis.—The prognosis depends upon several facts. Young children fare worse than children after the fourth year. City-bred children succumb more rapidly than children in the country, particularly if the weather is hot. Children who are strong and hearty at the onset have a better prospect than poorly nourished weaklings. High fever, many stools, much vomiting, much mucus, marked nervous symptoms and signs of toxæmia are of evil omen.

Treatment.—The treatment of ileocolitis consists in the application of mild, continuous, counterirritation over the abdomen by means of a spice poultice, which consists of equal parts of powdered nutmeg, allspice, cloves, and cinnamon, moistened with warm brandy or vinegar. If this cannot be had a mustard plaster, composed of one-quarter to one-half mustard flour and wheat flour, may be applied, the idea being to produce continuous, but not severe, counterirritation.

The child's diet should be carefully regulated. If it is passing undigested food in its stools, those articles which are not being properly dealt with by the digestive apparatus should be withheld. If undigested curds of milk are present, milk should be stopped, or diluted sufficiently to make its digestion easy, and pepsin and hydrochloric acid, or pancreatin with bicarbonate of soda, should be used to aid digestion. Beef-juice, or beef-broth, and chicken-broth may be administered, and if the child is not a very young infant their nutritional value may be greatly increased by adding to them strained barley or wheat gruel. The digestion of these vegetable broths should be aided by the use of liquid pancreatin or liquid taka-diastase. If there are any evidences of inactivity of the liver, minute doses of calomel every third or fourth day are advantageous.

If mucus is present in considerable quantity in the stools a moderate dose of castor oil, varying from a drachm to a tablespoonful, may be used once or twice. Griping may be prevented by the addition of a few drops of paregoric.

In some instances small doses, such as 1 or 2 grains of chloride of ammonium dissolved in fluid extract of licorice and water, may be given twice or thrice a day.

If the illness occurs in hot weather, it may be impossible to produce a cure without the aid of a change in climate. If the child's home is in the city, removal to the seashore may be absolutely necessary; whereas, if the condition develop while at the seashore, removal to a moderate altitude of 1000 or 2000 feet is advisable.

Antidysenteric serum may be given if an examination of the stools reveals the presence of the *Bacillus dysenteriæ.*

CHOLERA INFANTUM.

Definition.—Cholera infantum is an acute affection of infancy characterized by profuse watery purging, rapid emaciation, and profound depression. It is so closely related to that form of diarrhœa due to catarrhal enteritis in adults and to that met with in the ileocolitis of infancy that it scarcely deserves a separate consideration from the standpoint of etiology and pathology, yet its symptom-complex aids us to some extent in making it a distinct entity at the bedside. The malady is almost always met with in the hot months of the year.

Etiology.—The causes of cholera infantum are practically identical with those of enterocolitis, and in a considerable number of cases the bacillus of dysentery (Shiga's bacillus) is the cause. Thus, in a series of cases of typical summer diarrhœa occurring in Baltimore, Duval and Bassett isolated this bacillus from no less than 42 patients. It was found in large numbers in the stools of acute cases and in the mucous membrane of the bowel. They could not find this bacillus in the stools of 25 healthy children, nor in those of patients with ordinary diarrhœa.

Pathology and Morbid Anatomy.—When a case of cholera infantum comes to autopsy the mucous membrane lining the bowel presents a peculiar pallor, which is most marked in the ileum. The colon may show areas of congestion. The tissues of the body are shrunken because of the profuse purging, the body is wasted, the skin wrinkled, and the eyes sunken. The belly may be distended or collapsed. If enterocolitis has been present before the severe choleraic character of the purging is developed, the lesions described under the discussion of that disease may be found.

Symptoms.—Cholera infantum receives its name because its chief symptoms are like those of Asiatic cholera, in that *profuse watery purging, incessant vomiting,* and *collapse* soon develop. The *pulse* rapidly becomes *weak* and *feeble,* the *extremities cold* and the *face pinched,* so that the expression of the child may be shrunken like that of a very aged person. This anxious, pinched look, with a peculiar drawing down of the mouth, as if the child were about to cry, is very characteristic. The fontanelles are depressed.

At first the child may be exceedingly *restless* and *peevish,* but if the attack is severe it speedily becomes *apathetic, listless,* and finally *comatose.* Although the peripheral temperature is low, the *rectal temperature* is often *very high,* even to 105° or 106°. There is often a mottling of the skin, due to the poor capillary circulation. *Thirst is excessive,* and cannot be relieved because of constant vomiting. The urine is scanty or suppressed. As the end approaches, the patient develops irregular respirations, the head is retracted, the temperature is subnormal, and the life ends. When the cerebral symptoms are marked, the condition is called one of "spurious hydrocephalus," a most unfortunate and inaccurate name.

The symptoms are not only those of exhaustion, but of profound toxæmia as well.

Prognosis.—Given a case of well-advanced cholera infantum, the prognosis is very grave. It depends upon the vitality of the child, the severity of the purging and vomiting, the degree of response to treatment, and the age of the patient, for very young infants seldom recover if the disease is once well developed.

Treatment.—The treatment of cholera infantum consists first of all in the absolute prohibition of milk for twelve or twenty-four hours after the patient is first seen. It matters not whether the milk be from the breast or from the bottle, it must nevertheless be withheld from the child. Indeed, it may be said that there is little use in treating these cases medicinally if milk is given. This is particularly so when undigested particles of milk are passed in the stools.

During the period in which milk is forbidden, the child should receive 5 to 10 drops of Valentine's beef-juice in 1 or 2 tablespoonfuls of cool water every hour or two, according to its thirst and its age. In other instances a rump steak may be heated sufficiently to start its juices, then squeezed in a meat-press or lemon-squeezer, and this juice, pure or diluted with cool water, may be given to the child. When these juices cannot be obtained, or where for some reason they cannot be taken, barley-water, rice-water, or a water made by boiling and straining wheaten grits may be used.

Over the abdomen of the child should be applied a spice plaster, composed of a tablespoonful each of powdered allspice, cloves, nutmeg, and cinnamon. This should be moistened with warm brandy or vinegar, and renewed as frequently as it becomes hard or dry.

The child should receive internally $\frac{1}{50}$ of a grain of podophyllin dissolved in a few drops of brandy, and mixed with a little water just before it is taken, every hour until three or four doses have been used; or, instead, $\frac{1}{500}$ of a grain of bichloride of mercury may be given in the same manner. If the vomiting is incessant, it may be necessary to get the solution of bichloride or podophyllin into the stomach by dropping the medicine into the mouth of the child with a medicine-dropper, and only introducing a few drops at a time. It is of the greatest importance that the liver shall secrete and expel bile into the intestine. The appearance of a little bile upon the diaper of the child in place of the colorless liquid which has previously been expelled is a most encouraging sign, and its absence is correspondingly discouraging.

If there is much distention of the abdomen, some relief to the tympany may be given by introducing a rectal tube through which the gas may escape, and if the stools are exceedingly fetid and musty it is often advantageous to irrigate the lower bowel with normal salt solution once or twice a day, inserting into the rectum alongside the nozzle of the fountain syringe a soft-rubber catheter, through which the injected material may readily return. The tube attached to the syringe should, however, pass up into the bowel for eight inches or a foot; while the tube of exit should be just within the sphincter.

If much tenesmus is present, with a tendency to eversion of the bowel, 1 or 2 tablespoonfuls of olive oil containing 2 grains of iodoform may be injected once or twice a day, or even oftener, for its local anæsthetic effect upon the intestine, and after this injection the anus should be supported by the nurse's hand, the ball of her thumb, covered by a napkin, being placed between the buttocks to aid in the retention of the fluid. In children, older than six or eight months, good results sometimes follow the use of weak sulphuric acid solutions. Thus, 1 minim of aromatic sulphuric acid may be given in 4 tablespoonfuls of cool water every two hours. The acid not only acts as an astringent, but it probably also aids in destroying infecting micro-organisms.

When symptoms of collapse ensue the patient should be surrounded by hot bottles, but care should be taken that a peripheral low temperature is not considered as representing the temperature of the central portions of the body, which are often highly febrile. When the peripheral temperature is low, the central temperature high, and the circulation in the peripheral capillaries is impaired, so that the child is somewhat livid and its skin mottled, excellent results will often be obtained by immersing it several times, for a fraction of a minute, in quite hot water for the purpose of producing a certain amount of reaction, relieving internal congestion, and bringing the blood to the surface. If marked fever is present and the extremities are hot, equally good results may come from the use of a cold bath or cold sponging with friction.

Some practitioners have been in the habit of employing minute doses of morphine, hypodermically, or by the mouth. In some instances this method of treatment may be advantageous, but too often it seems to increase the toxæmia from which the patient is suffering.

A very valuable method, which should always be recollected in desperate cases, is the use of normal salt solution by hypodermoclysis.

APPENDICITIS.

Definition.—Appendicitis is an inflammation involving the appendix vermiformis.

History.—Although inflammation of the appendix vermiformis had been described by a number of physicians many years before Reginald Fitz, of Boston, prepared his classical paper on this subject in 1886, the importance and frequency of this condition was not appreciated until he called attention to it.

Etiology.—The causes of appendicitis may be divided into two classes, namely, those that depend upon the anatomical structure and position of the appendix and those that arise as the result of changes in its walls.

The appendix vermiformis is a vestige of what was a large and important portion of the alimentary canal in our early evolutionary ancestry, and, like most vestiges of this character, its tissues are possessed of less vital resistance than are those of active organs. This is the first reason why severe inflammatory processes so often arise in it. Again, it is a sac the neck of which is usually narrower than the rest of its cavity, and as a consequence it happens that infecting micro-organisms find their way into it, and when imprisoned there by swelling of the mucous membrane rapidly attack and destroy the epithelial lining of the appendix and migrate into its walls. As is well known, nothing is more favorable for the growth of micro-organisms than the presence of warmth, moisture, and a condition in which drainage is impossible. Nothing is less favorable to the vital resistance of a part than swelling, with pressure upon the bloodvessels and lymphatic channels. Still another anatomical cause of disease in the appendix is the fact that the mesoappendix is very short, and this results in the appendix being curved or drawn on one side. The mesoappendix carries the chief bloodvessel which nourishes the appendix, and if from swelling, or other cause, the appendix is distorted or twisted, the circulation of blood in the nutrient vessels may be so impaired that the vitality of the part is greatly decreased. Again, in those cases in which the appendix is very long (for it varies in length from one to six inches) the free end may become attached to other parts and become greatly displaced, the appendix itself being twisted. Finally, the fact that the appendix lies near, or on, the ileopsoas muscle may aid in provoking appendicular irritation, and this is probably one of the reasons why appendicitis so often follows violent rowing, golfing, and bicycling.

Among the causes which exist in the appendix itself, in the sense that they are present in its cavity, is the presence of fecal concretions (20 per cent.), and rarely foreign bodies, of which a multitude have been recovered, such as pins, tacks, seeds, and other objects accidentally swallowed. Occasionally intestinal worms, amœbæ, and other parasites have been found. Foreign bodies, however, are comparatively rarely found in this viscus (less than 4 per cent.). Primary tumor of the appendix, of which a number, mainly carcinoma, have been reported, appears to be at least a predisposing cause in some instances.

In a very large proportion of cases, nearly 85 per cent., the micro-organism which is directly responsible for the inflammation is the *Bacillus coli communis*, which is always present in the bowel, and is benign unless the conditions are such as to make it malignant, as when it is confined in a swollen and closed appendix. In some instances the streptococcus or the staphylococcus is the cause. Any micro-organism capable of exciting inflammation upon gaining access to the appendix may be the cause. Thus, the pneumococcus, the pyogenic staphylococcus and streptococcus, the typhoid bacillus, and even the ray fungus may act in this way.

Errors in diet may be a productive factor, for in a certain number of cases of appendicitis there is a history that the patient has, a few hours before the attack or immediately before it, eaten heartily of ordinary or indigestible food.

The age of the patient is undoubtedly an important factor in the development of the malady. Although it is met with in young children and in old persons—that is, after sixty years of age—appendicitis is certainly very much more rare at these periods of life than in the interval. For this there is no adequate explanation. The period of greatest frequency is from the fifteenth to the thirtieth year, and Fitz states that more than half the cases occur before the twentieth year.

Another predisposing factor is sex. About six times as many men as women have appendicitis. This is in part due not only to greater physical activity, but to the more frequent causes of intestinal catarrh in males. It also depends in part upon the fact that women have a second blood supply to the appendix, at least in many cases, namely, an artery which passes from the right ovary to the appendix by means of a fold of peritoneum, which has been called the appendiculo-ovarian ligament. By this means a greater blood supply enables the part to combat infection when the mesenteric vessel is twisted. Appendicitis is more frequent in the well-to-do than in the poorer classes, although it might be supposed that the greater muscular exertion in the latter class would predispose its members to the malady.

Pathology and Morbid Anatomy.—A knowledge of the pathology of appendicitis makes it possible to understand the symptoms which will be described farther on, for these depend, to a large extent, upon the severity of the changes in the appendix, and upon the extent to which adjacent tissues are diseased.

Appendicitis may be divided for pathological study into the *catarrhal, obliterative, ulcerative, gangrenous,* and *perforative* types.

In the *catarrhal type* hyperæmia and congestion of the deeper layers of the appendix may be present, but the chief lesion is in the mucous membrane lining the organ. This results in a free secretion of mucus and in distention of the appendix, the cervix of which is occluded by the swelling of its lining membrane. By this means pain is produced and colic ensues, partly as a result of the endeavor of the appendix to expel its contents into the colon and partly as a result of colic in the large bowel produced by reflex irritation. It can be readily seen that this state may from this point proceed to recovery by a decrease in the constriction of the neck of the appendix and the escape of its contents, or to a far more grave condition dependent upon a continuance of the stoppage, a local and general impairment of resistance, and the presence of a virulent micro-organism. If the attack has been preceded by others, so that the vitality of the part is already greatly impaired and altered, the case is even more grave.

Even in the mild catarrhal form just described there is usually left behind distinct traces of the presence of the acute attack, and this predisposes the patient to another seizure. In those cases in which the catarrhal process is severe and in which the submucous tissues are much affected, it not infrequently happens that after the acute process passes away a subacute or low-grade inflammatory condition ensues, which results in round-cell infiltration and in thickening of the mucous membrane and submucous tissues. The epithelium lining the appendix is desquamated and slight ulceration may occur, with the result that the calibre of the appendix may be greatly decreased in several places, or even entirely closed by the adhesion of its opposing surfaces. In this manner *appendicitis obliterans* is developed.

If considerable quantities of pus, or mucus, are imprisoned back of the constriction, pain, tenderness, and attacks of appendicular colic, or true appendicitis, are prone to recur. When the inflammatory process is severe enough to affect the external surface of the appendix the free end or side of it may become adherent to the bowel or other parts, and by this means the appendix may not only be distorted and held fast, but the infecting germs may pass through its walls and affect nearby structures.

When the *ulcerative type* is present, it is a condition but one degree removed in severity from that just spoken of, and it can be readily seen that no sharp dividing line can be drawn except that there is greater likelihood of the adjacent tissues being infected, owing to the fact that the unprotected submucosa, with its lymphvessels and bloodvessels, is a fair field for infection and for its further spread. Ulceration is particularly prone to occur if a fecal concretion or other foreign body is present which may damage the mucosa. Tuberculosis or typhoid fever may cause it. Sometimes a foreign body may be the cause of such deep ulceration that perforation occurs, or in other cases the floor of the ulcer is unable to stand the stress of accumulated pus or mucus, and the same accident happens.

The *gangrenous type* of appendicitis is the most important of all lesions of the appendix, not because it is the most frequent, but because it not rarely changes the apparently healthy man of one hour into a corpse within a few hours, and this without, it may be, any history of previous attacks which would lead to the belief that the appendix was gravely diseased and unable to resist infection. When this state is present in its most severe type, the appendix undergoes rapid necrosis, its tissues become gangrenous, and it may slough away completely in so short a time as forty hours, so that it may be impossible to find it in the pus which is set free by the surgeon's knife, only shreds of tissue being present. In other cases, however, the process is not so destructive, but nevertheless the organ is utterly necrotic and decomposed. In many instances the gangrenous process may not involve the entire appendix, but occur in one spot, speedily causing perforation and so endangering the life of the patient.

Gangrene of the appendix arises from the invasion of its walls by virulent infecting germs when its vitality is impaired by some unknown cause, or it follows from thrombosis or capillary stasis in its nutrient bloodvessels, whereby the same destruction ensues. Gangrenous appendicitis may be rapid, widespread, and fatal, or the vital forces of the patient may be sufficiently vigorous in the work of resistance to wall off the infected area by lymph, and so confine the morbid process to the immediate neighborhood of the part affected.

The secondary effects of the pathological processes just described are dependent entirely upon their severity and the ability of the patient to protect himself from general infection. The acute catarrhal form rarely leaves behind it anything more than some thickening of the walls of the appendix, with an associated susceptibility to another attack. If all the coats have been involved, the tissues about the appendix become filled with lymph, and the consequent induration may be extreme, the appendix becoming buried in an adherent mass, which may not only hide it from view, and form with the adjacent tissues a matrix, but prevent the surgeon from finding the appendix if operation is attempted.

When the inflammation is severe enough to result in the escape of infection into the adjacent tissues, we not only have pus in the appendix, but in the surrounding parts as well, a perityphlitic or periappendicular abscess, and in those instances in which the appendix is perforated either an abscess is formed and walled off from the general peritoneum by adhesions or a general peritonitis ensues.

The situation of the abscess varies with the direction in which the infection escapes from the appendix. If the infection escapes anteriorly the site of the abscess is often between the navel and the anterior superior spine of the ilium. When it escapes on the surface of the iliac fascia or in the pelvis behind the cæcum, it, of course, lies behind the peritoneum. not in the serous cavity, and retroperitoneal suppuration develops. From here the pus may burrow in as many ways as only pus can burrow, upward to the region of the kidney, downward along the psoas muscle into the thigh, or it may discharge into the bladder, the rectum, or even into the scrotum.

On the other hand, it must not be forgotten that appendicular abscess may develop with very little systemic disturbance and exist for a long time entirely unsuspected, being found by accident, it may be, when seeking for some cause of distress and discomfort with impaired health. At other times such an abscess, after having developed insidiously, produces signs of septic infection the source of which at first cannot be traced.

Finally, attention must be called to the possibility and frequency with which infection of the retroperitoneal lymphatics and of the portal vessels may occur from disease of the appendix. Attention has been called to this by several writers, notably by A. O. J. Kelly, and by Munro in the *Therapeutic Gazette*. Not only may retroperitoneal abscess be due to perforation of the appendix behind the peritoneum, but organisms passing along the mesoappendix produce pus when they reach the connective tissues in the retroperitoneum. Infection of the portal vessels and consequent hepatic abscess is not common, but several cases of this character have been reported.

Symptoms.—It must be manifest from the description just given of the pathological changes that appendicitis is a malady capable of producing very different symptoms in degree and kind. It is not possible, for this reason, to enumerate a set of symptoms present in all cases. There are, however, certain symptoms which are fairly constantly present. The most constant symptom is *pain in the abdomen*. This may be diffuse, or at least the patient may not be able to localize it. Not rarely, if the physician repeatedly asks that it be localized, it is described as being in the "pit" of the stomach or in the epigastrium. If the epigastrium is pressed upon the pain may be increased. This fictitious localization of the pain in the early stages of appendicitis may be most misleading. As a matter of fact, however, it should be most indicative, and every person seized with pain of this character should be suspected of suffering from appendicitis. In some cases the pain is referred to the left iliac region, and it is only when the physician applies pressure to the right iliac area that the patient appreciates that that is the real centre of his suffering, and by manifestations of an unquestionable character shows that the source of pain has been discovered. There is one spot called "McBurney's point," situated two inches from the anterior superior spinous process of the ilium, on a line drawn from this point to the navel, in which pain on pressure can nearly always be elicited. It corresponds rather with the origin than with the tip of the appendix.

The *pain* of appendicitis is usually *severe* and *sharp*, and in some cases agonizing. It is usually *sudden in onset*, and for this, and the other reasons just given, it may be confused at first with renal or gallstone colic. I have seen more than one case in which the diagnosis of acute pleurisy had been made. Occasionally cases are met with in which the pain is less spasmodic and more dull in character, but they are the exception.

Perhaps the most important fact that can be impressed upon the mind of the student in connection with the symptom of pain in appendicitis is this, viz., that the sudden cessation of pain in a case of appendicitis is not a good sign, but an exceedingly bad one in most instances, for it indicates that the distention of the inflamed appendix has been relieved by perforation or gangrene.

When the pain occurs in paroxysms, it is thought to be due to contractions of the appendix—appendicular colic.

Next to pain, the most important symptom in appendicitis is *rigidity* or *fixation of the right rectus muscle.* Barring voluntary rigidity of this muscle, which can usually be prevented by diverting the patient's mind from his abdomen, it is a sign of great reliability, and its degree often measures the severity of the inflammatory process.

Vomiting is very commonly present in these cases. In some of them it occurs so early as to seem to usher in the attack. This is particularly apt to be the case if the stomach has been overloaded with food just before the attack. If the stomach is empty at the time of onset, vomiting is often absent.

The *febrile movement* in a case of appendicitis is rarely very great. The temperature varies from 99° to 101°, and occasionally reaches 102° in adults. In children it may be higher.

The *pulse is quick*, but not very rapid, unless serious abdominal disturbance has already developed. It ranges from 90 to 110 per minute. If it goes higher than this, general peritonitis is probably present. Distention of the belly with gas is usually a late symptom, and if well marked may be indicative of general peritonitis, when it is, of course, a very grave symptom.

After the malady has been present for some days a *swelling in the right iliac region* may appear and be due to pus or to a large protective exudation of lymph. The latter formation is, however, usually met with in relapsing cases rather than in primary cases.

Finally, we meet with cases, usually in women, but sometimes in men, in which there is present a true mucous colitis with, it may be, a chronic catarrh of the appendix. In these persons a tiny discharge of mucopus may daily infect the colon. They are to be considered as cases of chronic catarrhal appendicitis and operated upon, not because the appendix is so gravely diseased as because it causes disorder in the colon.

Diagnosis.—When the question arises as to the cause of severe abdominal pain, an examination of the blood should be made. If a distinct leukocytosis is present, the white cells of the polymorphonuclear group being particularly increased, it is indicative of an acute inflammatory process somewhere in the body, and probably in the appendix, if the symptoms are appendicular. It is, however, a great mistake to allow the determination to do an operation to rest upon this sign, for it has at times proved a "hollow reed." At best it is to be regarded as collateral and not direct evidence of appendicitis.

One of the most important differentiations for the physician and surgeon is that between appendicitis and early typhoid fever. At first glance this would seem to be easy, but those of experience know that it is often difficult, not that typhoid fever often develops suddenly, but that appendicitis may develop slowly during a mild influenzal infection, or during an attack of gastrointestinal catarrh that has been obscure in its nature. Further than this, the lymphoid tissues of the appendix and nearby parts are usually involved in typhoid fever, and may cause appendicular symptoms. (See Plate I.) These localized typhoid lesions may cause pain and tenderness in the right iliac area. The absence of very severe pain, the failure to find the leukocytosis of acute inflammation, the peculiarly coated tongue, the presence of tympany, and the later development of rose spots and the Widal test will prove the case to be one of typhoid fever.

Care should be taken that pain and swelling in this area occurring in one who has tuberculosis is not taken for appendicitis. It is not rare to find these signs in consumptives who are by no means far advanced in their disease. The condition is often one of local tuberculous infection of a chronic type.

Hepatic colic is separated from appendicitis by the presence of a history of previous attacks, by the absence of jaundice, the absence of tenderness over the gall-bladder, and by the fact that in gallstone colic the pain is referred to the chest between the right shoulder-blade and the spine; whereas, in appendicitis it is not so referred.

Renal colic is differentiated from appendicitis by the pain being referred to the testicle, pelvis, or the inside of the thigh; by the fact that the urine contains blood, if not macroscopically, at least microscopically; there is no excess of pain on pressure over "McBurney's point," and there may be a previous history of renal stone. Irritability of the bladder is of no value as a differential symptom, as it is often present in both renal colic and appendicitis. In neither form of colic is leukocytosis marked.

Ovarian or tubal inflammation may simulate appendicitis, but a pelvic examination will usually reveal these states.

In some cases of intestinal obstruction the pain may resemble that of appendicitis, but the presence of obstinate constipation, the development of fecal vomiting, and the discovery of a mass in the belly elsewhere than at the appendix may enable the physician to make a differential diagnosis.

The possibility of acute hemorrhagic pancreatitis being present is to a large extent excluded by the fact that it is a very rare condition, by the site of the pain and the onset of early collapse in this disease.

Osler has pointed out that in persons subject to the erythematous eruptions, severe attacks of abdominal pain may develop which give rise to a diagnosis of appendicitis, which may be excluded only after a careful study of the case with reference to this state and urticaria.

Prognosis.—The prognosis of appendicitis depends largely upon the severity of the condition. Statistics which show that a certain percentage of all cases get well are of interest, but they do not help the physician in an individual case, because definite statements as to the character of the statistics are not given. A series of mild catarrhal cases will give a recovery percentage of 100; whereas, a series of severe gangrenous cases will give a mortality of 100 per cent. That recovery frequently takes place is shown by the fact that about one-third of all postmortems show signs of the existence of appendicular disease at some time in life, yet there may be no history of such an illness.

The substance of our present knowledge is that the prognosis in an ordinary attack of appendicitis is good for recovery from that attack, but that recurrences are likely. In the perforative or gangrenous type the prognosis is always grave and often fatal. Much depends upon prompt surgical interference. If this is delayed, death is the result in the majority of cases of this type.

If, however, all cases of appendicitis of whatever type are considered statistically, it is found that the percentage of mortality is only about 15 per cent. under medical treatment. (See Treatment.)

Treatment.—There is perhaps no more difficult point for decision in medical practice than that as to the treatment for appendicitis. It is impossible to discuss the vast array of arguments for and against early operative interference in this brief space.

Given a case of appendicitis of the acute type, the first thing for the physician to do is to call in a surgeon as a consultant, not as an operator, provided a surgeon qualified to do good abdominal surgery, if it is required, is obtainable. If none such can be had, the patient is far better off without than with operation. If by medical treatment the case can be controlled and carried through the acute attack, the surgeon should not interfere, for the mortality of operation in the acute stage is far greater than it is when the operation is performed in the interval between the attacks.

Fourteen years ago Fitz showed that 40 per cent. die after surgical measures, and 11 after medical treatment; but this does not prove that the latter is better than the former, but rather that the surgical cases did not get to the surgeon unless desperately ill. At present the percentage of deaths in surgical cases is far less, chiefly because they are seen early enough or operation is performed at the time of election; but even at a much later date than that of Fitz's paper we find Caley (1899) recording 98 medical cases with 3 deaths, and 102 surgical cases with 22 deaths. When we consider Sprengel's statistics of 516 cases, 232 of which were operated on in the interval with 2 deaths, and 284 during the attack with 57 deaths and Sahli's 7000 cases treated medically with 90 per cent. recoveries, the value of *delay*, in *mild* cases, is evident.

The plan of treatment in mild cases is as follows: The patient is required to take absolute rest in bed. An ice-bag is placed over the appendix. No purgatives are given nor pain-relieving drugs are to be used unless the pain is excessive, when enough morphine may be used hypodermically to take the edge off of the agony, but never enough to make the patient comfortable or to make him sleep, for such an effect masks the symptoms. No food is to be given by the mouth and no drink is to be taken. If need be, liquid can be given by hypodermoclysis or by rectal injection. If the bowels move, a bedpan must be used. If the pulse is excitable it may be quieted with a little aconite. Under this treatment the acute inflammatory process may be arrested, and the operation can be performed, if need be, after it has subsided. If by the end of twelve or twenty-four hours the symptoms are not rapidly subsiding, it is necessary to operate at once. Fowler has shown that when operation is done within forty-eight hours 83 per cent. recover. When the cases are left to the fourth day 60 per cent. recover; to the fifth and sixth days, 58 per cent.; to the seventh and eighth days, 50 per cent.; and to the ninth and tenth days, only 33 per cent.

The signs, however, which will force the surgeon to immediate operation when the patient is first seen, or if he does not improve under treatment, are: great rigidity of the rectus muscle, persistent vomiting, a rapid pulse (above 110), an anxious facies, and, perhaps, as an indication of some importance, a very high leukocytosis. In such cases the only salvation of the patient lies in immediate surgical interference, and each hour of delay diminishes the chance of recovery. The whole question is one of severity of infection. If there is reason to believe that the tissues are becoming infected and that the local tissues cannot resist the spread of the inflammation, then we must operate.

This question of deferring operation to the interval between the attacks is still under debate. There are some radicals who insist that operation should be resorted to without waiting, and, indeed, before the appendicitis is severe. There are others who are content to wait till the storm is past, and still others who believe that, given a patient who has had but one attack of moderate severity, he may go free until another, or a third attack, makes it evident that a recurrence is likely to take place at any time, when he should be operated upon in the interval. As Dennis well says: "The plan of allowing the simple catarrhal cases which are doing well after thirty-six hours to recover without immediate operation, and relegating them subsequently to the group known as interval cases, and the prompt operation after thirty-six hours when the cases are not doing well, seems to hold out the best prospects of recovery. The pendulum has swung too far toward indiscriminate operation. But now the introduction of the interval operation has brought the pendulum back to swing within the proper limits." (See Peritonitis.)

It must not be thought that patients who have had recurrent appendicitis can be promised perfect comfort by operation, for not rarely, while they recover from the operation, they continue to have tenderness and pain in the right groin for years.

INTESTINAL OBSTRUCTION.

Definition.—Intestinal obstruction is a term applied to a condition of the bowel in which, by reason of some mechanical impediment or intestinal paralysis, the normal movement of its walls and contents cannot take place. In its *acute form* it occurs as the result of no less than six causes: first, congenital *malformation;* second, *invagination,* or telescoping of one portion of the bowel within the other, or so-called intussusception; third, *strangulation* by bands, diverticula, membranous adhesions, or by attachment to other organs, and by the slipping of a coil of intestine through an aperture; fourth, as a result of *twisting* of the bowel, called *volvulus;* fifth, from lodgement of *foreign bodies,* as gallstones, etc.; and sixth, from *intestinal paralysis* and distention.

Chronic intestinal obstruction arises from *stricture,* from *tumors* in the bowel, from tumors external to the bowel, and from the *impaction of fecal masses.*

Congenital Malformation.—Congenital malformations usually consist in closure of the intestinal tube by reason of improper development. Such a closure may exist at any part of the alimentary canal from the œsophagus to the anus. Rarely the bowel becomes strangulated or incarcerated or twisted because of some congenital defect. A common congenital defect is imperforate anus and rectum. Less frequently there is atresia at the pylorus. Not rarely children born with this condition have other congenital defects in the alimentary canal. In the statistics collected by Martin and myself, 28 per cent. of such cases showed more than one point of obliteration. In cases in which the atresia does not exist in the anus or rectum, it is most commonly found near the ileocæcal valve, in the duodenum, or in the sigmoid flexure.

Symptoms.—The symptoms of intestinal obstruction due to congenital causes usually appear after food is first taken. There is no passage of meconium from the anus, and the vomited materials are often fecal in odor and in appearance. Not rarely *violent peristaltic waves* can be seen through the abdominal wall. There is also *pain* and *efforts at defecation.*

Diagnosis.—An examination of the anus or rectum will usually reveal the cause of the trouble. If the finger cannot reach the obstruction, a bougie may discover it. In other cases, water from a fountain syringe, hung, not over two feet, above the patient's buttocks, may be allowed to flow into the bowel to determine its capacity.

Prognosis and Treatment.—The prognosis is, of course, exceedingly unfavorable, but if the closure is near the anus a surgical operation may give relief, and, as all die without operation, the knife should always be resorted to. Death ensues from inanition or exhaustion.

Intussusception.—The invagination in this condition is composed of three layers of bowel. The intussusceptum is composed of the entering and returning layers, while the receiving sheath constitutes the intussuscipiens. To the point where the entering layer is turned sharply upon itself to form the returning layer, the name "apex" is applied. The word "neck" is applied to the ring which results from the flexure formed by the returning layer as it merges into the sheath.

Intussusception may be separated into divisions, according to the severity of the condition, or according to the part of the intestine which is involved. Rafinesque makes three divisions, namely, those which are ultra-acute, death taking place within the first twenty-four hours; those which are acute, death occurring in the first week; those which are subacute, lasting a month and upward. From an anatomical standpoint, intussusception may be divided into the enteric, in which the small intestine is alone involved; the ileocæcal, in which the ileum and cæcum, together with the ileocæcal valve, are turned into the colon; and the ileocolic, in which the ileum is prolapsed through the ileocæcal valve, the latter retaining its proper position, at least for a time. When the condition is called "colic," it involves the colon only. In still other cases, the rectum is solely affected, forming the rectal type of the malady. In the great majority of cases the upper segment of the gut is received into the lower, but occasionally the reverse condition occurs, and when this happens the term "retrograde intussusception" is applied. Double and triple intussusception has occasionally been noted.

Etiology.—The causes of intussusception are not clearly understood, but probably depend upon irregular innervation of the intestine, whereby a sudden, spasmodic contraction of one portion of the bowel occurs, the adjacent portion being relaxed. Intussusceptions of this character are not infrequently met with at the postmortem table, having occurred at the time of dissolution. Polyps of the intestine may be forced along the lumen of the canal, thereby dragging the wall at the point of attachment and causing intussusception.

Frequency.—In 1652 cases of intestinal obstruction, excluding hernia, collected by Leichtenstern and Bryant, 657 cases, or, approximately, 40 per cent., were due to intussusception. It is evident, therefore, that this form of obstruction is not rare. Intussusception occurs most frequently during the first twelve months of life. After the fifth year it becomes comparatively rare until the fortieth or fiftieth year, when it again increases in frequency. The ileocæcal region is the favorite site of invagination at all ages, but ileum invagination is exceedingly rare. If the colic form occur, it is usually at the sigmoid flexure.

Pathology.—The pathological changes resulting from intussusception consist in an extravasation of the blood into the mucous membrane and mesentery of the part affected, and in an acute inflammatory process in the walls of the intestine, which particularly affects the serous surfaces of the entering and returning layers, so that they become glued to one another. Not infrequently, however, this condition does not arise, and adhesions do not form. As a result of the strangulation of the invaginated bowel, it sometimes happens that this portion of the intestine sloughs away, and if sufficiently strong adhesions have formed between the neck and the upper portion of the intussusceptum, the coming away of this slough may result in the recovery of the patient. Very large portions of bowel have been known to be passed in this manner. Pampier has recorded one instance in which 124 cm., Böttcher another in which 112 cm. were passed. In other instances, however, if gangrene of the bowel develops, perforation and general peritonitis ensue.

Symptoms.—These depend upon the degree of constriction at the neck of the intussusceptum. Usually the first symptom is *sudden* and *violent pain.*

This pain sometimes *ceases as suddenly as it begins,* the patient being in comparative comfort. After an interval the *pain returns,* and the paroxysms become violent and prolonged, with shorter intervals of ease. Pressure does not always elicit tenderness; indeed, at times it seems to relieve the pain. *Vomiting* is an even more constant symptom than pain, and usually begins early in the attack, but in adults it may be absent. Of all forms of intussusception the ileum invagination is the one which is most frequently accompanied by early vomiting, chiefly because it produces the most complete obstruction. In children a very constant symptom is the *passage of bloody mucus.* Out of 108 cases, analyzed by Martin and myself, occurring in the first year of life, this symptom was absent in only 4.

Tenesmus and *bearing down* is also commonly met with. In about one-half the cases the tumor can be felt through the abdominal wall, and under the pliable abdominal wall of children it should always be most carefully sought for. Such a tumor is most commonly found when the ileocæcal type is present. Occasionally in the colic type the invaginated bowel can be felt in the rectum. The movement of the bowel may distinctly change the position of the tumor.

Prognosis.—The prognosis in intussusception is not very good. Treated by the expectant method, the mortality is 70 per cent., according to Leichtenstern. The statistics of Martin and myself give a mortality of 90 per cent. The mortality is greater in infants than it is in older persons. The sloughing and discharge of the intussusceptum is always to be considered distinctly favorable, and Martin and myself found that in 408 children in whom sloughing had not taken place 85 per cent. died, while of 149 who passed a portion of the intestine 41 per cent. recovered. Sloughing rarely occurs before the second or third week of the disease.

Treatment.—The treatment consists in the use of a fountain syringe filled with normal salt solution, at the temperature of 105°, and this fluid is to be injected slowly at the rate of 4 ounces to the minute. The pressure in the hydrostatic syringe should not be over two pounds. This method is available only when the intussusception occurs in the lower portion of the bowel. If it is in the ileum it is valueless. If, after pressure has been continued for the period of a half-hour, the tumor does not disappear under gentle manipulation, abdominal section must be resorted to at once. The older statistics in regard to this operation were not very favorable, most of them being gathered in preantiseptic days. At the present time operation gives much more favorable results.

Internal Strangulation.—Internal strangulation by bands is the next most frequent form of intestinal obstruction, forming about 36 per cent. of the classified cases. The condition occurs most frequently in males between the twentieth and fortieth years, and seems to arise in the majority of cases from a former peritonitis; although occasionally the bowel is strangulated by slipping through the foramen of Winslow or through a slit in the diaphragm. Numerous cases of obstruction due to a Meckel's diverticulum have been reported. The diverticulum may become twisted or by adhesions to neighboring structures form a constricting band.

Out of 151 reported cases the small intestine was involved in 133.

Symptoms.—The symptoms consist in *sudden agonizing pain* which is constant, although it has *paroxysmal increments.* The *pulse* becomes *rapid* and weak; the *temperature* is abnormal; the *vomiting is persistent,* and becomes *fecal,* but this condition of the vomit rarely develops before the beginning of the third day. Constipation is present, but fecal matter may be passed from the lower part of the bowel once or twice. If a large coil of gut is involved, a distinct area of distended intestine may perhaps be found. While the presence of this train of symptoms in a young child would be indicative of intussusception, in an adult it is indicative of strangulation by a band, for intussusception is rare in adults.

The only method of treatment which is satisfactory is operative.

Volvulus.—According to Brinton, this condition occurs in 8 per cent. of fatal cases of intestinal obstruction; according to Treves, in 2.5 per cent., and according to Martin and the writer's statistics, in 4 per cent. Sometimes the intestine is twisted for three or four complete turns. The condition occurs most frequently after middle life, and occurs more frequently in men than in women. In 18 cases collected by Haven, 16 were men. In Martin's and my own table of 100 cases, 64 were men. The twist is usually about the mesentery as an axis and involves the small intestine. Occasionally it may appear in the colon; rarely the stomach may be affected. The twisting of the intestine interferes with its circulation, and this, combined with the decomposition of the intestinal contents and the resulting distention, soon produces peritonitis, and even perforation. The abdomen is prone to become immensely distended.

Symptoms.—The symptoms consist in *absolute constipation, vomiting,* and *abdominal distention. Meteorism is constant.* The points in favor of a diagnosis of volvulus are the advanced age of the patient, the fact that the disease usually occurs in a male, that the pain is not as agonizing as in other forms of obstruction, and that the obstructed bowel is greatly distended.

Prognosis.—The prognosis is much more favorable than in other forms of intestinal obstruction. When intestinal obstruction is due to paralysis, the cause is most frequently some injury or an operation upon the abdominal contents. The bowel is simply dilated or kinked, and the failure in peristalsis is due to paralysis of its muscular fibres. This is the type of obstruction which all abdominal surgeons greatly fear as a sequence of operation upon the peritoneal contents.

Treatment.—When the volvulus is due to paralysis after operation it is to be treated by the administration of concentrated salines repeated until the bowels are moved. When distention has reached a very great degree and vomiting is present, salines are no longer useful. The rectal tube should be passed in the hope of exciting peristalsis and drawing off gas. The patient should be freely stimulated and the rapidly interrupted faradic current should be applied to the abdominal wall, or one pole may be placed in the rectum and the other passed to and fro over the abdomen.

If the condition is not due to paralysis after operation purgatives are absolutely contraindicated, and enemata can be of no value. Such cases should be subjected to operation.

Obstruction from Foreign Bodies arises from such articles as coins, pebbles, knives, and scissors, gallstones and enteroliths. While gallstones are usually small, they at times may be very large, and are often greatly added to by concretions. Thus, Leichtenstern states that one such stone was five inches in circumference, and he describes an enterolith nine inches in circumference. Such a stone is usually formed by concretions about a foreign body, as a cherry-stone. Cases of intestinal obstruction of this character are, however, very rare, about 0.2 of 1 per cent. of all cases. The obstruction is usually found in the small intestine, sometimes at the ileocæcal valve, and occurs more frequently in females than in males.

DUODENAL ULCER.

Ulcer of the duodenum is probably a more frequent condition than is generally supposed, and in some cases is associated with ulcer of the stomach. The proportion given by Burwinkel of gastric and duodenal ulcer is 12 to 1. On the other hand, von Wyl found only 3 duodenal ulcers in nearly 13,000 postmortem examinations, and Kinnicutt, in an analysis of 30,000 postmortems, places its frequency at 0.4 of 1 per cent. The condition may arise at any period of life, but is most frequent between the tenth and fortieth years. Hahn has recorded a case in a child only a day and a half old. Such an ulcer must have been antenatal.

Unlike gastric ulcer, the great majority of duodenal ulcers are found in men. Murphy, of Chicago, quotes Laspèyres as stating that men are affected two or three times oftener than women. Thus, Krauss, in 64 cases, found the ratio to be 10 to 1; Lebert, in 39 cases, 4 to 1; Trier, in 54 cases, 5 to 1; and out of 176 cases collected by Weir, 144 were in men.

Etiology.—Among the causes of duodenal ulcer may be mentioned burns, which in some unknown way produce ulceration in this portion of the bowel. Renal disease, which occasionally results in the ulceration of the large bowel, may also cause this lesion in the duodenum. Pulmonary tuberculosis, which produces its lesion by infection of a solitary follicle, and diseases of the heart and liver, which result in impairment of vitality in the intestinal wall, so that localized infections may occur, are also causes.

As with gastric ulcer, so with duodenal ulcer, a large number of theories have been advanced as to its direct causation. Most authorities at the present time believe that it is due to erosion produced by the gastric juice, the vital resistance of the part having been diminished by inflammation or circulatory changes. That acidity of the gastric contents may so result seems likely, from the fact that ulcer most frequently occurs in the duodenum near the pylorus, at a point where the acidity of the gastric juice may be but little modified by the alkaline secretion which it would meet a few inches farther on in the bowel.

It would seem probable, however, that a number of factors may produce this form of ulcer, acting in some cases together and in other cases singly. These factors are well summed up by Murphy and made into four divisions:

hyperchlorhydria, local infection, embolism with thrombosis, and foreign bodies. To these four divisions Murphy would add a fifth, namely, disorders of the organs of elimination, as in burns of the skin or other serious lesions in this part of the body, as pemphigus and erysipelas, and in other cases renal disease.

Pathology and Morbid Anatomy.—Duodenal ulcers are usually single, but they may be multiple. Out of 233 cases collected by Collins, 195 were single.

Ulcer usually occurs in the first part of the duodenum. Out of 149 cases collected by Perry and Shaw, the first portion of the duodenum was involved 123 times, the second portion 16 times, and the third and fourth portions twice. These statistics agree with those which have been collected concerning the area and greatest frequency of perforation complicating ulcer of the duodenum.

When perforation occurs, it takes place nearly twice as often in the anterior as in the posterior wall, still more rarely in the superior wall, and almost never in the inferior wall. Perforation occurs much more frequently in ulcer of the duodenum than in ulcer of the stomach, if we can rely upon the statistics which have so far been collected. Thus, in 404 cases mentioned by Chvostek, Collins, and Oppenheimer, perforation took place in 246. On the other hand, it must be remembered that a very large number of cases of duodenal ulcer are not recognized unless perforation does occur, and it is highly probable that this accident is far less frequent in duodenal ulceration than these statistics would indicate, because it is a well-known fact that duodenal ulcer is a condition most difficult to recognize unless it is found in the course of an abdominal section, and, further, it is well known that these ulcers frequently heal. Thus, Perry and Shaw found evidence of repair in half of their cases, and Krug, in 1220 autopsies, met with 30 cases of healing of duodenal ulcer.

As in perforations of the stomach, so in perforation of the duodenum, a general peritonitis ensues, or a localized peritonitis may develop, the extravasated materials being walled off from the rest of the peritoneal cavity by an inflammatory exudate. As with gastric ulcer, so again with duodenal ulceration, adhesions may take place in neighboring organs, and perforation may take place into them. Thus, it has occurred that the duodenum has been perforated, and so permitted its contents to enter the gall-bladder, the abdominal aorta, the vena cava, the portal vein, the superior mesenteric vein, and the hepatic artery; but Murphy asserts that a gastroduodenal fistula has never been found as a result of perforation of a duodenal ulcer. In some instances perforation of the duodenum has resulted in subphrenic abscess.

Symptoms.—The symptoms of duodenal ulcer, unless the ulceration proceeds to hemorrhage or perforation, are too indefinite to make a positive diagnosis possible in most cases. Indeed, in fully half the cases in which duodenal ulcers are found at autopsy, there have been no symptoms during life which have raised suspicion of its existence.

When the symptoms do occur, they so closely *resemble those of gastric ulcer* that a differentiation may be impossible. There is *pain* and *vomiting*, and if a bloodvessel is ulcerated there may be *hæmatemesis* or *bloody stools*.

The pain is usually much less severe than in ulcer of the stomach, but at times it may be agonizing. There seems to be a general consensus of opinion that it rarely radiates toward the back, as does the pain of gastric ulcer, but cases have been reported in which pain in the neighborhood of the shoulder-blade has been a pronounced symptom. It differs from gastric ulcer in that the taking of food is not immediately followed by pain, the pain being delayed for several hours after a meal, then developing when the food enters the duodenum from the stomach. Moynihan says that the nearer the ulcer is to the stomach, the sooner is the pain developed.

The hemorrhage, when it takes place, may be sufficiently profuse to cause death, or it may be small in amount and be frequently repeated, in which case death may ultimately occur from exhaustion. Vomiting rarely occurs except when the stomach is overloaded, or when blood enters it from the duodenum.

Perforation of a duodenal ulcer may be the first manifestation of any lesion in this portion of the bowel. According to Schwartz, patients suffering from perforation of the duodenum were healthy in 20 out of 25 instances prior to the accident, and in Weir's 51 cases they were without gastric or duodenal symptoms in 25 out of 34. So, too, in Perry and Shaw's 151 cases, 91 per cent. presented no evidences of disease until perforation or hemorrhage developed.

While cases of ulcer may recover, the tendency is to a progression of the disease. On the other hand, progress does not necessarily mean early death, for Chvostek has reported a case in which there had been present symptoms of duodenal ulcer occasionally for thirty-nine years.

Symptoms of perforation of duodenal ulcer are *severe epigastric* or right *hypochondriac pain*, followed it may be by *collapse*; the symptoms resemble, perhaps, acute hemorrhagic pancreatitis, and death occurs sometimes as early as twenty-one hours after the accident. The symptoms of general peritonitis soon develop, or, if the lesion is localized by adhesions, localized peritonitis is found, as already stated. Leukocytosis is usually marked.

Diagnosis.—The tests for minute traces of blood in the stools, which are described in the article on gastric ulcer, may be used in these cases.

Duodenal ulcer must be separated from gastric ulcer, if possible. In the majority of instances this, perhaps, cannot be done unless there are bloody stools, the character of the blood being dark and tarry, owing to its alteration by the intestinal juices. If it is bright in character, it probably comes from ulceration of a lower portion of the bowel. Another important point in the diagnosis is the period at which pain develops after food is taken, for, as already pointed out, the development of pain is delayed in cases of ulceration of the duodenum, and is immediate, as a rule, in gastric ulcer. While von Wyl admits that it is impossible to make a differential diagnosis in 90 per cent. of the cases, he gives us the following points which are of value in differentiation:

Gastric Ulcer.	*Duodenal Ulcer.*
1. Usually in women twenty to thirty-five years of age.	1. Most frequent in men.
2. Pain comes on soon after eating.	2. Pain two to four hours after eating, and located in right hypochondrium.
3. Pain lessened by vomiting.	3. Vomiting does not relieve pain.
4. Vomitus contains mucus, food remnants, and often blood.	4. Vomiting more rare than in gastric ulcer, and does not often contain blood.
5. Severe dyspeptic symptoms usually present.	5. Dyspeptic symptoms slight.
6. Melæna rare.	6. Melæna comparatively frequent.

Gallstone colic is to be separated from duodenal ulcer by the fact that hemorrhage does not occur in gallstone colic, and by a previous history of gallstones; but it is to be remembered that the absence of a history of jaundice is of little value, for jaundice is not a constant symptom in cholelithiasis. As Murphy well points out, jaundice was present only 16 times in 400 cases of cholelithiasis operated on by him.

Acute fat-necrosis often can not be differentiated from duodenal ulcer with perforation. As a rule, the vomiting in fat-necrosis is more persistent, and the depression or collapse is more prompt and severe. A high-pitched percussion note is found in the right hypochondrium in fat-necrosis, but this area is usually flat in perforation of the duodenum, unless peritonitis has already progressed to the stage of general tympanitic distention. In fat-necrosis there is an absence of leukocytosis; in perforation there is a marked leukocytosis. In fat-necrosis there is usually no rise in temperature; in perforation there is not infrequently a primary rise.

Intestinal obstruction may closely resemble perforated duodenal ulcer. The pain in ulcer is constant; in obstruction colicky; there is hyperperistalsis in obstruction; there is an absence of peristalsis in perforation; there is absence of leukocytosis in obstruction; there is marked leukocytosis in perforation. In both there is usually a history of constipation.

As illustrative of how difficult it is to make a correct diagnosis, even when perforation occurs, Moynihan tells us that in only 12 out of 51 cases of duodenal ulcer was a correct diagnosis made before operation, and that in 49 cases of perforated duodenal ulcer 18 were operated upon for appendicitis.

Prognosis.—The prognosis, like that of gastric ulcer, is not good for recovery. In perforative cases, if operation is not performed, the outlook depends entirely upon whether the infective material is walled off by adhesions. If this is the case, and a subphrenic abscess is formed, much depends upon the point at which this abscess ruptures. If the extravasated material is not confined by adhesions, death occurs from general peritonitis.

When the condition is recognized and operation is performed, the prognosis is much more favorable. The difficulty is that in many cases the diagnosis is so obscure that operation is not performed until so many hours have passed that recovery is impossible. Thus, out of 51 cases operated upon in Moynihan's collection there were only 8 recoveries, and in 20 cases collected by Darras only 3 recovered. In 79 cases collected by Weir and Foote the mortality after operation was 71 per cent.

Treatment.—The treatment of these cases consists in absolute rest in bed. Aside from these measures it is purely surgical. If perforation has taken place and surgery cannot be resorted to, then there is nothing left for the physician but to relieve pain by the use of opium and to hope that the inflammatory process may be localized.[1]

ENTEROPTOSIS.

Definition.—Enteroptosis is a condition in which the intestines fall to a lower level than that which they normally occupy. Not only the intestines, but the stomach, liver, spleen, and kidneys may be displaced downward, the displacement being due to stretching or relaxation of the mesenteric and peritoneal ligaments and to laxity of the abdominal wall, so that it fails to support the contents of the belly cavity. Of the various names which have been applied to this state, other than enteroptosis, may be mentioned splanchnoptosis, visceroptosis, and "Glénard's disease." When the stomach is chiefly affected it is called gastroptosis. Enteroptosis is a condition, not a disease.

Etiology.—Glénard thinks that overloading the transverse colon with feces may cause so great a strain upon that portion of the mesocolon which supports the large transverse bowel, particularly at its right flexure, that this part may sag and so predispose the rest of the colon to drop downward, drawing with it other parts. The objection to this argument is that the right flexure of the colon is practically never loaded heavily with feces, or, at least, the instances in which it is so loaded are far more rare than is enteroptosis. Further than this, the ligaments concerned in the support of the abdominal contents are not the chief source of support. Schwerdt states that they do not bear more than one-eighth of the weight. The upper organs are buoyed up by the lower ones, provided these are retained in a normal position. Although constipation may be a minor factor in producing this state, the chief factors are relaxation of the abdominal wall and the loss of fat produced by an acute illness or some chronic disease, and occasionally by old age. This affection is not uncommon in the insane, particularly when chronic constipation, inactivity, and wasting are associated. The relaxation of the abdominal wall may also be due to loss of fat and to repeated pregnancies, particularly if the woman has, by wearing corsets, weakened her abdominal muscles and then had them subjected to prolonged distention in pregnancy. Occasionally the rectus muscles not only atrophy, but separate. I have in my ward as I write a woman who has this very condition. (Figs. 84 and 85.) Cases occasionally arise in which enteroptosis follows the removal of a large ovarian cyst or of ascites.

To appreciate the failure on the part of the abdominal muscles in a well-developed case of enteroptosis, it is only necessary to stand behind the patient and place the palms of the hands upon the lower zone of the abdomen, pressing upward and inward, when the entire weight of the abdominal con-

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[1] The literature of this subject, which is of increasing importance, can best be obtained by consulting the valuable papers of Weir in the Transactions of the American Surgical Association, and of Murphy in the American Journal of Obstetrics for December, 1902. From these contributions much of the information in this article was obtained.

FIG. 84

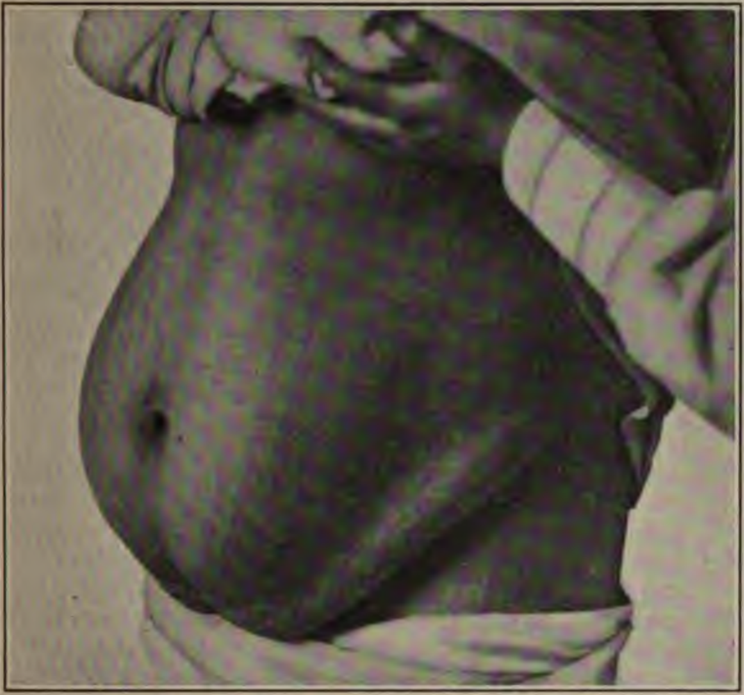
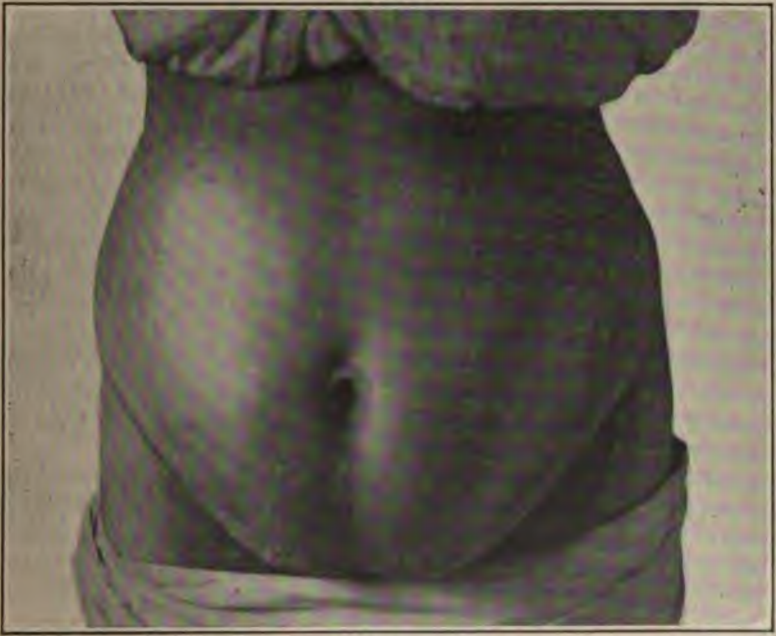


FIG. 85



Enteroptosis due to relaxation of the abdominal wall. Hornets'-nest belly.

tents may be felt resting upon the hands. The complete inability of the abdominal wall to give support is then appreciated.

Enteroptosis is far more frequent in women than in men. Glénard found it in women in 306 out of 404 cases.

Symptoms.—The symptoms in many cases are by no means definite. The patient is often regarded as a *chronic dyspeptic*, as, indeed, she is. There is more or less *constant discomfort in the abdomen*, and the intestines may be in a state of *peristaltic unrest*, so that *borborygmi* and *rumbling* are annoying. At times the bowels seem hyperæsthetic, and the patient complains, not of pain, but of a sense of movements which in health are never felt. Some patients describe the sensation as if their abdominal contents were "falling out" of them. Some have a distaste for food; others crave it, with the hope that it will relieve the sense of emptiness, and then regret taking it because its presence in the displaced stomach increases the distress. *Constipation* is usually persistent, and the use of purgatives may serve to cause a great increase in rumbling without causing a satisfactory evacuation. The reason for this is evident, for the fallen bowel presents sacculations or depressions that act like a plumber's trap and prevent free progress of the contents of the intestinal tube.

Associated with these symptoms there is often a good deal of nervous unrest and *mental depression*, and not rarely some *vertigo* on changing the posture of the body. There may also be cardiac palpitation and breathlessness.

An examination of such a patient will reveal on inspection, provided a certain degree of leanness or emaciation is present, that the abdominal wall is thinner than normal, that it is relaxed, and that when the patient stands erect its muscles are soft and without tone. In health palpation of the abdominal wall reveals some resistance, whereas in this state it yields readily to pressure like the side of a partly filled water-bag. Inspection not rarely reveals the fact that the zone of the abdomen between the ensiform cartilage and the navel is empty, and that below the navel the abdomen is unduly prominent and sags.

The appearance of such a patient is often noteworthy, for emaciation may be so marked as to raise the suspicion of malignant disease, a suspicion which is increased by the anæmia which is present. Rarely the patient suffering from enteroptosis may develop jaundice because adhesions constrict the bile-ducts.

If the patient with enteroptosis be placed upon the back in a good light, and the abdominal wall observed at a distance, peristaltic waves may often be seen traversing it. Tapping the knuckles of intestine through the abdominal wall with the finger-tip will increase or arrest these movements for a moment.

Deep palpation may reveal the liver much lower than normal. That this is not due to an enlargement of this organ may be proved by the discovery that as its lower border passes down into the abdomen its upper border also becomes lower, the actual area of liver dulness on percussion being the same as in health. The liver is, however, rarely out of place, except in extreme cases.

Distention of the stomach with gas from a Seidlitz powder, or by pumping air into it with an atomizer bulb attached to a tube, will reveal its abnormal position, and if it is carefully outlined it may be found that this viscus occupies a more vertical position than in health, the pylorus being greatly displaced, while the cardiac portion is in a relatively normal posture because it is more firmly suspended. The cardia is very rarely greatly displaced, but Steele has reported five such cases in a comparatively short time of observation. The gastrodiaphane of Einhorn may be used for the purpose of defining the site of the stomach. Ptosis of the stomach is not rarely associated with dilatation and with motor insufficiency. (For the measures by which the presence of gastroptosis can be determined see article on Gastric Dilatation.)

The spleen is very commonly displaced. It may be well forward in the median line, or it may fall more directly downward and be found as low as the pelvic organs

Nephroptosis is described in the section devoted to Diseases of the Kidneys.

During the performance of deep palpation there can sometimes be felt a moderately firm mass lying transversely in the abdomen in the epigastric area. This is said by Glénard to be the colon, but if this be the case the colon is not much displaced, and is certainly contracted rather than dilated—a condition opposed to that stated by Glénard to be usually present. Ewaldi believes that this mass is the pancreas which has been uncovered by the enteroptosis.

Auscultation of the belly in these cases often reveals a large number of liquid sounds, and if the patient is shaken there may be heard succussion notes and sounds which may be called "slopping."

While some patients with moderate enteroptosis present many of the symptoms just described, it is a fact worthy of note that others with very marked falling of the abdominal contents often have no complaint to make of the abdominal state, and if they are told of it at once become mentally "centred" on their alimentary tract, and, if already neurasthenic, speedily drive themselves and their medical attendant almost demented by their constant discovery of new symptoms.

Treatment.—The treatment of enteroptosis is manifestly to be directed to the support of the displaced organs and their replacement. Not rarely, if the physician stands behind the patient and presses upon and lifts the abdominal contents by pressing the hands in front of the abdomen, relief from the sense of abdominal relaxation is at once noticed.

The adjustment of a properly filled abdominal belt or binder is, therefore, a valuable aid in this condition. It should be applied every morning before rising and not until after the patient, by gentle strokings with his hands, has placed the abdominal contents at about the proper level. Its greatest pressure must be exercised inward and upward in the zone below the navel. Sometimes the use of a broad flannel binder about the lower zone may be sufficient, but the support must be upward as well as inward.

Great care should be taken as to diet. Starches and milk, both of which are prone to produce flatulence, should be avoided. If starch is used in an easily digested form, as rice and cornstarch, some taka-diastase should be given with it. Cheese and beans are absolutely forbidden, and fats are also harmful. Small quantities of green vegetables may be taken, and roast or broiled beef and mutton allowed at each meal. Eggs are also permissible. The patient should be warned against eating heavily at any one time, since an overweighted stomach or colon will make the ptosis much worse. If gastric dilatation exists, lavage may be useful.

In the way of drugs, there are only three which produce much benefit, namely: nux vomica in full dose—say, $\frac{1}{4}$ grain four times a day; extract of physostigma in the dose of $\frac{1}{8}$ grain four times a day, and capsicum, 1 grain three or four times a day. Sometimes it is well to combine all of these in one pill or capsule.

In the way of digestants, hydrochloric acid and pepsin, soda and pancreatin, and taka-diastase are to be employed. As laxatives, cascara sagrada and aloes may be used, 1 grain of the extract of the former and $\frac{1}{10}$ grain of aloin being given once, twice, or thrice a day, according to the obstinacy of the bowels. The bitter fluid extract given in capsules is the best preparation to employ.

In cases in which the symptoms are so severe as seriously to impair health and comfort and even the chances of life, operative interference is indicated, the displaced organs being fixed by suturing. Up to the present time quite a number of such cases have been operated on by different methods. Duret, in a case in which the stomach came within four inches of the pubis, placed stitches through the lesser curvature of the stomach, then through its anterior wall, and made it fast to the peritoneum of the anterior abdominal wall. Recovery followed. In other cases the intestines have been raised by taking a reef in the mesentery. Rovsing has fastened the stomach by three stout sutures passed through the abdominal wall and through the outer coats of the stomach, with the result that the patient gained forty pounds in weight. Beyea has taken tucks in the gastrophrenic and gastrohepatic ligaments, with good results, and Webster has treated a large number of cases by excising the tissues between the recti muscles and then stitching the edges of these muscles together, thereby affording support for the abdominal contents.

COLITIS.

Acute Colitis.—This is a very common condition and follows exposure to cold, particularly if the abdominal contents have been the parts chiefly deprived of warmth. The inflammatory process chiefly affects the lower part of the colon and extends to the rectum as well, so that proctitis is developed. The primary hyperæmia of inflammation is followed by an increasing secretion of mucus, with the throwing off of dead epithelial cells mixed with white and red blood corpuscles which have escaped from the engorged vessels. If the process is very severe, a suppurative state may be developed. In most cases the tendency is to rapid recovery, but if the inflammatory process persists, one of two conditions may be developed, either small areas of necrosis or ulceration occur, or there is deposited in the submucous tissues a considerable amount of connective tissue, which may by its contraction impair the function of the glands and perhaps narrow the calibre of the bowel.

Symptoms.—The symptoms consist in *severe abdominal pain*, with *tenderness* in the region of the sigmoid flexure, and in *frequent movements of the bowels*, which movements soon become very small, so that they finally consist in nothing but a little mucus, which is passed with *great tenesmus.*

Treatment.—The treatment consists in the use of absolute rest in bed. The application of a mustard plaster over the sigmoid flexure and the injection into the bowel of 4 ounces of starch-water, with 40 grains of potassium chlorate and 30 drops of deodorized laudanum, every three or four hours.

Acute infectious colitis has already been considered in the article on Dysentery.

Mucous Colitis. Definition.—Under the name of mucous colitis physicians meet with a condition which is, next to dysentery, the most common affection of the colon, and in temperate zones is more frequent. It affects persons suffering from neurasthenia, in the great majority of cases, and is met with in the overworked or overwrought of both sexes, but most frequently in women of from twenty to forty years of age.

The affection is a chronic one, often lasting for several years, and during its continuance causing a great impairment of nutrition and much general ill-health. Not rarely the irritable state of the colon causes constant abdominal distress. Severe colicky pain is also present, and a state of hyperperistalsis of the small bowel exists, so that food is often hurried on into the large bowel before it can be digested and absorbed, the patient suffering from lienteric diarrhœa, not because the digestive power is impaired, but because the food does not remain in one part of the small bowel long enough to be digested. The stools are not as frequent as those of other kinds of chronic diarrhœa.

Mucus in considerable amount is often passed, and this mucus may be so thick that it resembles a false membrane, whence the term "mucomembranous enteritis." Not rarely the patient has excessive peristalsis every time food is taken.

Blood is almost never passed unless there are hemorrhoids which bleed. There is no fever, but profound mental depression. Areas of marked tenderness can be found in the abdomen on palpation, and the cutaneous sensibility is often increased.

Treatment.—The treatment of mucous colitis, while it is not capable of producing, in the majority of cases, very marked improvement within a short time, is nevertheless successful in a large proportion of patients, provided that it is carefully and persistently carried out, and if the patient's mode of life and her diet is arranged in such a way as to be favorable in their effects. As the majority of these patients have been subjected to nervous stress and are neurasthenic, it is essential that they shall be subjected to the rest cure, in order that by re-establishing nervous tone and equilibrium a normal intestinal peristalsis and normal digestive functions may be established. Without rest in cases of this character other treatment is commonly useless.

In order that the greater part of digestion and assimilation may be carried out in the stomach and duodenum, foods easy of digestion and readily assimilated should be given, and should consist chiefly in proteids, that is, broiled or roasted meats. Green vegetables and fatty foods should not be allowed. Easily digested starches, such as rice and cornstarch, may be given, provided that pancreatin or taka-diastase is given with them to hurry their digestion. All vegetable foods which leave a bulky residue should be forbidden, as, for example, oatmeal and wheaten grits. The patient should take liquids in small quantities frequently, rather than in large quantities at long intervals, and should avoid taking liquids with her food. She should also avoid taking liquids before going to bed at night, as not infrequently liquids taken at this time seem to lie in the bowel unabsorbed, and on the assumption of the erect posture by the patient in the morning a morning diarrhœa is developed. All fatty articles of diet should be avoided.

Continuous counterirritation, produced by frequently repeated applications of tincture of iodine over the whole abdominal surface, should be maintained, and if there is much tenesmus a suppository containing 5 to 10 grains of iodoform may be inserted into the rectum in the morning after a movement of the bowels, not only for its local counterirritant effect, but for the beneficial influence of the iodine, when absorbed, upon the catarrhal condition of the bowel. In some cases where the colon is chiefly at fault, clysters of 1 or 2 quarts of hot normal saline solution, or of pure water containing 20 grains of sulphocarbolate of zinc to the pint, should be gently given, care being taken that the fluid does not run in so rapidly as to irritate the bowel. The patient should lie on the left side until the sigmoid flexure is filled, then turn on the back while the transverse colon is filled, and perhaps after this turn on the right side with the hope that the fluid will enter the ascending colon.

In those cases in which there is a history of repeated mild attacks of appendicitis, or of pain in the right iliac region, appendectomy sometimes produces excellent results, the chronic colitis being due to the infection of the colon by small quantities of pus which escape from the appendix. Probably the enforced rest which follows an operation for the removal of the appendix also is advantageous in producing a cure in these cases.

Follicular and Croupous Colitis.—*Follicular colitis*, sometimes called nodular colitis, is a form of inflammation of the colon characterized by marked swelling of its solitary glands, or lymph nodes, rendering these structures unusually protuberant. After this primary stage of enlargement necrosis and sloughing ensue, leaving round ulcers, which are frequently numerous. By the failure of these to heal the more chronic state of ordinary ulcerative colitis is developed.

Ulceration of the colon is, of course, also due, in many cases, to the typhoid bacillus, the tubercle bacillus, the *Amœba dysenteriæ*, and sometimes to infection by Shiga's bacillus. It also develops as a terminal infection in some cases of chronic renal disease. Ulcerative processes in the intestines are very common in the insane, and they have been associated with locomotor ataxia.

Under the name of *croupous colitis*, a condition exists in which the mucous membrane of the colon becomes engorged and coated with a false membrane, and the underlying tissues becoming filled with dead leukocytes and fibrin. As in the small bowel, so here, this false membrane may be widely diffused or occur in patches. After the formation of the membrane the disease either disappears by the exfoliation of necrotic material or the deeper coats of the bowel are affected, so that areas of submucous tissue become necrotic and are passed in the stools as sloughs. At the site of these sloughs healing by cicatrization develops, or the process extends still more deeply to the peritoneum and causes serious secondary lesions. In this way is formed a necrotic colitis, the ulcerated areas being gangrenous in appearance and of great size. A large number of pathogenic organisms have been found in the bowel in such cases.

Treatment.—This consists in following a plan identical with that advised for mucous colitis, and in addition in giving an injection of nitrate of silver in the strength of 40 grains to the quart each evening in place of the normal saline already spoken of.

Pseudomembranous Colitis.—This is a condition in which not only the large intestine, but the small bowel as well is affected by a superficial necrosis, which may be diffuse, but is more commonly distributed in patches. The process closely resembles croupous colitis, and by some writers the two conditions are held to be identical. The false membrane consists of dead epithelium, mucus, fibrin, and white blood cells which have passed out of the bloodvessels. In some cases the false membrane is almost purely mucin-bearing and quite fibrin free; the latter element is only exceptionally abundant. Not rarely the submucous tissues may be infiltrated by serum and leukocytes.

Pseudomembranous enteritis develops in the course of a number of the acute infectious diseases, in pyæmia and septicæmia, in persons who suffer from chronic Bright's disease, and occasionally after the taking of poisons which cause gastrointestinal irritation. It is important to recall the fact that this pseudomembranous condition is not diphtheritic in the sense that it is due to the Klebs-Loeffler bacillus, and that the proportion of fibrinous exudate is far less than in the membrane of that specific disease called diphtheria. Perhaps the most common cause of this lesion is the ingestion of poisonous quantities of arsenic, for this drug is eliminated by the mucous membrane of the alimentary canal and in the process a necrosis of the lining epithelium takes place.

DILATATION OF THE COLON.

Hale White places dilatation of the colon in four divisions. The *first* of these is that type of dilatation which is due to acute distention from the accumulation of gas. This is not rarely met with in severe infectious diseases, as in the pneumonia of drunkards and in severe cases of typhoid fever with toxæmia. The distention of the colon under these circumstances often interferes with the action of the lungs and heart by mechanical pressure against the diaphragm. The zone of the abdomen between the umbilicus and the ensiform cartilage, and between the right and left hypochondrium is distinctly bulging and tympanitic on percussion. Tympanites of this kind possesses a double significance: first, it is an evil in itself by reason of the pressure which it produces, and, second, its presence is evil in that it indicates a lowered vitality and an inability of the intestine to expel gas which otherwise would not be allowed to accumulate, and which in health would not form.

The *treatment* of this form of tympanites consists in the application of a hot turpentine stupe over the abdomen and the injection into the rectum of 6 ounces of milk of asafœtida containing 1 drachm of oil of turpentine, the two fluids being thoroughly mixed in order to prevent the turpentine from damaging the bowel. In other cases, where it is considered advisable to stimulate the circulation at the same time that the gas is expelled, and when it is feared that the turpentine may be absorbed and irritate the kidneys, excellent results will follow the use of this quantity of milk of asafœtida with the addition of ½ to 1 ounce of Hoffmann's anodyne. When obstinate constipation is present, and the sigmoid flexure is filled with feces, an ordinary soapsuds enema, followed by 1 ounce of sulphate of magnesium in 4 ounces of water and 2 ounces of glycerin, may be injected. A few years ago it was suggested by Ogle, and others, that puncture of the bowel through the abdominal wall should be performed in those cases in which the gas could not be dislodged and when it was causing dangerous pressure. While this advice is theoretically good, practically it is of little value. I have tried it in a number of instances, and it has either failed entirely or has permitted but a small quantity of gas to escape from a single knuckle of intestine, the bowel contracting in such a manner as to prevent most of the gas from finding its way to the aspirating needle. If a fine needle is used but little gas can escape, while if a coarse needle is employed a sufficiently large puncture may be made in the bowel to permit of the escape of gas or liquid into the peritoneal cavity after the needle is withdrawn.

The *second group* of cases depends upon the accumulation of foreign bodies. These are so rare in human beings as to be scarcely worthy of consideration. Occasionally, however, the dilatation may be due to the presence of enormous gallstones which have been still further increased in size by fecal additions. Such cases are to be treated by operation.

The *third form* is that due to obstruction of the lower part of the colon, so that fecal accumulation and secondary ulceration may occur. The obstruction may be due to volvulus, to a band or to a coil of adherent small intestine. It is also due to stricture or to syphilitic, cicatricial, or neoplastic growths, particularly cancer. These cases are very rare, and the treatment is operative.

Finally, in the *fourth type* we find cases of so-called idiopathic dilatation of the colon, which are also exceedingly rare. Many years ago Formad reported an extraordinary case of this character, and Hale White has collected several from literature. In most of these instances the enormously dilated colon is loaded with accumulated fecal matter.

Treatment can be of little value in the last type of cases, for a congenital defect in the muscular and other tissues forming the wall of the intestine is responsible for the condition. Relief may, perhaps, be given by making an artificial anus at the sigmoid flexure.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—Peritonitis is a term applied to inflammation of the serous membrane, the peritoneum, lining the abdominal cavity and covering in its reflections the organs which this cavity contains.

Etiology.—Within comparatively recent years it was generally considered that acute peritonitis was usually idiopathic, but with an increasing knowledge of the methods by which infection occurs, we have come to learn that most, if not all, cases of peritonitis are due to an infection which has come to the peritoneum through primary disease or the presence of infecting organisms in other organs. While we are not in a position to deny the existence of idiopathic peritonitis, we should, nevertheless, always doubt it, and use every effort to discover the source of the infection which is present, even if it is not readily found. Nearly half a century ago Habershon found, in an analysis of 501 autopsies after death from peritonitis, that over 50 per cent. resulted from some primary disease not involving the peritoneum, and more recently Kelynack, in studying 124 cases of acute peritonitis, found that every one of them developed the disease as a secondary lesion.

It may be true that exposure to cold and severe strain are productive of peritonitis, but if this is the case it is only because these influences diminish the vital resistance of the peritoneum.

The two great causes of peritonitis are appendicitis and disease of the Fallopian tubes. In both of these instances it is due to the extension of an inflammatory process, which in turn arises chiefly from the spread of infecting micro-organisms.

The method by which pathogenic micro-organisms are enabled to pass through the walls of an inflamed appendix has already been spoken of in the article on Appendicitis, and it is worthy of note that any cause which seriously interferes with the health of even a small part of the intestinal wall may permit the escape of micro-organisms into the general peritoneal cavity. Of the micro-organisms which commonly produce peritonitis under these circumstances, the *Bacillus coli communis* is, perhaps, the most frequent, but a large number of other micro-organisms are often present, and there is every reason to believe that they are active in the production of the inflammatory process. Next to the *Bacillus coli communis* stands the *Streptococcus* and the *Pneumococcus*, the *Staphylococcus albus*, and the *Bacillus pyocyaneus*. The *Bacillus aërogenes capsulatus* is also not infrequently present. Occasionally the *Bacillus typhosus* seems to be responsible for the process.

When infection takes place by means of the Fallopian tubes, the peritonitis may be due to the gonococcus; but in the majority of instances the inflammatory process is not due to this organism, but to the streptococci or staphylococci which are associated with it; the presence of which, perhaps, enables the gonococcus to become pathogenic in this serous membrane. Bumm, however, believes that the escape of the gonococcus into the peritoneum is not usually followed by evil results. On the other hand, pure cultures of the gonococcus have been obtained from the abdominal cavity in two cases of acute general peritonitis by Young and Cushing.

Subacute or chronic peritonitis is often due to the *Bacillus tuberculosis* and acute miliary tuberculosis of the peritoneum, which is usually looked upon as a form of acute peritonitis, is necessarily the result of the infection by the tubercle bacillus. In the acute peritonitis following labor, the so-called septic peritonitis, the streptococcus is the chief factor. Cases of peritonitis due to the pneumococcus have been frequently recorded.

While we know, therefore, that peritonitis in its acute forms is a secondary infection, it must not be forgotten that in a very large number of cases the peritoneum is capable of resisting infection and of destroying micro-organisms which may gain access to it. Indeed, the vital resistance of this membrane when in health is very remarkable, and a number of investigators have shown that it is possible to place in the peritoneal cavity considerable quantities of septic material without serious result, provided this serous membrane is not subjected at the same time to insult whereby its vitality is decreased.

Certain diseases which greatly decrease vital resistance greatly increase the susceptibility to peritonitis, as, for example, typhoid fever, Bright's disease, and advanced arteriosclerosis.

Peritonitis in children, of course, develops as a result of the causes already enumerated. It also is sometimes seen in young infants suffering from congenital syphilis, and in those who have intestinal obstruction. In still other cases it follows infection of the umbilicus after birth. In still others it is due to an extension of infection in empyema, and a few cases are on record in which sewer-gas poisoning has seemed to produce an epidemic of this character among children exposed to its influence.

Strümpell states that a form of localized peritonitis in the left groin is occasionally met with in children, that it is prone to be purulent, and that the pus usually escapes through the rectum.

Pathology and Morbid Anatomy.—The characteristic appearance of the peritoneum in primary acute peritonitis is hyperæmia, with a diminution in the normal glossiness of the membrane involved. This is followed by a more or less copious fibrinous exudate, which may be well distributed, or appear chiefly in patches, upon the parietal and visceral peritoneum. In many cases there is little fluid exudate, the small quantities present being found in pockets formed by the coils of intestine which become agglutinated. In other instances the fluid portion of the exudation is very much more copious, and the quantity of fibrin thrown out is also of considerable amount, so that it is not only found well distributed over the surface of the membrane, but free flakes may be found floating in the serous exudate as well.

When the infection is due to pyogenic micro-organisms, and particularly in those cases in which the vital resistance of the patient is very low, a septic peritonitis speedily develops. It is usually very diffuse in such cases, the entire peritoneum being involved. The quantity of exudate is moderately large, and is often offensive in character, forming what has been called "putrid peritonitis." In other cases when the vital resistance is not so depressed, the presence of pyogenic micro-organisms produces a peritonitis in which pus alone is present. This form of peritonitis may be widespread, but is often localized—the so-called loculated or circumscribed peritonitis or peritoneal abscess—nature being able to wall off the area of acute infection by a plastic exudate, which prevents the infection from becoming well distributed throughout the peritoneum. In those cases of septic peritonitis in which death occurs early, the physician may be surprised on opening the abdomen at autopsy to find that but little change has taken place in the appearance of the peritoneum and its contents. Save for some duskiness of the peritoneum and the presence of sanious fluid, the abdominal contents may seem to the naked eye to be but little altered.

Occasionally we meet with what is known as hemorrhagic peritonitis, which may follow severe septic infection, and in cancerous and tuberculous cases, with ulceration, the fluid in the abdominal cavity may be blood-stained.

Localized peritonitis, such as has already been referred to, is most frequently found in connection with diseases of the pelvic organs in women and with cases of appendicitis. It may be considered the rule rather than the exception, for the disease to be limited by an inflammatory exudate in such cases. Other forms of localized peritonitis which are not so frequently met with depend upon an extension of infection from the gall-bladder, from perforation or infection through a gastric ulcer, and occasionally we find a suppurative peritonitis in the lesser peritoneum as the result of disease of the pancreas or fat-necrosis. In other instances this condition arises as the result of renal calculus and nephritic abscess.

Symptoms.—There are few diseases which, when well developed, produce a train of symptoms more characteristic than are those of acute peritonitis. This holds true, however, only when the disease is well advanced, and, indeed, is so severe that there is grave doubt as to the patient's recovery. In most cases of peritonitis, when the physician is first called to the patient, *severe pain* in the abdomen is the chief condition which is complained of. The pulse is usually quick, small, and hard, and the belly wall tender on palpation, and distinctly rigid. The face will be found to wear an *expression of anxiety*, which seems to be far out of proportion to the length of the illness and its severity. In many instances, even when the pains are exceedingly severe, the patient considers that he is suffering from acute indigestion, but acute indigestion is often relieved by pressure, and is usually accompanied by tumidity of the abdomen; whereas, peritonitis is characterized by great abdominal tenderness and by a flat or scaphoid appearance of the belly wall. The fever is usually not very high. The *pulse* is tense and rapid. It often does not go above 102°, and frequently not over 101°. *Vomiting* is frequently present.

After the pains have been present for a few hours, the *exquisite tenderness of the abdomen* makes the weight of the bed-clothes insupportable, and, in order to obtain some relief for the abdominal tension, the patient usually lies on his back, with the knees drawn up, and supports the bed-clothes over his abdomen by his hands, looking with dread upon the approach of the attendant lest he touch the abdomen or jar the bed. *Thirst* which cannot be relieved, because of constant retching, may add to the patient's distress, and *hiccough* of a very persistent and exhausting character often develops.

As the disease progresses, the belly, which has been tense and scaphoid, becomes hard, not from muscular spasm, but from abdominal distention. In the flanks percussion may reveal some flatness due to the accumulation of the exudate in these parts. The *face* not only is anxious in appearance, but rapidly becomes *pinched* and *peaked*, the *eyes* appear *sunken*, the *nostrils* are *thin* and *drawn*, the *skin pale* and *livid*, and the *tongue dry* and *parched*, the typical "Hippocratic facies." The *pulse* at this stage is *exceedingly rapid, running*, and *wiry*, and, as the end approaches, loses its tense character. A *cold sweat* may break out about the wrists and on the forehead.

The *bowels* are usually *obstinately confined*, but in some instances diarrhœa may be present, particularly if diarrhœa has been a symptom of the case prior to the development of the peritonitis. The *respirations* are usually a little *quickened*, but are *shallow* and *superficial*, in order that the abdominal movement may be as slight as possible. A remarkable fact in connection with these cases is the preservation not only of consciousness, but the development of *intense mental activity*, which in some cases persists up to the moment of death, the patient showing an acuteness of mind which is startling. In some instances during the last hours there may be a mild delirium, or even slight stupor.

In *septic cases pain is absent* in the majority of instances, but the temperature in the early stages may be much more febrile than in the ordinary types of the disease. Sometimes it is distinctly like that of early septicæmia. By the time that the septic inflammation is well developed, however, the fever usually falls to the neighborhood of normal, and it may reach subnormal.

Careful examination of the abdomen in cases of well-developed peritonitis not only reveals the local symptoms already described, but it may also show localized patches of tympany where gas has accumulated in the coils of intestine, which are more or less fixed in one position by inflammatory adhesions. These coils, partly because of the inflammation and partly because of distention, may soon become paralyzed, so that distention increases.

When the peritonitis is due to a perforation of the intestine or of the stomach, the accumulation of gas in the peritoneal cavity may mask the area of liver dulness or completely obliterate it. At one time this was considered a very valuable sign in the diagnosis of perforation with secondary peritonitis, but we now know that in many instances this symptom is absent.

Complications and Sequelæ.—Peritonitis usually runs such a rapid course, either to recovery or death, that complications are rarely met. The most important and most frequent complication of a serious nature is pneumonia. In 100 cases of peritonitis observed in the London Hospital, Treves found that no less than 17 developed pneumonia or pleurisy after the peritonitis began. Retention of urine is frequent.

As a sequel intestinal obstruction may develop as the result of adhesions or by strangulation of the bowel, produced by the slipping of a knuckle of intestine through an opening under a band or a false ligament, or by the development of a twist of the bowel through interference with its peristaltic movement.

Diagnosis.—The diagnosis of acute diffuse peritonitis is usually readily made, even in its early stages. In its late stages its symptoms, except in the septic form, are so characteristic that the diagnosis can be made on a most superficial examination of the patient.

In certain cases of typhoid fever in the early stages, when the inflammatory process in the intestines is acute, there may be a good deal of abdominal pain and considerable tenderness. The apathetic expression of the face, the higher temperature of typhoid fever in the stage of onset, the tumid belly, and the coated tongue, with red edges, will aid in its differentiation.

The separation of intestinal obstruction from peritonitis is exceedingly difficult; but as they often are coincident, the one following the other, and the treatment of such cases the same, differentiation is unnecessary. The rapid onset of severe pain of a cramp-like character, the complete absence of any movement of the bowels, the presence of intestinal unrest, and in intussusception the palpation of a mass may make the diagnosis possible.

Certain cases of hysteria at times present symptoms so characteristic of peritonitis that even the most skilful may be misled. Every symptom may be presented, yet the patient always recovers.

Acute hemorrhagic pancreatitis may also so closely resemble peritonitis that a diagnosis is impossible, but this malady is exceedingly rare. In it there may be a preceding history of gallstone disease, whereas in peritonitis, unless perforation of the gall-bladder has occurred, there is no such history.

The pain of gallstone colic and renal colic is so localized that much difficulty in diagnosis does not exist, as a rule.

In cases of perforation of the stomach with secondary pyopneumothorax subphrenicus, the differentiation may be exceedingly difficult, save that swelling in the epigastrium and a history of gastric ulcer may be present.

When perforation of the stomach has occurred without the formation of abscess, so that the gastric contents and gas escape into the peritoneum, great tympany and modification of the area of liver dulness is found. Not infrequently in subphrenic abscess a pleural effusion exists, so that serum may be drawn from this level and pus from the level below the diaphragm.

Occasionally, in children suffering from pleurisy, pericarditis, and pneumonia, violent pain is complained of in the abdomen, which may mislead the physician, if he be not on his guard.

Prognosis.—The prognosis in every case of well-developed acute diffuse peritonitis is distinctly unfavorable. If the physician has reason to believe that the peritonitis is localized, the outlook becomes more promising. A good deal depends, too, upon the cause of the peritonitis, and upon the character of the infecting micro-organism. Thus, if it follows perforation of the stomach or bowels, and it is not walled off from the general peritoneal cavity, and again, if a skilful surgeon is not at hand to operate at once, the prognosis is hopeless. When the infection after perforation is localized, the mortality is not so great, but this form of localized peritonitis is far more fatal than that form which is due to appendicitis.

The duration of life in fatal cases of peritonitis varies very greatly. Death may come as early as thirty-six or forty-eight hours, or may be deferred for a week or ten days, or even longer.

Treatment.—The treatment depends so entirely upon the cause of the peritonitis that it behooves the physician to study the case most carefully. If seen shortly after the onset of the malady, the physician should at once consider the possibility of perforation of some portion of the alimentary canal being responsible, and should examine carefully into the history of the patient as to the possible presence of gastric ulcer or intestinal ulcer due to typhoid fever or dysentery. If the patient is an adult, careful consideration of the possibility of extension of inflammation from the gall-bladder should be followed. It is hardly necessary to state that as appendicitis and diseases of the Fallopian tubes are the most common causes of peritonitis, the condition of these two parts should be most carefully inquired into, both as to previous history of the patient and as to the physical signs which may be present. If the peritonitis is diffuse and has followed perforation or strangulation, the salvation of the patient depends upon immediate surgical interference, unless, perchance, shock prohibits operation, when it is permissible to wait two or three hours, in the hope that by the use of external heat and stimulants the patient may be enabled to bear operation. If, on opening the belly, a diffuse general peritonitis is found, it would seem best, in the majority of cases, to resort to the plan suggested by J. B. Murphy, namely, to remove the appendix if it can be reached, close the perforation if it exists, introduce a drainage tube into the pelvis, place the patient in a semi-recumbent posture and give a quart of normal saline solution by the rectum every two or three hours.

When it is due to appendicitis, the age and general physical condition of the patient, must largely influence the decision as to operative interference. I agree with McCosh, who states that in aged persons, particularly if they have been dissipated, medical treatment gives better chances than surgical interference, while the reverse holds true in young persons. If a surgeon of experience cannot be obtained, medical treatment will always give the best results.

The question as to the procedure which should be followed if the cause lies in the appendix has already been discussed in the article on that disease. Fulminating, gangrenous, or perforative appendicitis should, of course, be operated upon at once; whereas, on the other hand, if the appendicitis has simply produced an adjacent peritonitis, temporizing measures should be resorted to, and operation performed in the period of quiescence.

The profession has passed through three periods of fashion in regard to the drug treatment of peritonitis itself. Twenty or thirty years ago it was extensively taught that general peritonitis should be treated by the administration of massive doses of opium, which were not only sufficient to relieve pain completely, but also to produce mental quiet. Under the leadership of Alonzo Clark, of New York, enormous doses were sometimes given, as much as 258 grains of opium being given in a day; and while it is undoubtedly a fact that patients with peritonitis are able to take these doses without being poisoned, this plan of treatment received its deathblow with the discovery that nearly all cases of peritonitis are due to an infection, and that the source of infection must be discovered, and, if possible, removed. It has therefore become obsolete because it masks the symptoms, and is thought to have no definite influence upon the progress of the disease, save that it diminishes the suffering of the patient. It is probably safe practice to administer a sufficient quantity of morphine or opium to diminish agony, but not enough to mask the symptoms or make the patient so comfortable that he will refuse operative interference when the physician thinks it advisable.

Soon after the infectious nature of peritonitis was recognized, the profession went to the extreme of purging with saline cathartics, and even with vegetable cathartics, all cases in which symptoms of acute peritonitis were manifest. There is no doubt that this method was used to excess, and at the present time we know that it is unnecessary, unless there is reason to believe that the bowels are overloaded with fecal matter. In the writer's experience cases of this character are usually relieved by the bowels expelling the feces in the early stages of the peritoneal inflammation.

The opium treatment was excessive, the purgative treatment was also excessive, when either of these plans was applied to every case, but both of them used in moderation may be advantageous in certain cases.

Counterirritation applied over the abdomen in the shape of a large number of leeches may be useful in sthenic cases. In other instances a light mustard plaster may be used for relief. In still others an ice-bag has been employed. Thirst may be relieved by the use of small pieces of ice, or, better still, by rinsing the mouth with glycerin 1 part and water 3 parts, to which has been added a few drops of lemon-juice. Liquids should not be swallowed, as they increase the tendency to vomiting. If thirst is excessive, fluid may be supplied to the tissues by hypodermoclysis or by rectal injection. As a rule, the patient does not live long enough in well-developed peritonitis to make the question of feeding an important one. If the focus of infection is removed by operation, the feeding is that used after all abdominal sections.

CHRONIC PERITONITIS.

Chronic peritonitis occurs in four forms, namely: a *local adhesive process;* a *diffuse process; one characterized by a proliferation of inflammatory material and connective tissue;* and in a *hemorrhagic form* as a complication of severe disease in adjacent organs or malignant disease of the serosa.

The *local adhesive type* is often found in the neighborhood of such organs as the liver, spleen, and stomach, when, as a result of an acute inflammatory process in the visceral peritoneum covering the organ, an adhesion takes place, and, perhaps, thick fibrous bands develop. In many of these cases this condition is not even suspected during life. In the neighborhood of the pelvic organs this type of peritonitis is exceedingly common, and is, perhaps, the most frequent peritoneal lesion met with by the gynecologist. Sometimes intestinal obstruction results from a slow, chronic, inflammatory process, which glues a knuckle of intestine to the omentum or the anterior wall of the pertitoneum.

In the *diffuse but chronic type* of peritonitis a condition closely resembling that of fibrous tuberculous peritonitis develops, so that the peritoneal cavity is practically obliterated, and the coils of intestine are often matted together so that it is impossible to separate them. The parietal layer of the peritoneum is greatly thickened, and all the abdominal organs seem to be constricted and drawn by the cicatricial process.

Closely allied to the last type is the *proliferative form*, in which the changes are not very different, except that there is, in addition, a considerable quantity of serum in the abdominal cavity. Sometimes this may be present in such quantities that the belly is greatly distended. The omentum is rolled up as a window-shade is rolled up, and extends across the upper zone of the abdomen in a round mass. The intestines may, or may not, be adherent to one another, the presence of the fluid serving to separate them and to prevent dense adhesions taking place. At times some of the fluid may be divided off into pockets by the adhesions. This form of proliferative peritonitis is usually due to tuberculosis, and not infrequently complicates alcoholic cirrhosis of the liver, but it is generally believed that in some cases it may arise from other causes than tuberculosis. Even if the cause is not tuberculosis the condition may, however, very closely resemble it on palpation, because nodules may be found.

In cases of carcinoma of the viscera a chronic form of peritonitis associated with the exudation of blood-stained or hemorrhagic serum is occasionally met with. Indeed, the obtaining of such serum from a case of ascites is always to be considered as indicative of that form of peritonitis depending upon malignant growth. As a rule, the chief lesions are found in the pelvis, or there may be present a general carcinomatosis of the peritoneum. In other cases the pelvic viscera may be coated with fibrinous exudate, which undergoes connective-tissue changes, and becomes highly vascularized.

Friedreich has described a form of chronic hemorrhagic peritonitis which follows repeated resort to *paracentesis abdominis*, the entire peritoneal surface being granular, reddened, and dotted with extravasations of blood.

CHRONIC ADHESIVE SCLEROTIC PERITONITIS.

This is a very rare state, apparently met with more frequently in Germany than in the United States. It was described by Virchow in 1853, and in more recent times by Riedel. It consists in an extensive subperitoneal fibroid infiltration or sclerosis, without ascites and without serous, or serofibrinous, or purulent fluid in the abdomen. In other words, it is rather a disease primarily involving the subperitoneal connective tissue than a true chronic peritonitis. This hyperplasia results in a sclerotic process, which in turn produces contractions and retractions, and, by the formation of adhesions, fastens organs to the abdominal wall. The symptoms are those of the chronic fibroid type of tuberculous peritonitis already described, but the condition is not due to tuberculosis. Wetherill states that the peritoneum shrinks so that when abdominal section is performed it is impossible to approximate its edges on closing the wound.

For a description of the so-called "iced liver of Pick" see Adhesive Pericarditis.

CANCER OF THE PERITONEUM.

This is an exceedingly rare condition as a primary lesion. In all probability when it does occur, it is an endothelioma rather than an epithelioma.

Carcinoma and carcinomatosis of the peritoneum are usually if not always secondary to cancer of some contained viscus. The primary growth may be so small as to escape superficial examination even at autopsy, and is commonly in the stomach, pancreas, liver, or biliary passages, or, less frequently, in the rectum; in the female the pelvic organs are by far the commonest site of the primary growth. On account of the pervious nature of the diaphragmatic lymphatics, the pleuræ, pericardium, and peritoneum may be simultaneously affected, or invasion of one may be quickly followed by extension to the others.

An interesting case of colloid cancer has been reported by Ferguson. Both the visceral and parietal layers of the peritoneum were involved, as were also the omentum. The mesentery was in some places 10 cm. thick.

The *symptoms* of either the primary or secondary carcinoma of the peritoneum are *emaciation, ascites,* and, it may be, the discovery of *nodules,* or of a furled omentum, such as occurs in certain types of peritoneal tuberculosis. The fact that the *fluid is often hemorrhagic* has already been referred to. In the colloid cases the peritoneum may be filled, not with fluid, but with a jelly-like substance, which is so firm that it will not fluctuate.

OTHER GROWTHS OF THE PERITONEUM.

Hydatid cyst of the peritoneum is occasionally found, although, as a rule, it develops in the abdominal organs rather than in the peritoneum itself. A cyst the size of an orange has been reported by Jones as occurring in the mesocolon. It was successfully removed. Rein has reported a multiple hydatid cyst occurring in the omentum of a woman in the third month of pregnancy. She was operated on and recovered. Other instances have been reported by various clinicians, the largest number of cases collected being those of Monéger, who has reported 32. He tells us that such cysts are nearly always secondary to rupture of cysts in neighboring organs, and there is usually a history of violent pain at the time of rupture. Perhaps the most extraordinary case is that reported by MacDonald, who has reported the case of a man with thirty hydatid cysts of the peritoneum. Other cysts of the mesentery are chylous, dermoid, serous, and sanguineous.

Very rarely sarcoma and cystic adenoma affect the peritoneum.

ASCITES.

Definition.—The term ascites is applied to the accumulation of serous fluid in the abdominal cavity. In some cases the quantity of fluid is very small, but in others it amounts to several gallons. In the majority of instances ascites is due to atrophic cirrhosis of the liver, tuberculosis of the peritoneum, or cardiac disease. In cases of chronic Bright's disease of the parenchymatous type, ascites is often present as part of the general anasarca. It also develops as the result of pressure upon the abdominal bloodvessels, whereby the blood in the large venous trunks is obstructed in its flow.

The fluid in ascites is usually of a light straw color, and does not coagulate when exposed to the air.

Etiology.—The intra-abdominal causes of ascites, as just stated, are atrophic cirrhosis of the liver, tuberculous peritonitis, and morbid growths, which, by pressure upon bloodvessels, or by producing changes in the peritoneum, result in a transudation of fluid. A thrombophlebitis, or other form of venous obstruction, may also cause ascites. Thrombosis, tuberculosis, or neoplastic invasion of the thoracic duct, or its obstruction by parasites (filariæ), or other causes, and also wounds of the duct or of the receptaculum, or larger lymph-vessels or chyle-vessels may produce an ascites the fluid of which contains chyle.

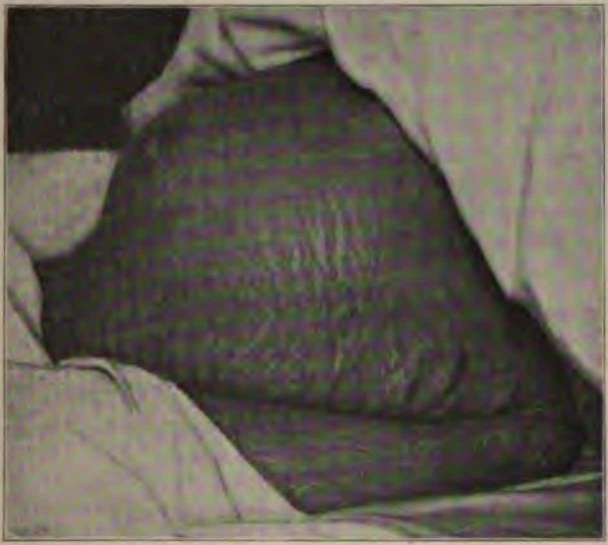
Symptoms.—When the abdomen of a patient suffering from ascites is exposed, it is seen to be greatly enlarged, this enlargement being chiefly in the lower and lateral zones, although if the intestines are by chance distended by gas the upper and middle zone may be most enlarged. The line of the ribs is usually sharply defined, by reason of the fact that they do not yield readily to the pressure and are held in place by the diaphragm.

If the ascites be due to hepatic cirrhosis, the venules about the navel will often be found engorged (see Cirrhosis of the Liver), and in all forms of ascites due to venous obstruction the veins under the skin in the right and left hypogastrium and groins may be surcharged with blood in an endeavor to establish a collateral circulation, and so relieve deep pressure in the venous trunks.

The signs of fluid in the abdominal cavity are dulness on percussion in the flanks, and in the suprapubic region when the patient is semi-recumbent, with tympany over the anterior and middle zone of the abdomen extending upward to the epigastrium, owing to the intestines being floated up against the anterior abdominal wall by the fluid beneath. If the hand of the nurse is placed with its ulnar edge upon the middle line of the abdomen, the left hand of the physician placed on the right flank, and the right hand used to lightly strike the left flank, distinct fluctuations will be felt by the left hand, the impulse being transmitted by the fluid from one side to the other, the hand of the nurse being used to prevent the transmission of this impulse through the abdominal wall. Changing the patient's position from the recumbent to the erect posture will change the area of dulness on percussion and the shape of the abdomen, owing to the alteration in the position of the fluid. Palpation will reveal fluctuation if the belly is not too tense. Percussion will give a tympanitic note in the epigastrium when the patient is sitting up and flatness below the navel and at the sides of the abdomen.

A patient who has ascites to any considerable degree is usually *unable to lie down* with the head low, because if this attitude is assumed the pressure of the fluid against the diaphragm is such that breathing is interfered with. For this reason he usually sits propped up in bed or in a reclining chair. The face, which is usually thinner than in health and somewhat haggard, forms a striking contrast to the large abdomen, which is "aldermanic" in appearance, and if the legs be dropsical as well, the massiveness of the lower half of the trunk, as compared to the upper half and to the neck and face, presents a striking picture. Not rarely the face bears the expression known as the "abdominal facies."

Fig. 86

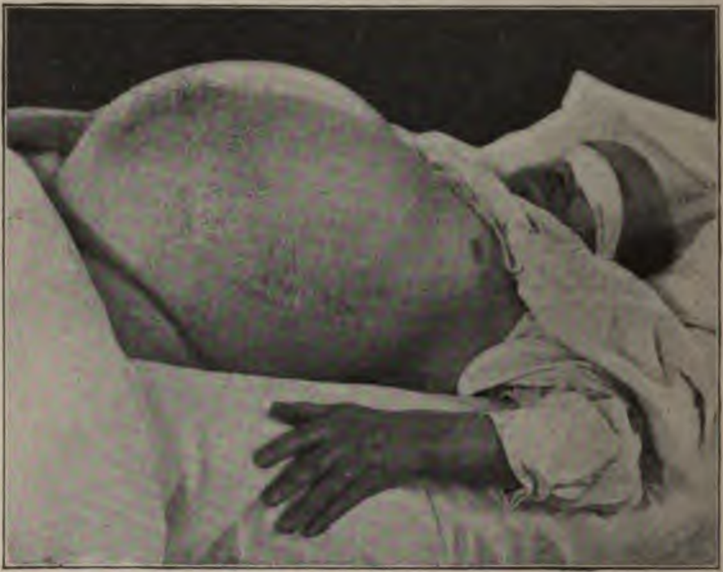


Ascites due to cardiac dropsy, with diastasis of the recti muscles, so that there is a subcutaneous hernia in the tissue around the umbilicus. There is also enteroptosis due to great relaxation of the abdominal wall, which actually overlies the thighs.

Dyspnœa may not be noticeable when the patient is at absolute rest, but it not rarely happens that so slight an exertion as conversation will develop this symptom, particularly if, in addition, there be some tendency to œdema at the bases of the lungs. As a rule, men are more uncomfortable when suffering from ascites than are women, because their respiration is naturally more diaphragmatic than that of women, whose respiratory movement is chiefly costal.

Diagnosis.—Ascites must be differentiated from distention of the abdomen due to a large ovarian cyst. This can usually be accomplished by palpation percussion, and vaginal examination. Inspection of a case of a cyst will usually reveal somewhat greater distention of one side of the abdomen than the other. The area of dulness on percussion will not be in the lower zone of the abdomen alone, but will extend upward toward the ribs and will include part of the area in the anterior and middle zone of the belly, which in ordinary ascites is tympanitic. Further than this, a large ovarian cyst of this character will usually be tense and will offer more resistance when the abdomen is palpated with both hands.

FIG. 87



Case of enormous ascites due to atrophic hepatic cirrhosis.

From enlargement of the spleen in chronic leukæmia ascites may be differentiated, by reason of the fact that in this disease the area of dulness is chiefly in the upper zone instead of in the lower zone of the belly, that tympany is usually not present in the middle line if the spleen extends so far, and that the edge of the spleen can be readily palpated. In some cases, however, in which the spleen is enlarged in leukæmia, ascites is also present, and it may be impossible to feel the edge of the spleen until some of the fluid is removed. This removal may be more difficult than in an ordinary case of ascites, because the spleen may be so close to the anterior abdominal wall and may extend so far down toward the pubis that ordinary paracentesis cannot be readily performed without danger of puncturing the spleen. Ascites must also be separated from great enlargement of the liver, as in hypertrophic cirrhosis. Here, again, the presence of dulness in the right upper zone of the abdomen and the ability to feel the lower edge of the large liver will aid materially in the differentiation. In both enlargement of the spleen and enlargement of the liver the area of dulness and of tympany is not materially altered by changing the posture of the patient as it is in ascites.

Treatment.—The treatment of ascites depends to some extent upon its cause. If it is due to interference with the circulation by pressure, as in atrophic hepatic cirrhosis, little can be done except to remove the fluid by paracentesis, for the purpose of giving the patient relief from distention. If it is due to cardiac disease an improvement in the condition of the heart by the use of digitalis and rest, and the judicious administration of saline purges, may remove the fluid. In renal disease the use of purgatives may also be of value, but paracentesis has usually to be resorted to if the fluid is present in large amount. In peritoneal tuberculosis paracentesis may be of value, but the best method of producing cure is to resort to abdominal section, permitting the fluid to escape through the incision, and then maintaining drainage.

Before performing *paracentesis abdominis* the patient should be made to evacuate his bladder, in order that by no possibility can it be punctured by the trocar. If the patient be a woman, great care should be taken that an ovarian cyst is not punctured. Puncture of a papillomatous cyst not infrequently results in the speedy death of the patient.

DISEASES OF THE LIVER.

INFLAMMATION OF THE LIVER.

Acute Hepatitis or Hepatic Abscess. **Definition.**—Acute exudative hepatitis is a state of inflammation of the liver in which, after a stage of hyperæmia with exudation, the area involved undergoes necrosis, and abscess results.

Etiology.—Inflammation of the liver, severe enough to result in suppuration, may arise from injury, from inflammation of the portal vein or of the bile-ducts, or from adhesions to neighboring organs which are infected and from which infection may spread, as, for example, in cases of gastric ulcer.

Suppuration within the substance of the liver beneath its capsule or in the bile passages occurs under many varying conditions.

Traumatic Abscess.—Liver abscess may result from traumatism. The traumatism may be a severe blow or contusion, or a penetrating wound in or near the liver from a bullet, knife, or other weapon. Traumatic abscess of the liver is usually single. When it occurs as a result of contusion in the absence of direct infection, the injury acts by lessening resistance and permitting colonization of pyogenic bacteria brought to the organ by the portal vein or hepatic artery.

Pyæmic Abscesses.—Pyæmic abscesses are, as a rule, multiple. One group of cases arises from pyogenic embolism of the portal vein. There is phlebitis or thrombophlebitis of the portal trunk or its branches, the infection being due to ulcerations in the colon and rectum, or to appendicitis, ulcerations and suppurative processes about the neck of the bladder, and typhoid fever. Another group of cases arises from embolism of the hepatic artery, as in ulcerative endocarditis and other pyæmic conditions. Infection may also reach the liver through the lymphatics. Abscess may also arise from the direct extension of infection from the gall-bladder and the biliary ducts. Ascarides, liver flukes, echinococcus, and the *Balantidium coli* may also cause abscess of the liver, and it has also been observed as a sequel to measles, epidemic influenza, and ulcer of the stomach.

Amœbic Abscess of the Liver.—In the consideration of the etiology of tropical abscess, we find predisposing and direct causes. As a predisposing cause, the passive congestion of the liver which exists, to some extent, in a large proportion of colonists in the tropics, must be remembered. Other predisposing factors in the production of tropical abscess are malaria and exposure to cold and wet. Abuse of alcohol is probably an important predisposing cause. In Waring's careful study of the subject of abscess of the liver, he found a clear history of the abuse of alcohol in 65 per cent. of the cases.

The direct *cause* of tropical abscess is the *amœba dysenteriæ* which may or may not have previously excited intestinal lesions. Various observers have found that tropical abscess of the liver has been preceded by dysentery in from 72 to 97 per cent. of all cases. Woodward, in 3680 dysentery autopsies, found liver abscess in 779, or 21 per cent. Boston collected data of 2430 autopsies, with 486 abscesses, or 20 per cent. Legrand, of Alexandria, found that in 109 cases of hepatic abscess which occurred in children 31 were due to dysentery. Hepatic suppuration may develop very shortly after the dysentery, or may be delayed for years. (See Dysentery.)

Pathology and Morbid Anatomy.—Abscess of the liver usually occurs either in one or two large purulent collections or in a number of small abscess cavities.

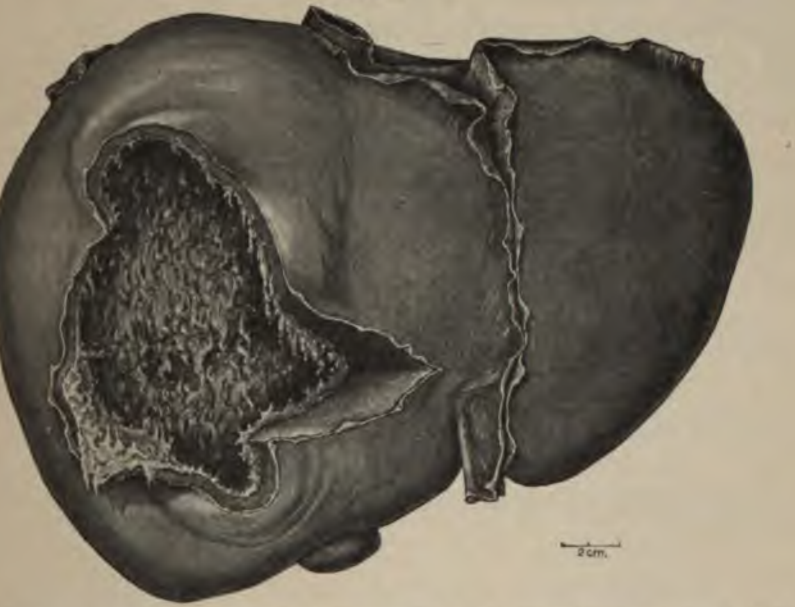
The *single large abscess* is usually the result of dysentery, and the infection reaches the liver through the veins, which closely anastomose with the hemorrhoidal plexus. If the cause be dysentery of the amœbic type, the amœba is found in the wall of the abscess, and less constantly in the abscess contents. In still other cases an examination of the pus reveals the presence of the *Bacillus coli communis*, or the *Streptococcus pyogenes* or a *pyogenic staphylococcus*. In still other cases, if the abscess be very chronic, the pus may be sterile.

In tropical abscesses the lesion is solitary in about 60 per cent of the cases; it is single, from coalescence, or double in about 15 per cent. of the cases, and the remainder are multiple. The abscesses vary in size from a pigeon's egg to a cocoanut.

The single abscess may be very large, and often fills an entire lobe of the liver. The right lobe is usually affected, and as the abscess gradually nears the surface of the organ it bursts through the capsule into the peritoneal cavity, or, as is far more common, the advancing inflammatory zone causes the surface of the liver to become adherent to adjacent structures, so that when rupture takes place the pus breaks into the bowel, as in a case recently under my care, or through the diaphragm into the pleura, or even into the lung, so that the pus from the liver escapes by the respiratory tract.

Accumulations of pus in burrowing a way for escape often cause extraordinary effects, and cases are on record in which the pus from one of these abscesses has escaped into the pericardium, and even into the pelvis of a kidney. Still other instances have occurred in which the pus has found its way into the great veins of the abdomen or into the gall-bladder. Rupture externally is not common.

FIG. 88



Liver, amœbic abscess of right lobe; case of dysentery. Note the shaggy necrotic wall and that the abscess has approached the superior surface of the organ.

The pus from such an abscess is often very offensive, and it generally differs from ordinary pus in appearance, being thin instead of creamy, reddish instead of yellow, and oftentimes it is quite green from the presence of bile. Sometimes, however, the pus is quite like that commonly found in abscesses.

The lining of the abscess cavity is shaggy, because of the pieces of dead hepatic tissue which hang upon it (Fig. 88). In abscesses of long standing more or less imperfect encapsulations of the pus may occur.

Large multiple abscesses are sometimes met with as the result of suppuration about an echinococcus cyst.

Small multiple abscesses are usually pyæmic—*i. e.*, of metastatic origin. Septic emboli, or micro-organisms, from septic foci elsewhere are carried by the blood into the liver and cause multiple areas of necrosis, and suppuration. The liver, therefore, presents not one large abscess, but a large number well distributed through its tissues. These abscesses vary in size. Several small necrotic cavities, which may hold several drachms of pus, may be present, or a number may coalesce to form one large abscess. Although each abscess seems isolated, it is usually in communication through a branch of the portal vein with others, so that by this vascular pathway the whole gland is riddled with pus. The pus may vary from foul, reddish, or greenish material to the character of what used to be called, in preantiseptic days, "laudable pus."

When the infection takes place along the bile-ducts, as the result of the entrance of micro-organisms, the introduction of which is facilitated by the presence of gallstones, it is often found that the pus is not only distributed widely through the organ, but, in addition, that the gall-bladder is full of pus as well, so that the entire biliary tract is involved in the suppurative process.

FIG. 89

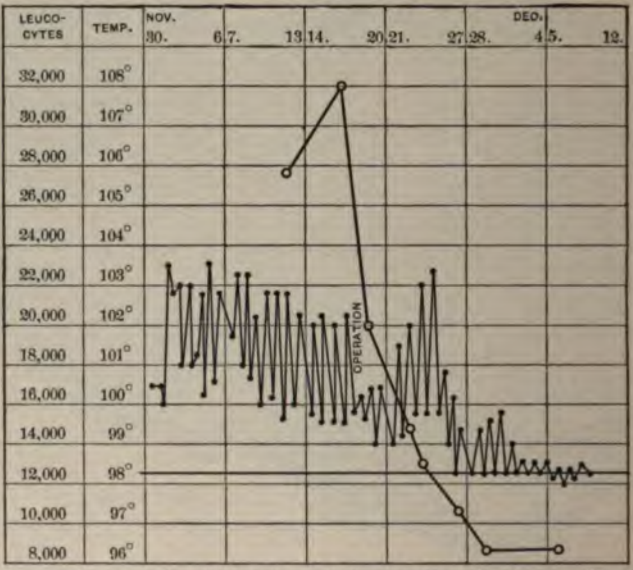


Chart showing septic fever and marked leukocytosis in case of hepatic abscess. (Bassett-Smith.)

Small multiple abscesses may be due not only to the ordinary organisms of suppuration, but to infection by the *Amœba dysenteriæ*.

Symptomatology.—The symptoms of hepatic suppuration are usually marked. In some cases, however, even with the existence of large abscesses, the lesion is latent, and the disease is not suspected until rupture of the abscess occurs. The chief symptoms are *fever, sepsis, enlargement of the liver*, and *pain*.

The pain is felt not only in the right hypochondrium, but in the region of the right shoulder blade, and, as the abscess approaches the surface and causes inflammation of the peritoneum, the pain may be sharp and even severe.

There is *loss of weight* and *strength* and pronounced feebleness in muscular effort. *Dyspeptic symptoms* become marked. There is *anorexia, nausea, morning vomiting,* with a *heavily coated tongue.* The patient becomes *anæmic* and gradually takes on a peculiar subicteric color. The *fever* begins early and is the most constant symptom. At first it does not run high, but later an evening temperature of from 104° to 105° is not uncommon. The fever is irregular or intermitting in type. It is preceded by a chill and then followed by a sweat. These sweats are very severe and contribute greatly to the depression and exhaustion of the patient. They follow the fastigium of the fever and are prone to come on during sleep, whether it be by day or night; so that they may be properly called sleeping sweats rather than night sweats.

Enlargement of the liver is constant. It is symmetrical, and in extreme cases may reach as high as the third rib in front and may extend as far down as the crest of the ilium, or over as far as the umbilicus. The right hypochondrium may appear full and bulging, and there may be an apparent fulness or sleekness of the right side, and in marked cases obliteration of the lower intercostal spaces.

When pus approaches close to the surface it is always preceded by an *œdema of the skin* overlying the abscess, and in cases with large abscess fluctuation may be present. Auscultation over the liver may reveal peritoneal or liver friction.

Occasionally sharp *pain* is felt *in the œsophagus,* when a food bolus passes the level of the diaphragm. A dry, hacking, *unproductive cough* is very commonly present and frequently leads to error by directing attention to the lungs rather than to the liver.

The decubitus of the patient is characteristic. He lies on his right side with that shoulder drawn down and knee drawn up to relieve the tension on the abdominal muscles.

Jaundice is not common and only appears when the enlarged liver or abscesses make pressure on the bile-duct. Pressure on the portal vein may cause a moderate degree of ascites.

Pneumonia of the right base often occurs when the abscess is high up in the dome of the liver.

Diagnosis.—The condition which in all probability most closely resembles hepatic abscess is infection of the gall-duct or gall-bladder, produced by the presence of a stone or stones, for here, too, there is septic absorption, high fever, chills, sweats, and tenderness about the liver, although pus may not be actually present. In this condition, however, there is a history of gallstone colic in most instances, and of jaundice. Further, the emaciation and anæmia are not so marked, nor is the liver so generally increased in size. A marked leukocytosis is present in either case.

The absence of marked swelling of the spleen, of any history of malarial infection, and the lack of the malarial parasite in the blood, combined with the fact that the fever does not yield to quinine, all go to prove the febrile state not malarial. The blood condition may also aid in the diagnosis. There is usually a marked leukocytosis, which ranges from 12,000 to 53,000. Unfortunately, this symptom is not constant, but when it does occur it makes a clear distinction between this disease and malaria. Other conditions that simulate liver abscess are hepatic colic with fever, suppuration in and about the gall-bladder, suppuration in or near the right kidney, subdiaphragmatic abscess, empyema or pneumonia of the right base, and ulcerative endocarditis.

An empyema or pleural effusion on the right side can be excluded by the decrease in vocal resonance and vocal fremitus caused by that state, and by the presence of Skodaic resonance just above the area of dulness on percussion.

It is important to remember that amœbic abscesses may be present without diarrhœa or dysentery, the amœbæ, nevertheless, being present in the stools and in the liver.

In all obscure cases attended by the signs of hepatic disease and sepsis, an effort should be made to establish the condition of the liver by exploratory operation.

Prognosis.—The prognosis of abscess of the liver depends on two factors, the number of abscesses and the time when the case is brought to operation. Eighty to 90 per cent. of single abscess brought to early operation should recover, but often operation is postponed too long. In cases of spontaneous rupture into the colon 50 per cent. recover. The prognosis is not so good where rupture takes place into the lung or pleura. Recovery occasionally takes place when two and three abscesses are present.

In 162 fatal cases of hepatic abscess the mortality is given as due to the following causes: severity of the accompanying dysentery, 125 cases; bursting of abscess into the peritoneum 12 cases, into the pleura 11 cases; gangrene of the abscess wall, 3 cases; rupture of adhesions, 2 cases; pneumonia, 2 cases; and rupture into the pericardium, 1 case. The prognosis in multiple abscess is hopeless, for manifest reasons.

Treatment.—The treatment of hepatic abscess consists in sustaining the patient's strength by good food and by iron and arsenic, and, if the abscess is single, by opening and draining it as soon as its existence is determined.

Exploratory puncture should not be practised unless the surgeon is prepared to go ahead and operate at once. If pus be found, the puncture may spread infection or cause a leak along the wound. This is especially a danger with large needles, and large needles must be used on account of the thickness and viscosity of the abscess contents in some cases.

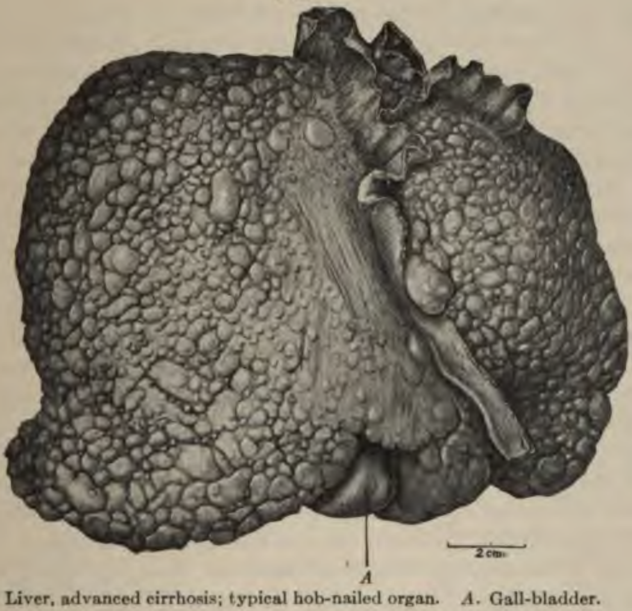
CIRRHOSIS OF THE LIVER.

Definition.—Cirrhosis of the liver is a state in which there is an overgrowth of connective tissue of the gland. In some instances this overgrowth results in an atrophy and shrinkage of the organ (atrophic cirrhosis); in others the liver becomes greatly enlarged (hypertrophic cirrhosis).

Recently Kretz, MacCallum, Kelly, and others have called attention to regenerative changes in the liver cells as being a prominent feature of cirrhosis. They do not regard the normal liver as made up of distinct lobules but as consisting of continuous mantles of cells surrounding the bloodvessels. In cirrhosis, following degenerative changes, these cells regenerate and rearrange themselves and, according to Kretz, cirrhosis is consequently to be regarded as a focal recrudescent chronic atrophy of liver cells modified by parenchymatous regeneration and not as a disease entity. He believes that practical extension of our knowledge on this subject is to come from investigating the causes of degeneration of liver cells rather than from attempts further to differentiate and classify so-called types of developed cirrhosis.

Cirrhosis of the liver derives its name from the Greek word κιρρος, meaning yellow or tawny. The term cirrhosis was first applied by Laennec, because the liver, when cirrhotic, is yellow or tawny in color. Cirrhosis is an unfortunate term, in that it in no way describes the pathological state which is present. Further than this, the word cirrhosis is now applied to pathological states of other organs in which no yellow hue is seen.

FIG. 90



Liver, advanced cirrhosis; typical hob-nailed organ. *A*. Gall-bladder.

Atrophic Cirrhosis.—The liver in cases of atrophic cirrhosis is often enlarged in the early stage of the disease, but after this primary change it undergoes a diminution in size, so that eventually it is much smaller than normal. This primary enlargement, which does not always occur, is perhaps due to hyperæmia, cellular infiltration, and œdema. The characteristic picture of atrophic cirrhosis is, however, that of a small, contracted liver, tawny in hue, and possessing a roughened surface, which in some cases may be so irregular as to be called "hob-nail" liver, because of its resemblance to a rough shoe the sole of which is filled with hob-nails (Fig. 90).

Atrophic cirrhosis of the liver is a not uncommon malady in adults, and it is by far the most frequent of all the types of cirrhosis which affect this organ.

Etiology.—The causes of atrophic cirrhosis of the liver are chronic alcoholism and other chronic intoxications, of which lead is certainly one of the most important. There is good reason to believe that prolonged gastrointestinal indigestion and disorders of nutrition, such as gout and its allied states, may exert a similar effect. Experimental cirrhosis has been produced in animals by acetic, lactic, butyric, and valerianic acid, all of which are present in cases of gastrointestinal disorder. Syphilis may cause it (see Syphilis of the Liver), and hepatic cirrhosis has been known to develop after severe infectious fevers. Cardiac disease, with great and prolonged hepatic congestion, may also produce cirrhotic changes, and it is a noteworthy fact that cirrhosis may be present as a part of a general fibroid process involving the bloodvessels and the kidneys.

Pathology and Morbid Anatomy.—In cirrhosis of the liver the dominant lesion is an increase in its connective tissue. This overgrowth varies very greatly in different cases. Although the fibrous overgrowth may penetrate the lobules, it is principally increased at the periphery of these structures. Again, it may be equally distributed throughout the entire liver or affect certain areas very much more than others, and, finally, the overgrowth of fibrous tissue may be so great that bands, both large and small, may traverse the liver substance, separating it into masses of compressed glandular tissue.

The fibrous tissue formed in this process, like fibrous tissue formed elsewhere in the body, undergoes cicatricial contraction, and by this means fatty degeneration or atrophy of the liver cells composing the lobules is facilitated. These changes are due, not only to the pressure exerted on the lobules, so that their cells are flattened and deformed, but also to the effects produced on the circulation of blood in the liver.

It will be recalled that the hepatic artery carries to the liver the blood which is to nourish its cells, just as the bronchial arteries carry the blood which is to nourish the lungs. The blood from the branches of this vessel in performing its nutritive function passes through the so-called interlobular vessels, and from these into the intralobular vessels, which carry blood from the digestive organs.

The overgrowth of fibrous tissue in the interlobular spaces, in the fibrous sheath of the interlobular veins, and sometimes even between the cells about the intralobular veins results in obstruction to the flow of blood. The arterial supply is little affected, but the venous flow is interfered with, and in this manner the cells suffer, not only from the pressure of the fibrous tissue, but from the pressure in the vessels and the lack of fresh blood as well. Nor is this all, for the fibrous tissue obstructs the smaller bile-ducts and so prevents the escape of bile, with the result that atrophy takes place from retained secretion, and the tissues of the liver become bile-stained.

Many pathologists adhere to the view that the destruction of the parenchyma of the organ takes place first, and that the overgrowth of the connective tissue already described is secondary to this change.

The remote effects of the interference with the circulation of blood in the liver is catarrh of the stomach and duodenum, due to the obstruction of the blood in the portal veins. This state, finally, may cause varicosities in the gastric or œsophageal vessels, and hæmatemesis may ensue, or it may cause fatal hemorrhage from the bowels because of similar varicosities in the intestinal wall. The marked portal obstruction leads to transudation from the peritoneal vessels, constituting the ascites of hepatic cirrhosis.

Not rarely in well-developed cases of hepatic cirrhosis the veins of the abdominal wall will be found enlarged in an endeavor to supplement the deep abdominal veins in the transfer of blood from the portal area to the vessels of the thorax. Still another state, called the "*caput medusæ*," is the development of a bunch of enlarged veins about the umbilicus. This has been generally considered as due to the stasis in the paraumbilical vein or in the umbilical vein, which has not closed, as it usually is after birth. As a result a collateral circulation is established by an anastomosis with the internal mammary, epigastric, and cutaneous veins.

From the description which has just been given of the effect of cicatricial contraction, it is easily seen why the liver presents upon its surface so many excrescences or projections (the so-called "hob-nail liver"), for parts of the gland are pressed out of place, or irregular bands pulled in, by the ever-growing fibrous bands. (See Fig. 90.)

The atrophy of the parenchyma of the liver in the true atrophic form causes a very great diminution in the size of the organ, so that the organ may be less than one-half its natural dimensions.

The spleen is usually enlarged, and arteriosclerotic changes are often present. Secondary fibroid changes in the pancreas have also been described.

In the so-called fatty cirrhosis, in which the deposit of fat is more pronounced than the cicatricial contraction of fibrous tissue, the gland may not be decreased in size, and it may be very much larger than the normal. Such a liver is rarely hob-nailed, but smooth, or but slightly roughened.

Peritoneal and pleural tuberculosis is a very common complication of atrophic cirrhosis of the liver.

Symptoms.—The symptoms of atrophic cirrhosis of the liver are, to a large degree, dependent upon the obstruction to the circulation of blood in the intralobular and interlobular vessels, and if the effects of this obstruction are relieved, or prevented from developing, by the establishment of an efficient collateral circulation, there may be no symptoms at all for many months, or. indeed, for years. Occasionally we meet with cases in which an extreme degree of atrophy of the liver seems to be present with no symptoms of any importance, and yet the patient is taking far more alcohol than is good for him. It is scarcely conceivable that the establishment of a collateral circulation can be responsible for the absence of all systemic disturbance, but there is no other explanation for it.

When obstruction to the flow of blood in the portal vessels is produced *gastric catarrh* develops, and this causes *indigestion* and *distress in the epigastrium*, with *morning nausea* and *vomiting*. Usually the patient *loses strength* and is prone to become *spare and lean* if previously stout.

The occurrence of *hemorrhage from the stomach*, and bowel has already been mentioned when discussing the secondary lesions of the disease, as has also the presence of *enlarged abdominal veins* and the *caput medusæ* on the abdominal wall. (See also the article on Hæmatemesis.) The skin of the trunk is often more sallow and yellow than normal, but it is rarely discolored by a true jaundice. At times the temperature may be subnormal, and at others slightly febrile.

In some cases which have manifested few or none of these symptoms, the patient, with little warning, develops a state of *delirium*, which is often of a noisy and joyous type, but he soon sinks into a state which proceeds to coma, and then to death. To this state the term "*hepatic coma*" has been applied. It was thought at one time to be due to cholesteræmia, but this view has now been cast aside without any satisfactory explanation of the state being offered. I have so often seen this condition follow free drainage of ascites that I believe this operation predisposes to its development.

The physical signs of atrophic cirrhosis consist in an inability to palpate the lower margin of the liver by ordinary effort, and in the small area of hepatic dulness on percussion. In those cases in which ascites is marked, it is often impossible to discover the state of the liver until the fluid is withdrawn.

Some have held that the ascites is really the result of an associated low-grade peritonitis.

Hess has lately directed attention to obliterating endophlebitis of the hepatic veins as producing symptoms almost identical with those of cirrhosis. Most of the 23 cases on record, as also the one he reports, were diagnosed cirrhosis of the liver. The signs leading one to suspect obliteration of the hepatic veins are absence of the history of a cause of cirrhosis, pain over the hepatic area or localized in the upper abdomen, and rapid swelling of the liver and development of ascites.

Prognosis.—The prognosis as to the duration of life depends entirely upon the degree of obstruction to the circulation and upon the severity of the patient's symptoms. If ascites is well developed the outlook for more than a few months of life is bad, yet there are cases on record in which repeated tappings have resulted in the prevention of recurrence of ascites and in the apparent recovery of the patient. Probably in these cases the ascites was not due to the cirrhosis, or the relief of pressure has permitted the establishment of a collateral circulation. The occurrence of hemorrhages is always a most grave omen, yet patients often live for months after severe bleedings. On the other hand, I know of one case in which a man, in apparently perfect health, while washing in his bath-room bled so freely from the stomach as to die in half an hour, although he had never had a hemorrhage before.

Treatment.—The treatment consists in removing the cause of the disease, if it be alcohol, and in an endeavor to prevent intestinal fermentation and disorder by mild purgatives, digestive stimulants, acids, and antiseptics. Each morning the patient should have the bowels well moved by a glass of hot Hunyadi or Carlsbad water, and while this is acting he should receive a few drops of Fowler's solution for the nausea and lack of appetite at breakfast. During breakfast, luncheon, and supper he should take a capsule made up as follows:

℞—Pancreatin,
Taka-diastase,
Sodii bicarbonat. . . . . āā gr. ij.—M.
S.—Take with each meal.

In some cases the catarrhal state of the stomach and bowels is benefited by the use of small doses of iodide of potassium, 5 grains three or four times a day, or of ammonium chloride in the same amount.

Within the last few years it has been proposed by Talma and others that an endeavor be made to relieve the obstructive symptoms of atrophic cirrhosis by establishing a collateral circulation by surgical procedure. The anterior surface of the liver, of the spleen, and of the parietal peritoneum and intestines are roughened by being rubbed with surgical gauze, and the omentum is attached to the parietal peritoneum of the anterior abdominal wall by sutures. By this means it is hoped to cause adhesions through which the collateral circulation may be established. According to Greenough's statistics, only 9 cases have been cured in 105 operated upon. The operation is not devoid of danger because of the malnutrition of the patient.

Ascites should be treated by tapping. (See Ascites.) The use of violent hydragogue cathartics, in the hope that the quantity of fluid will be materially decreased, is not very successful, as a rule, and may weaken the patient. It is, however, important to keep the bowels opened freely to avoid distention and pressure.

Hypertrophic Cirrhosis. Definition.—Hypertrophic cirrhosis is a condition in which the liver is very much enlarged, its surface is smooth, jaundice develops, but ascites is absent. It is called by the French *cirrhose hypertrophique avec ictère,* or hypertrophic cirrhosis with jaundice.

Etiology.—The causes of this malady are unknown. Alcohol is not a factor. In the cases I have seen a history of severe and prolonged malarial infection has been present in several instances. It is a disease of young adult and early middle life, but cases occur in children of tender years.

Pathology and Morbid Anatomy.—An essential difference between atrophic and hypertrophic cirrhosis is that in the former the connective tissue which is developed undergoes contraction, whereas in the hypertrophic form it remains more cellular and does not contract. The second difference lies in the fact that in hypertrophic cirrhosis there is a very considerable increase of connective tissue about the biliary ducts (biliary cirrhosis), with comparatively little or no increase of this tissue about the bloodvessels. For these reasons we do not find any marked decrease in the blood supply of the gland, but we do find jaundice because the biliary ducts are obstructed. In the atrophic form, therefore, venous obstruction causes ascites; in the hypertrophic form biliary obstruction causes jaundice, but there is usually no ascites. Between these two types of cirrhosis intermediate cases develop, in which sufficient overgrowth of connective tissue about the bloodvessels may be present to cause ascites, for in hypertrophic cirrhosis there is an overgrowth of connective tissue about the interlobular vessels, and even between the cells of the lobules.

Although the liver is enlarged, it does not present the hob-nailed appearance of atrophic cirrhosis because contraction does not occur. On cross-section it presents a firm surface of a yellowish-green hue, and strands of connective tissue can be seen traversing it.

It has been shown that in some instances hypertrophic cirrhosis is a secondary condition arising from disease of the bile-ducts, and that in others it is a primary state without connection with any direct cause yet discovered. As a consequence, some writers have divided this disease into two types and have asserted that these are separate entities. The pathological changes in each case are so nearly identical that this separation is not justifiable. To the primary form the term "Hanot's cirrhosis" has been applied. In this form there is pigmentation of the skin in addition to jaundice, there is usually more pain than in the commoner variety, and the spleen is usually very large. It is better to call such cases instances of Hanot's type of hypertrophic cirrhosis.

Symptoms.—The predominant symptom of this disease is *enlargement of the liver*, which often extends to a lower level than that of the navel and far to the left of the middle line. When the liver is palpated it is found to be hard and its *surface fairly smooth*, its edges rounded, and its movement heavy and difficult when it is pressed upon. Percussion reveals the fact that it not only extends downward, but upward far above the ordinary hepatic level, and laterally even to the sixth rib, pushing the diaphragm and lung before it. The *spleen* is usually considerably *enlarged*. *Jaundice* is generally present and varies from a faint lemon-yellow to a dark-olive hue, but it is a noteworthy fact that the stools are not without bile, as in ordinary jaundice. The urine, of course, contains bile. At times attacks of severe *hepatic pain* develop, and the liver may seem more enlarged than usual at these periods. *Pruritus* is often a most persistent symptom, and *xanthoma* may be developed to an extraordinary degree. In a case now under my care the yellow xanthomatous patches about the eyes form a striking contrast to the dark-olive hue of the rest of the face. More or less *disturbance of digestion*, due to gastric catarrh, from an impaired circulation in the stomach, is nearly always present.

Diagnosis.—An enlarged liver, like that seen in hypertrophic cirrhosis, is also seen in leukæmia, but the association of profound anæmia and pallor of the skin separate it from the condition now under consideration. Great enlargement of the liver also occurs in heart disease from hepatic congestion, and in cases of adherent pericardium. The state of the heart makes the diagnosis possible in this instance. Malignant growth of the liver can often be differentiated by the presence of a primary growth in the gall-bladder or stomach, and by the nodules felt in the liver substance.

Prognosis.—The prognosis as to cure is hopeless. As to duration of life, it varies from one to ten years. Death comes from some terminal infection, from a hemorrhage into the bowel or stomach, or from an ever-increasing feebleness. At times coma develops.

Treatment.—There is no treatment for the disease itself. The digestion should be kept in good order by the line of treatment outlined for this function under Atrophic Cirrhosis.

Syphilitic Cirrhosis.—Syphilis may produce, in its inherited form or tertiary stage, a remarkable degree of cirrhotic change in the liver. The overgrowth of connective tissue in this type projects itself everywhere between the lobules and between their cells, or forms large bands which, as they contract, produce extraordinary irregularities in its surface, so that the organ appears to be as well covered with knobs as a tuberous root is covered by projections, a polylobed organ. (See Syphilis.)

Because of the frequency of syphilis, this form of cirrhosis is not very rare. The importance of recognizing it lies in the fact that while we cannot cure the state already developed, by active antisyphilitic treatment we may be able to arrest or delay its progress, and in this sense the prognosis is better than in the non-syphilitic type. In some cases, however, this peculiar nodular formation does not occur, and the course of the disease may be identical with ordinary atrophic cirrhosis. Sometimes tumors due to the formation of gummata can be felt. The symptoms differ in no way from those of ordinary cirrhosis, except when gummata are present.

Treatment.—The treatment of syphilitic cirrhosis, while it presents greater opportunities than are offered by therapeutic measures in ordinary cirrhosis, cannot be expected to produce very remarkable alterations in the liver. Wonderful as are the effects of the iodides and mercury in the treatment of syphilis, they cannot regenerate tissues which have been destroyed, and the most that we can expect from them is that they will do something to arrest the progress of the disease, and, perhaps, cause a removal of some of the cells which have been proliferated, but which have not as yet become organized tissue. In those cases in which there are any evidences of active syphilis, it is hardly necessary to state that the specific treatment should be carried out most thoroughly. (See Syphilis.)

PERIHEPATITIS (CAPSULAR CIRRHOSIS).

Inflammation of the capsule of the liver and of the tissues immediately beneath it, when it occurs in a chronic form, may be associated with chronic peritonitis and with hepatic cirrhosis. More rarely it develops as a result of chronic pleurisy on the right side. The thickening which ensues may, by its contraction, result in deformity of the surface of the liver. This effect is increased by the fact that abnormal projections of connective tissue dip downward from the capsule into the liver substance and divide it into masses of parenchyma, which undergo atrophy from pressure. The condition is, therefore, in some cases, at least, not very different from that met with in ordinary atrophic syphilitic cirrhosis.

The condition is very rare and is thought by Hale White to be due to, or a sequence of, contracted kidney. Unlike most instances of chronic contracted kidney, the patient often has marked ascites because of the state of the liver. As already stated, it sometimes happens that the capsule of the liver is secondarily involved in cases of chronic peritonitis, particularly in cases of adherent pericardium and chronic inflammatory changes in the mediastinum. (See Adherent Pericardium and Mediastinitis.)

AFFECTIONS OF THE HEPATIC BLOODVESSELS.

These consist, aside from those already considered when discussing the subject of cirrhosis, in three chief changes.

Hyperæmia occurs physiologically whenever the liver is actively engaged in disposing of foodstuffs. Pathologically, it probably takes place when an extra amount of stimulating food and irritant drink are taken. Neither condition is capable of being diagnosticated.

A much more important state is the *congestion due to cardiac disease,* or to other causes which retard the egress of blood from the gland. We find, therefore, that all the causes which tend to result in interference with the free flow of blood in the inferior vena cava above the liver may cause hepatic congestion. Disease at the tricuspid orifice of the heart, pulmonary emphysema, fibroid lung, bronchiectasis, and valvular disease of the left side of the heart, with secondary obstruction in the right side, all produce it.

The congestion is, of course, due not to an increase of blood in the portal vein but in the hepatic veins. As a result of this, the centre of each lobule is congested, and its periphery contains relatively less blood. Frequently the cells in the areas of greatest congestion are pigmented. As a result of this, the homogeneous hue of the normal liver is altered, and it presents what is called a "nutmeg" appearance, particularly if the cells at the periphery become still paler from fatty changes. In some cases, however, nothing more than the appearance of intense congestion is present. When the continued pressure by the excess blood causes atrophy of hepatic cells, the condition is known as "red atrophy" of liver.

During life the congested liver is usually much larger than normal, and may attain enormous dimensions, but not rarely, when the congestion has lasted for a long time, it grows smaller than it is in health, and its surface may be roughened, so that it may somewhat resemble the roughened surface seen in early stages of atrophic cirrhosis. In fact the connective tissue is usually increased.

A third vascular change is *thrombosis of the portal vein.* The formation of this thrombus may be due to pressure on the vein produced by a tumor, by gallstones, by traumatism, and in some instances it may arise from septic infection. When the thrombus forms from pressure, and is not infectious, it may become organized and gradually close the vessel permanently. This does not necessarily work much havoc in the liver, because the nutrition of this organ is carried out by the branches of the hepatic artery, which speedily enter into anastomosis with the neighboring vessels. While the liver itself may not suffer very greatly, the abdominal circulation is usually much disturbed by obstruction of the portal vein, and the spleen, the kidneys, and the veins of the entire abdominal network become engorged, so that ascites usually occurs and hæmatemesis or bloody stools may be produced.

If the thrombus is infectious (*suppurative pylephlebitis*), the pathological and clinical picture is quite different, for in this case the clot does not act as a mechanical obstruction alone, but as a source of septic disease. Necrosis and suppuration take place in the wall of the infected vessel and in the hepatic tissues around it. The thrombus may break down and minute pieces of the infected mass pass into smaller divisions of the portal vein, and so spread the disease until multiple abscess of the liver develops.

The sources from which such septic infections arise are found in ulcer of the stomach and bowels, appendicitis and suppuration of the lymph nodes in the mesentery. Another origin is suppurative angiocholitis. (See Abscess of the Liver.) In infants infection may take place by way of the umbilicus.

Symptoms.—The symptoms of congested liver consist in finding its lower margin below the ribs to an abnormal degree and *distinct tenderness* on pressure, particularly in the epigastrium. Pain in this region is often complained of by the patient even when he is at rest. If tricuspid regurgitation is present, the liver may have *systolic expansile pulsation*, which must not be confounded with movement of the liver due to direct transmission of the impulse of the apex beat of the heart. In some cases distinct evidences of gastrointestinal catarrh develop, and the congestion of the gastric vessels may be so great as to cause rupture and *hæmatemesis*. *Ascites* due to the interference with the venous return in the lower parts of the body, and œdema of the legs from the same cause may be present.

Treatment.—The treatment consists in unloading the gastroduodenal and hepatic glands and bloodvessels by a full dose of blue mass (10 grains), followed, if need be, by a saline purge. This is to be followed by the use of digitalis to support the heart, and, if the arterial tension is high, by the administration of nitroglycerin, to lower the arterial pressure and so relieve the heart of work. (See Valvular Disease of the Heart.)

AMYLOID LIVER.

Amyloid disease of the liver occurs as the result of severe and prolonged suppurative changes in other parts of the body, as in chronic bone disease, in pulmonary tuberculosis, and in syphilis, particularly if the latter disease has caused suppuration over a long period of time. The liver is usually very large and may be easily felt far below the ribs, presenting a smooth surface. Occasionally an amyloid liver is small. When it is cut across it is found to be hard and infiltrated by amyloid material, which stains a mahogany hue when it is touched with a watery solution of iodine.

Symptoms.—The symptoms are not typical. Indeed, it may be said that the changes in the liver produce no manifestations that call attention to hepatic disease, for there is no jaundice and no pain, neither is there any ascites. The presence of enlargement of the liver, with a smooth surface of the gland, and the presence of a suppurative focus, combined with an absence of any of the signs of hypertrophic cirrhosis and morbid growth, make the diagnosis easy.

Treatment.—There is no treatment for amyloid liver, except the removal, if possible, of the suppurating area, which is the underlying cause of the disease, and in the maintenance of as great a degree of health as possible by the use of fresh air, sunshine, iron, arsenic, and good food.

FATTY LIVER.

Fatty liver, like fatty heart, occurs in two forms, namely, as a true fatty degeneration of the hepatic cells and as an infiltration of fat into the cells. The true fatty degeneration without cirrhosis is rarely met with, except as a result of the ingestion of some poison, such as phosphorus, antimony, iodoform, sulphate of copper, and carbolic acid. A similar change is present in acute yellow atrophy of the liver.

In uncomplicated fatty infiltration the liver is enlarged and smooth. On autopsy it is pale and yellow and renders the incising knife greasy. Its specific gravity is so low that the organ may almost float in water. No distinctive hepatic symptoms are present.

Often fatty infiltration is part of general obesity, and the large deposit of fat in the abdominal wall and in the tissues near the liver make a diagnosis difficult.

Large, fatty, cirrhotic livers have been recorded, and fatty liver accompanying alcoholism, severe anæmia, and cachexia, as in tuberculosis, also occurs. In such cases the change is thought to be due to faulty oxidation.

TUMORS OF THE LIVER.

The most important morbid growth in the liver is *carcinoma*. It is rarely primary.

Secondary carcinoma is quite common and is nearly always due to metastasis from the alimentary viscera, as from carcinoma of the gall-bladder, of the stomach, of the pancreas, or of the intestines. In rare instances the metastasis is from more distant organs, as the uterus or mammary gland.

Carcinoma of the liver rarely occurs as a solitary nodule. Usually it is in the form of multiple growths, which vary in size from a small seed to an orange. When these growths are immediately subcapsular they appear as large protuberances on the surface of the organ, which may be felt as nodules through the belly wall, or they lie buried in the tissue of the liver and present a disk-like surface upon the level of the capsule, so that they may be felt in thin persons as a slightly flattened or umbilicated elevation. The nodules are sometimes very hard, at other times quite soft, and in the centre softening may take place, so that an apparent cyst is formed (Fig. 91).

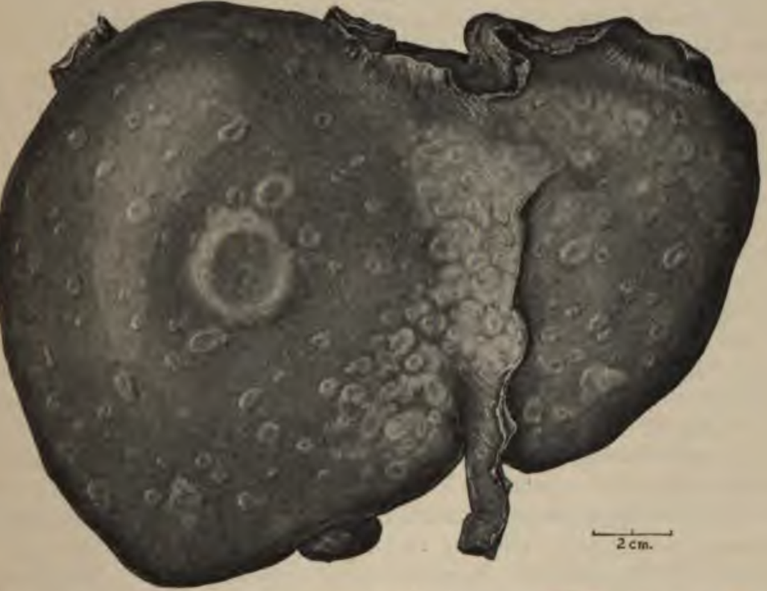
The liver is sometimes enormously enlarged and may extend far below the navel, thereby causing great distention of the belly. A very rare form of hepatic cancer is that in which the liver diminishes in size with the development of the growth.

Sarcoma of the liver is rarely encountered.

Cavernous angiomata have been described, and cysts, single and large, or multiple and small, have been found in this organ. The latter are congenital, the so-called "cystic disease of the liver." (See also Hydatid Disease.)

Symptoms.—The evidences of tumor of the liver may be so easily observed in many cases that there is no difficulty in making a diagnosis, particularly if emaciation, anæmia, and profound cachexia are present. In other cases the fact that the patient is still well nourished, and the growths deeply situated, may render it impossible to discover the existence of a mass by palpation. In some instances there is present nothing more than a vague sense of distress in the hypochondrium, with loss of weight and strength. In still others, if the gall-ducts are obstructed, jaundice may come on, and if the pancreas is involved, fatty stools may be present, or glycosuria is found. Pain may be severe, but usually it is not. At times the veins of the leg on the right side may be obstructed by a thrombus, or a phlebitis may be present. Moderate fever may occur.

FIG. 91



Liver, secondary carcinoma. Even the smaller nodules show more or less umbilication, which is marked in the larger masses.

Diagnosis.—The diagnosis requires that we exclude hypertrophic cirrhosis, which can be done by the more rapid development of the enlargement of the liver in carcinoma, and by the presence of cancerous cachexia. From echinococcus cyst it must be separated, by the fact that the cyst usually fluctuates, or, at least, is not so hard as is nodular cancer. From gumma of the liver other tumors can be differentiated only by the history of the patient and the response to antisyphilitic treatment.

Prognosis.—When the growth is malignant, the outlook is, of course, hopeless as to recovery.

Treatment.—If the growth is a gumma, antisyphilitic treatment is of great value. (See Syphilis.) If it is a cyst or non-malignant, surgical interference is a possible source of relief. If it is malignant, no efficient treatment except the relief of pain can be instituted.

ACUTE YELLOW ATROPHY OF THE LIVER.

Definition.—Acute yellow atrophy of the liver is a condition characterized by marked fatty degeneration of the organ and violent headache and delirium. It is a very rare disease.

Etiology.—The causes of acute yellow atrophy are unknown, but it occurs more frequently in women than in men, and has been associated in many instances with the puerperal state. A micrococcus has been found in the liver in some cases, but the suspected bacterial origin of the disease has not been proved. Some pathologists regard it as a local state representing a general infection, and others as a distinctly local disease, but its toxic nature is generally admitted.

Pathology and Morbid Anatomy.—The changes which take place in the liver are a rapid decrease in its size due to necrosis, fatty degeneration, and cellular fragmentation. So rapid may be the process that after three or four days the organ is not one-half its normal size. The gland is soft and its capsule shrivelled, as if it were too large for the organ. If an incision is made into the viscus the lobules will be found almost destroyed, and the cut surface is mottled, softened, and gray, red, or yellow, according to the stage of the disease. In the gray areas the forms of the hepatic cells are recognizable, and their protoplasm is granular in appearance. In the yellow areas the cells are more or less filled with fatty globules and yellow pigment, and in the red areas the cell outline may be lost, and only cell *debris*, or the remains of broken-down cells, may be found. The spleen is usually enlarged, the kidneys are the seat of parenchymatous degeneration, and the heart muscle is also degenerated. Hemorrhagic extravasations may occur, not only in the liver, but in the stomach, bowels, bladder, and kidneys as well. When death has been long delayed, evidences of universal tissue degeneration may be found.

If the urine is examined, it will be found loaded with bile and an excessive quantity of leucin and tyrosin, or, perhaps, only one of these products. Leucin appears in round disks, and the tyrosin in needle-shaped crystals, which are usually bunched together.

Symptoms.—The symptoms of this malady are *jaundice, severe headache, vomiting*, and finally *delirium, fibrillary muscular tremors, convulsions*, and death. As a rule, there is little or no fever, but cases with high temperature have been recorded. *Petechial spots* and *large hemorrhages* may develop beneath the skin.

Diagnosis.—The development of what might be called fulminating jaundice in a woman during the puerperium should awaken a suspicion of this disease at once, but such an onset does not necessarily prove the presence of acute yellow atrophy, unless there is marked decrease in the size of the liver, and leucin and tyrosin are present in the urine. Even these additional signs are not pathognomonic. Particular care must be taken that the coma of hypertrophic cirrhosis is not taken for acute yellow atrophy of the liver, for sometimes hypertrophic cirrhosis belies its name, in that the liver is not greatly enlarged, and acute yellow atrophy may occur without the liver being greatly decreased in size.

Prognosis.—Death nearly always occurs. A few cases are said to have recovered.

Treatment.—Beyond the use of mild purgatives, diuretics, and stimulants there is no treatment for this malady.

DISEASES OF THE BILIARY TRACT.

1. **Acute Catarrh of the Bile-ducts, or Acute Cholangitis.**—As its name indicates, this is a condition in which the mucous membrane lining the gall-ducts becomes inflamed and swollen, and its secretion thick and tenacious. These two conditions produce partial or complete occlusion of the ducts, which in turn results in biliary stasis and jaundice. In the liver itself marked pigmentation also occurs, because of the accumulation of pigment granules in its cells. When the inflammatory process affects the gall-bladder, it is called catarrhal cholecystitis. Acute catarrh of the bile-ducts is the cause of most attacks of jaundice which last for a few days.

Etiology.—This condition nearly always arises from a primary catarrh of the duodenum, which extends to the common bile-duct. It may follow exposure to cold, particularly if the exposure follows heavy eating and drinking. It usually accompanies the presence of gallstones if they arise in or enter the common or hepatic duct. It also is a result of infection in many cases, as in influenza, or more rarely in pneumonia. Still more rarely jaundice occurs after a severe fright or a paroxysm of intense anger. Any cause which interferes with the circulation in the liver and duodenum may also indirectly produce this state, as, for example, mitral stenosis.

In nearly all cases the inflammation does not extend beyond the lower part of the larger ducts.

Symptoms.—The symptoms vary very greatly. In some persons a well-marked jaundice develops, with such slight general symptoms that the patient does not know he is ill until he sees his reflection in a glass or a friend remarks upon his *yellow hue*. In other cases the patient may feel and seem *wretchedly ill*, probably because he has in the intestine certain substances which, in the absence of bile, develop toxic materials which the torpid liver does not destroy. *Headache* is often marked, and *profound weakness* may be felt. The *stools* are *putty color* because of lack of bile, and the *urine* is the *hue of porter*, because of the presence of this secretion in excess. The color of the skin varies from a faint lemon to a much deeper hue, but the deep olive-green jaundice of chronic hepatic disease is not met with. The *pulse* and *respiration* are *remarkably slow*, owing to the pathological action of the retained biliary salts. The *temperature* may be *subnormal*, but if the condition is due to an acute infection it may reach 102° or more. The liver on palpation and percussion is usually found to be moderately enlarged, about two to three fingers' breadth below the ribs, and it is often tender on pressure.

Catarrhal jaundice usually lasts from a few days to several weeks. After the patient has been ill for some days he usually *loses weight* very rapidly. There is no mild, brief acute illness which causes a greater loss of weight in a few days than this affection.

Diagnosis.—The development of jaundice in persons under forty without any signs of grave disease renders a diagnosis of acute catarrh of the bile-ducts probable. In older persons, particularly if the jaundice develops gradually, the possibility of malignant growth being present must be excluded. In other cases when jaundice comes on suddenly, the cause may be gall-stone, and in such instances a history of colic may be given. If distinct enlargement of the gall-bladder is present, the probability of carcinoma is great. (See Tumors of the Gall-bladder.)

Prognosis.—In cases of acute catarrh of the simple form the prognosis is always good.

Treatment.—In acute catarrh of the bile-ducts the treatment consists in placing the patient in bed, and in the application of hot compresses over the liver, renewing the compresses as rapidly as they become cooled. In some instances it is advantageous to wet the compresses with hot water which contains a drachm of dilute nitromuriatic acid to the pint; or, in other cases, for the purpose of producing counterirritation, a turpentine stupe may be employed.

The kidneys should be kept acting freely by the administration of large quantities of Vichy water, or in other instances Poland water, if Vichy water is not to be had. In some instances, if the kidneys are inactive, it is advantageous to add to the water 5 grains of bicarbonate of potash in each glass.

If the bowels are at all confined, as they are prone to be, they are best moved by one of the saline purgatives, of which sodium phosphate is usually considered most advantageous. This may be given in quantities varying from 20 grains to a drachm in half a glass of hot water every two hours until the bowels are thoroughly moved.

The administration of calomel in the early stages of catarrhal jaundice is not rational. The object of the physician is to re-establish biliary flow, and the difficulty which exists is that the liver is unable to get rid of the bile which it secretes. To stimulate this gland to a greater secretion of bile by calomel, when its ducts are blocked, is manifestly not good therapeutics.

After the acute stages of jaundice have passed by, it is often advantageous to give broken doses of calomel, in order to overcome the natural inactivity of the liver after acute inflammation in its bile-ducts, and these should be followed by the doses of phosphate of sodium, already named.

Patients with jaundice frequently insist upon getting up and going about. This is not a safe thing for them to do, as it is entirely possible for them, if the cause be gallstones, to convert an attack of acute catarrhal jaundice into one of acute cholecystitis.

In regard to diet, the patient had better subsist upon nutritious broths, thickened it may be with barley or rice, partly digested with pancreatin, and well flavored with salt. Milk is usually not well digested by patients suffering from this condition, particularly if it contains any considerable quantity of cream. All fatty articles of food should be avoided, as the emulsification of fats in the intestines in the absence of bile is imperfectly carried out.

2. **Chronic Catarrh of the Bile-ducts.**—This state rarely arises as a sequence of the acute type just described. More commonly it is due to obstruction of the common duct caused by the presence of gallstones, growths, or stricture. Two results ensue from such a state, depending upon the degree of obstruction. When the duct is totally blocked, we find on opening it that it is not filled with bile, but with clear or slightly tinged mucus which is devoid of bile, and that the mucous membrane is not much affected, for it remains smooth. Such patients present marked and *persistent jaundice,* the skin often being olive-green in hue. When the duct is not quite occluded, the retained mucus is bile-stained; it is cloudy, not clear, and it may contain micro-organisms from the bowel. Because of the infection of this mucus from the bowel *marked fever* may be present, characterized by sharp intermittency, and associated with *fever* and *sweats,* just like those met with in the suppurative type about to be described.

Treatment.—Chronic catarrh of the bile-ducts cannot be materially modified by medicinal measures. Prolonged counterirritation in the form of tincture of iodine over the liver may be tried, but it must be continued for long periods of time before any possible influence can be expected from it. If constipation exists, the bowels should be relieved, preferably by saline purgatives, of which the phosphate of sodium, Hunyadi water, or some similar mild purgative, are considered the best.

The diet should be composed of easily digested meats and easily digested starches, the digestion of which should be aided by the use of pancreatin and taka-diastase. Milk and fatty foods are usually not well digested by such patients, and are prone to cause fermentation and distention of the bowels by gas. The best treatment for these patients is operative interference, for the presence of fever indicates infection and hints at the existence of pus.

3. **Suppurative Inflammation of the Bile-ducts.**—When infection of the mucous membrane results from the growth of the *Bacillus coli communis,* or more rarely the *Streptococcus pyogenes* or other pyogenic organisms, suppuration occurs. It is sometimes called "suppurative cholangitis." The solution of continuity in the mucous membrane which permits infection may be due to gallstones. In some cases typhoid fever, cholera, typhus fever, or pyæmia may be the cause.

This form of inflammation extends much farther into the smaller biliary passages than the acute catarrhal form, and by this means the liver may become generally infected, the ducts containing pus and the gall-bladder also being filled with the same material. In some cases the suppurating ducts may cause small abscesses in the liver substance outside their walls, and these again may grow large enough to communicate, and so abscesses of considerable size develop. When the process is severe the ducts may be perforated, and the pus and bile escape into the peritoneal cavity, causing peritonitis, or fistulous tracts may communicate with the bowel or the exterior of the body.

Symptoms.—The *symptoms* are usually so *sharply developed* that there is little difficulty in deciding that pus is present in the liver. The *fever* ranges from high to low, as in *sepsis;* the *liver is enlarged* and *very tender;* the gall-bladder may be palpable, and *jaundice* is also well marked in some cases, but slight in others. The presence of pus in so vascular and important an organ causes *profuse sweats* and *rapid loss of flesh,* but *pain* is usually not severe.

Diagnosis.—Suppurative cholangitis must be differentiated from abscess of the liver, and, more important than all, from severe catarrhal cholangitis without the formation of pus.

In hepatic abscess there will be found, on careful examination of the patient's history, that at some time in the near or remote past there has been an attack of dysentery or of an infection in some part of the body from which pyogenic micro-organisms have been carried to the liver. Very rarely there may be a history of trauma. While such a history may be found in a case of suppurative cholangitis it is much more indicative of abscess. A history of gallstone colic is indicative of cholangitis, and it must be recalled that only moderate attacks of pain in the hepatic or gastric region may be present in cases of gallstones. In other words, every person that has passed a gallstone does not give a history of severe colic. Finally, the presence of jaundice is a sign of cholangitis, for this symptom is usually absent in abscess. In both cases the liver is enlarged and there is a distinct leukocytosis.

From severe catarrhal cholangitis suppurative cholangitis is separated by the facts that in the former state the leukocytosis is not so marked as in the suppurative type, there are no marked chills, sharp fevers, or sweats, nor is there so much tenderness and enlargement of the liver.

Treatment.—There is no medicinal treatment for this condition. The same rule holds good in regard to pus here as for pus elsewhere; whenever it is present, the safety of the patient demands that it should be given an exit, but if the suppurative process is diffuse this, of course, cannot be done.

4. **Occlusion and Constrictions of the Bile-ducts.**—Occlusion of the bile-ducts sometimes takes place by the entrance of an intestinal worm, by the impaction of a gallstone, or by the pressure produced by an aneurysm, a carcinoma, or other growth, such as enlargement of the lymph nodes, enlargements or tumors of the head of the pancreas, inflammation and fibroid changes around the ducts, and twisting or angulation, caused by hepatoptosis or other changes in the visceral relations. If the cystic duct is obstructed the gall-bladder is greatly enlarged, but no jaundice is present, but if the common or biliary ducts are closed, intense jaundice is developed. (See Tumors of the Gall-bladder.)

Congenital occlusion of the ducts sometimes is met with. When it is complete death occurs within a few weeks after birth. Hemorrhages from the navel and other parts of the body are usually present in these cases.

Symptoms.—The symptoms are those met with in chronic catarrh of the bile-ducts and vary as these vary with the degree of obstruction and the degree of infection. Cases occur, however, in which life is prolonged for long periods if the ducts are not completely closed and infection does not take place. A remarkable instance of this character has been reported by Cocking, of Sheffield, in which a woman was jaundiced for fifty years from her third week of life, yet was in perfect health otherwise.

Treatment.—The treatment is surgical.

ACUTE CHOLECYSTITIS.

Definition.—This is a state in which the gall-bladder suffers from an acute inflammatory process, which varies from catarrh of the mucous membrane to suppuration, and even to phlegmonous change in the walls of the viscus. The process may be catarrhal, pseudomembranous, gangrenous, or suppurative. It may be restricted to the lining mucosa and submucosa, or extend to all the coats. When the overlying serosa is affected the process is called pericholecystitis or paracholecystitis.

Etiology.—Cholecystitis arises from the presence of gallstones, which, by injuring the gall-bladder, permit infection to occur, and by the entrance of pathogenic organisms, which, by reason of lowered vitality of the patient, or other causes, are able to produce more or less severe inflammatory changes by their presence. The time at which the micro-organism enters the gall-bladder and that at which it makes its presence felt may be widely separated; for while it is true that bile is antiseptic in its influence under certain conditions, the germ may remain alive but quiescent for months or even for years, and produce its effects only when some illness or other cause offers an opportunity for it to develop. This period of inactivity, so far as inflammatory action is concerned, may be utilized in the formation of gallstones about the nucleus formed by those bacilli which have become agglutinated. (See Cholelithiasis.) The organisms found in the gall-bladder are very numerous as to kind. The typhoid bacillus, the tubercle bacillus, the *Bacillus subtilis*, the streptococcus, the staphylococcus, and the colon bacillus have all been found here, although it is probable that the latter does not remain active, except for a short time.

Morbid Anatomy.—The gall-bladder is found to be filled with dark, mucopurulent material, in which, if the wall of the gall-bladder is seriously involved, there may be traces of blood. Occasionally the distention of the gall-bladder is due not only to blocking of the cystic duct by a stone, but the canal is closed by the intense inflammatory process. Perforation of the gall-bladder or gangrene of its walls may develop if the inflammation is very severe, and it not rarely happens that adhesions form between it and the nearby tissues.

Riedel states that such adhesions develop in no less than 75 per cent. of cases of cholecystitis. These adhesions are of importance because as a result of their formation a gallstone perforating the gall-bladder may find its way into adjacent organs. (See below.) Riedel also states that the adhesions depend as to their location to a large extent upon the position at which the stone exists. Thus, if it be in the gall-bladder the adhesions are between this viscus and the colon or the omentum. If it be in the cystic or common duct the adhesion is to the stomach, in the region of the pylorus. These adhesions are also of importance because they may cause pain or obstruction of the pylorus or duodenum.

Symptoms and Diagnosis.—The symptoms are those of acute inflammation in the hepatic area, varying in severity from a *slight discomfort* and *soreness* to *violent and alarming pain and collapse.* There is *tenderness,* particularly about the region of the gall-bladder, and this speedily may amount to exquisite pain on pressure. The point at which the greatest tenderness is felt is where the lower third of a line drawn from the navel to the ninth rib joins the middle third. With these symptoms there is *fever,* often ushered in by a *chill.* When the development of the condition is sudden, as it very frequently is, the patient may be seized with *nausea* and *vomiting, threatened collapse,* and other symptoms of fulminant abdominal disease.

The *pulse* is *rapid;* the *belly is distended* and its *walls rigid.*

Diagnosis.—Unless the pain is so localized as to aid materially in diagnosis, and unless the physician is provided with a history of gallstone colic, or of an inflammation in the gall-bladder after one of the acute fevers, as typhoid fever, the symptoms may mislead him into a diagnosis of intestinal obstruction or acute appendicitis; for paralysis of the bowel may be present, on the one hand, and in appendicitis the pain is often referred to the region of the epigastrium or liver. In certain cases of appendicitis, with a history of recurrent attacks or of a recent attack, the physician must also recall the fact that pain in the hypochondrium may arise from a septic focus, carried there from the appendix by the lymphatics. So, too, a gastric ulcer with perforation and subdiaphragmatic abscess may simulate acute cholecystitis. When palpation reveals an elongated gall-bladder projecting below the edge of the liver, which is very tender on palpation, the diagnosis is readily made. Jaundice may or may not be present. It is often absent. It is important to bear in mind the fact that attacks of hepatic colic may occur in cases of cholecystitis without any gallstones being present.

But in hepatic colic an examination of the blood will not reveal leukocytosis of polymorphonuclear cells, which will be notably increased by the presence of an acute inflammatory process in or about the gall-bladder. In questioning the patient as to the possible presence of gallstones it should be remembered that mild attacks of pain in this region may be as indicative of the passage of these bodies as a history of typical gallstone colic. Acute cholecystitis is rarely characterized by the suddenness of onset of pain and abdominal tenderness which are met with in acute pancreatitis or perforation of the stomach due to ulcer. If these symptoms are present they may, however, be due to perforation of the gall-bladder due to chronic cholecystitis arising from gallstones. (See Cholelithiasis.) Sometimes when there is an infection of the gall-bladder, as in the third or fourth week of typhoid fever, the onset of cholecystitis may be as severe as that of perforation. Occasionally during the course of acute ulcerative endocarditis with secondary cardiac failure the liver becomes enlarged and tender and chills and fever are met with. Pyopericardium must also be excluded if possible.

Treatment.—The treatment in all cases in which the symptoms are severe is prompt operative interference. Temporizing measures consist in the use of rest in bed, counterirritation over the region of the gall-bladder, and the use of gentle saline purges to unload the bowels.

CHOLELITHIASIS.

Definition.—The term cholelithiasis is applied to a condition in which the gall-bladder or the other parts of the biliary passages contains one or more gallstones.

Etiology and Pathology.—The predisposing causes of gallstone formation are all conditions which produce catarrh of the stomach and duodenum and biliary passages. A sedentary life with high living is a factor. So, too, enteroptosis may aid in its development. The condition is commonly met with after forty years of age, but cases have been seen in childhood and even in the newborn. More than 75 per cent. of all cases occur in women, and 90 per cent. of these women have been pregnant one or more times.

Biliary calculi are formed as the result of the deposit of certain of the ingredients of the bile about a nidus, which we now know is often, if not always, an accumulation of micro-organisms. The presence of these infecting agents has already been discussed in the article on Cholecystitis, but it is particularly important to bear in mind the fact that typhoid bacilli are frequently the origin of stone, probably because they often remain in the gall-bladder for years, and because, when they agglutinate, they form with epithelial cells a good nidus for the deposition of biliary materials. That micro-organisms play this part is now proved not only by many observations on man, but by experiments on animals.

The mere presence of micro-organisms, however, is not sufficient for the formation of stone. It is necessary that a catarrhal state of the mucous membrane be present, since in this condition three ingredients of the stone are excreted by the walls of the gall-bladder, namely, mucus, cholesterin, and a substance called "bilirubin calcium." Healthy bile prevents the deposition of bilirubin calcium, but if albumin is present this action is arrested and the deposit is made. When inflammation is present enough albumin enters the bile from the diseased mucous membrane to permit of this effect, and the small quantity of cholesterin present in normal bile is also much increased. It is evident, then, that for the deposit of the materials forming a gallstone an unhealthy mucous membrane is primarily essential.

Gallstones when composed chiefly of cholesterin are transparent or slightly tinged by bile. If broken, such a stone appears crystalline, with radiating lines. In other cases the stone is composed not only of cholesterin, but of biliary pigment and salts of magnesium and calcium. Such stones are usually dark in color, brown or green. They may be round or marked by facets, due to attrition, where they have rubbed against other stones.

In these stones also a radiating crystalline formation is present on fracture. These dark-faceted stones are the ones commonly found. More rarely stones of small size are found, composed almost entirely of bile pigment. Calcium carbonate stones are still less frequently met with.

In size gallstones vary from fine gritty sand to masses as large as a small banana.

In the vast majority of cases of cholelithiasis biliary calculi are formed in the gall-bladder. Very rarely small particles of biliary sand form in the bile-ducts of the liver itself. The large stones found in the cystic and common duct have formed in, and then slipped from, the gall-bladder.

The number of stones found in the gall-bladder may vary from one or two to several thousand, if the tiny, sand-like pieces are counted. When the number is large, they usually show signs of lateral pressure, but sometimes several may exist without facets being developed.

If a gallstone lodges in the common gall-duct so as to completely occlude it, there is usually found at autopsy a condition of dilatation of this duct, which is filled with a clear, mucus-like fluid. (See Occlusion of the Bile-ducts.) If the obstruction is not complete and infection of the duct takes place, the state is one of cholangitis, already described, or even of suppurative angiocholitis.

When the cystic duct is completely obstructed by a stone, the gall-bladder may be greatly enlarged and filled with clear fluid or with other gallstones. If the gall-bladder is infected suppurative cholecystitis develops, and perforation may occur. (See Symptoms.) In other cases the gall-bladder undergoes atrophy, and may be so shrunken as to be nothing but a small mass of fibrous tissue the size of a large nut, hidden in the hollow naturally occupied by the gall-bladder. Less commonly a process of calcification is developed, and the gall-bladder becomes coated or infiltrated by lime-salts.

Symptoms.—It is important to bear in mind the fact that the mere presence of gallstones in the gall-bladder does not necessarily cause any symptoms whatever. The records of autopsies in Germany, in particular, show that a very large proportion of all women who come to autopsy in the later years of life have gallstones, and yet there has been no suspicion of their existence prior to death. Only about 5 per cent. suffer from distinct symptoms due to this cause. On the other hand, if the biliary tract becomes infected, or if an acute congestion of its mucous membrane occurs, more or less severe symptoms may be at once produced and fever may develop; or if a stone becomes dislodged from the gall-bladder and slips into the cystic or common duct, this mechanical difficulty may at once produce biliary colic.

The *symptoms of biliary colic* usually consist in *severe pain*, which amounts to an agony in most instances. Occasionally, however, the pain is very moderate, and is thought to be due to indigestion. The patient often *vomits* and *sweats* profusely. The pain manifestly originates in the gall-bladder, but is radiated to the right shoulder-blade and to the epigastrium. The *facial expression* is one of *anguish* and *anxiety*, and the color of the skin is pallid.

After the attack has lasted for some hours, or on the day after an attack, a *moderate degree* of *jaundice* may appear, but it is rarely well marked unless the attack lasts for several days or the obstruction is persistent. If the stone is in the cystic duct, no jaundice occurs unless the neighboring mucous membrane in the common, or hepatic duct, becomes swollen and inflamed, or unless the stone is so placed in the cystic duct that it presses upon the hepatic duct. The presence of jaundice in a case which suffers from severe pain in the region of the gall-bladder is a positive diagnostic sign of much value, but the absence of jaundice does not in the slightest degree negative the view that gallstone is present. Kehr states that in 720 cases operated on for gallstone, 80 per cent. showed no jaundice.

The urine, if the stone is in the common duct, may soon show the presence of bile, and not rarely albumin is found in it. In still other cases red blood cells may be found in the urine, and this may lead us into the belief that the pain is due to renal colic.

An attack of biliary colic lasts, as a rule, for but a few hours, but occasionally the patient suffers from a prolonged seizure lasting over several days and marked by temporary remissions, which are, perhaps, due to exhaustion of the irritated gall-bladder or to temporary restoration of biliary flow.

The presence of a stone or stones in the common or cystic duct produces not only symptoms of biliary colic in some cases, but other signs as well, which may be of use in diagnosis. If the stone blocks the *common duct* completely, the jaundice which develops is persistent and well marked, and further attacks of colic may never occur, or, indeed, there may not be a single attack in the patient's history. Febrile movement is usually absent, because the complete obstruction of the duct prevents infection from the intestine. The gall-bladder is usually not distended.

When the common duct is not completely closed, and in the majority of cases it is not occluded, the attacks of biliary colic are more frequent, and the degree of jaundice varies. This is due to the fact that at times, when the mucous membrane surrounding the stone is not acutely inflamed or congested, bile is permitted to escape into the bowel, so that the pressure is relieved and the stools become bile-stained. Such a condition of repeated attacks of colic with varying degrees of jaundice may also be due to the stone becoming so fixed in the ampulla of Vater that it forms a ball-valve, which sometimes permits the passage of bile and sometimes prevents it. In rarer instances the stone becomes encysted in the wall of the duct, and so acts as a valve, and in still other cases it may become lodged at the junction of the cystic and hepatic duct, and by pressure cause symptoms characteristic of obstruction in both the cystic and common duct.

These cases of partial obstruction differ from those of complete obstruction, in the fact that they not rarely develop fever, owing to infection of the common and hepatic duct by micro-organisms from the bowel. The febrile attacks which ensue may be so irregular or so intermittent in type that they closely resemble those of malarial fever, but they are in reality septic fever, the so-called "intermittent hepatic fever of Charcot." Such attacks may persist for years with no more serious changes in the ducts than a chronic catarrh with thickening and the proliferation of an exudate about the parts.

In some cases, however, the degree of infection is so severe that suppuration takes place not only in the common duct, but in the hepatic duct as well, and even in the gall-bladder, producing suppurative cholecystitis and suppurative angiocholitis. See Suppurative Inflammation of the Bile-ducts and Acute Cholecystitis.

The additional symptoms to those of biliary colic which arise when the cystic duct becomes the lodging-place for a stone are chiefly enlargement of the gall-bladder from distention and the negative, but nevertheless valuable, evidence of absence of jaundice. The size of the gall-bladder in cases in which the obstruction is complete is sometimes marvelous. Instances have been recorded in which the enlarged gall-bladder has been mistaken for an ovarian tumor, and not rarely the enlarged pear-shaped mass can be felt near the median line of the abdomen. See Diagnosis. On the other hand, the facts in regard to atrophy of the gall-bladder, already named in the discussion of the pathology of this affection, should be recalled; in other words, the absence of a large gall-bladder does not exclude obstruction to the cystic duct.

If the gall-bladder which is distended by retained bile and gallstones can be palpated, it may be possible to produce what is called "gallstone crepitus" by the rubbing of the stones one upon the other.

Complications and Sequels.—A stone may perforate the gall-bladder, and, by way of an adhesion, gain the cavity of the duodenum and escape with the feces. In still other cases the perforation takes place through an adhesion to the colon, but very rarely does the stone escape into the small bowel below the duodenum. In other instances the gall-bladder becomes adherent, by an inflammatory exudate, to the abdominal wall, and the stones finally escape from the fistulous opening. I had a case in my clinic at the Jefferson Hospital some years since in which the patient passed almost daily a little pus and a little bile with one or more stones through such an opening, yet seemed in excellent health, probably because nature had established free drainage. More commonly the perforation takes place so that the stone enters the peritoneal cavity and then the associated infectious material causes local peritonitis. When the gall-bladder becomes adherent to the diaphragm and perforation ensues, the stone with pus and bile may escape into the pleura or into the lung. In the *Transactions of the Association of American Physicians* for 1897, Schachner, of Louisville, reports 11 cases of cholelithiasis perforating into the lung, and in 4 of them the stone passed through an adhesion which existed between the gall-bladder, the diaphragm, and the pleura.

The spitting of bile, with a distressing cough, and dulness on percussion in the area just above the liver, where pulmonary resonance is usually present, make the diagnosis certain.

Gallstones have been carried far away from the gall-bladder by suppuration after perforation, and have even been found in the urinary bladder as a result of this process. Perforation of the gall-bladder with fatal syncope has been reported during an attack of biliary colic.

Diagnosis.—This is not difficult if we are able to exclude appendicitis, inflammatory diseases, gastric ulcer, gastralgia, the gastric crises of ataxia, and renal stone. Appendicitis is detected by finding even greater pain on palpation in the appendicular area; pleurisy is excluded by careful auscultation to reveal a friction sound. In gastric ulcer there is a history of pain immediately after the taking of food, and perhaps of hæmatemesis; and the patient is usually a young woman, whereas gallstones are usually present in women past forty years of age. A person with an ulcer is usually poorly nourished and anæmic, whereas the patient with gallstones is usually plump and possessed of a thick abdominal wall. Hyperchlorhydria is present in ulcer, but absent in cholelithiasis, as a rule. In those cases, however, in which there are adhesions between the gall-bladder or its duct and the pylorus or duodenum, these differential signs may fail because pyloric obstruction causes pain after taking food and produces hyperchlorhydria. Gastralgia is pointed to by the fact that the patient is subject to neuralgia, and is neurotic in type, rather than stout, as are most gallstone cases. An attack of gastric crisis in ataxia, while often associated with vomiting, can be detected by the history of an ataxic gait and by the presence of an Argyll-Robertson pupil or other signs of that disease. Renal stone causes pain to be radiated to the inside of the thigh and to the testicle. Movable kidney may, by dragging or pressing upon the common biliary duct, cause obstruction, and so produce an attack of biliary colic and jaundice not due to stone. Further, the pain due to twisting of the ureter in such a case may simulate hepatic colic. The finding of a floating kidney clears up the diagnosis. A new-growth may produce similar symptoms, and it may be impossible to differentiate the obstruction from that due to gallstones. In none of these states is the greatest degree of pain on pressure over the gall-bladder.

It is essential that attention be paid to one very important differential point, which must always be borne in mind, the so-called "Courvoisier's law," namely, that an enlarged gall-bladder *with jaundice* is a sign of malignant growth of the gall-bladder rather than that of obstruction due to stone.

Treatment.—The treatment of biliary colic, like that of renal colic, consists in the administration of a hypodermic injection of $\frac{1}{4}$ of a grain of morphine, with $\frac{1}{150}$ of atropine, and $\frac{1}{100}$ of a grain of nitroglycerin, to relieve pain and to relax spasm. If the first injection does not give relief at the end of fifteen or twenty minutes it may be repeated, the atropine being left out. After the attack is over the patient should rest quietly in bed for two or three days, in order to hasten the disappearance of the inflammation of the mucous membrane, which is usually associated with the attack, and thereby decrease the danger of a subacute or chronic inflammatory process developing in the gall-bladder or common duct.

No medicines have any effect upon the gallstones which are already formed, but a number of remedies may be given to patients who suffer from gallstones, with the object of preventing the formation of others, and with the hope that by their use catarrh and irritation of the mucous membrane lining the gall-bladder and the common duct may be materially diminished. These remedies consist in the mild saline purgatives, such as the various imported purgative waters, which are gentle in their action, and which may be preferably taken hot in the dose of one or two teacupfuls before breakfast. Chloride of ammonium, in the dose of 5 to 10 grains three times a day, is useful for its effect upon mucous membranes, and is, perhaps, best given in equal parts of fluid extract of licorice and water.

Until within recent years the physician was content when a case of cholelithiasis escaped month by month from a return of biliary colic, deeming it inexpedient that any radical measure of relief should be instituted. With our present knowledge, however, it cannot be doubted that the question of operative interference must be carefully considered in every case in which a positive diagnosis of cholelithiasis can be made. The time for operation is, of course, during a period of quiescence, since at the time of an attack the acute inflammation which exists in and around the gall-bladder may seriously complicate the work of the surgeon.

The question as to how frequently the patient should be allowed to suffer from gallstone colic before operative interference is urged is one which varies with each individual case. If the jaundice which is present with the first attack is not severe and lasts but a very short time, and if the temperature of the patient is not disturbed, or returns to normal within a period of twenty-four hours, and if, again, palpation in the neighborhood of the gall-bladder some days after the attack fails to reveal evidence of a low-grade inflammation, as manifested by tenderness or pain, it is then permissible, and, indeed, advisable, that the patient should not be operated upon. Even when as many as two or three such mild attacks have occurred at long intervals, the condition may not be such as to require that the physician should strongly recommend surgical aid. When, however, the attack of biliary colic is violent or repeated, when the jaundice persists for a long period, and when distinct evidence of persistent cholecystitis continues, then operation should be resorted to, particularly if a mass, caused by the gall-bladder being distended by stones, can be distinctly felt. To delay operation in a case of this kind until frequent attacks have resulted in the formation of a large amount of inflammatory material about the gall-bladder is very unwise. By this means cases which would offer under ordinary circumstances no surgical difficulties may become almost inoperable, and what should be an easy convalescence may be instead a difficult and prolonged illness, testing the skill of both the physician and surgeon to the utmost.

Mayo has performed 510 operations for gallstone, with a mortality of only 3 per cent. On 208 cases in which stones were present in the gall-bladder alone, and which were uncomplicated by any other condition, there were 2 deaths—a mortality of less than 1 per cent.

Grouping together all cases complicated by the presence of stones in the common or cystic ducts, cases of cholelithiasis in which infection occurred, cases of biliary infection and malignant disease, Mayo finds the mortality to be 5 per cent., or 16 deaths out of 326 cases which he operated on. He believes these figures to be a strong argument in favor of early operation in cases of cholelithiasis.

MALIGNANT GROWTHS OF THE GALL-BLADDER AND BILIARY PASSAGES.

Etiology.—The cause of the development of morbid growths in these parts is unknown, and only some of the predisposing causes are recognized.

In the case of carcinoma of the biliary ducts and the gall-bladder, there can be no doubt that age has a very distinct influence. The growth usually develops later in life than does carcinoma in any other part, namely, about the fifty-sixth year; whereas, the period of greatest frequency of cancer of the mammary gland is, according to Kelynack, about the fortieth year. It would also seem probable that gallstones very distinctly predispose to the development of carcinoma in these parts, for they are found present in from 90 to 95 per cent. of all cases; whereas, the frequency of gallstones in persons dying from other diseases than cancer of these parts is from 6 to 12 per cent. (Kelynack). On the other hand, it has been claimed that the presence of carcinoma of the gall-bladder leads to the rapid formation of stone. Probably the pathological condition of the mucous membrane which aids in the formation of stone (see Cholelithiasis) also predisposes to the development of a morbid growth, and this effect may be increased by the irritation produced by the stones after they are formed.

In women carcinoma of the gall-bladder is far more common than it is in men. Musser, in his classical paper upon this subject, found that out of 98 cases 75 were in women and only 23 in men, and other clinicians have noted an even greater percentage among women. Curiously enough, this is not the case when the growth affects the biliary passages other than the gall-bladder, for in such instances the number of men and women affected is practically the same.

Pathology and Morbid Anatomy.—Carcinoma of the gall-bladder is usually of the type of cylindrical-cell epithelioma, but in statistics there is much contradiction as to this point. This form of cancer is also the type which most commonly affects the biliary ducts. When the gall-bladder is affected the fundus is the part that usually suffers. If the biliary passages are involved, the common duct is the part that is usually affected, and that portion of it where it enters the bowel is the favorite site of the growth. (See Fig. 92.)

When cancer attacks the gall-bladder, as already pointed out in the article on Cancer of the Liver, the liver is affected in a large percentage of cases by secondary growths (Musser says 54 per cent.). The pancreas is also very commonly involved. When the growth is in the biliary ducts, metastasis to other parts is rare.

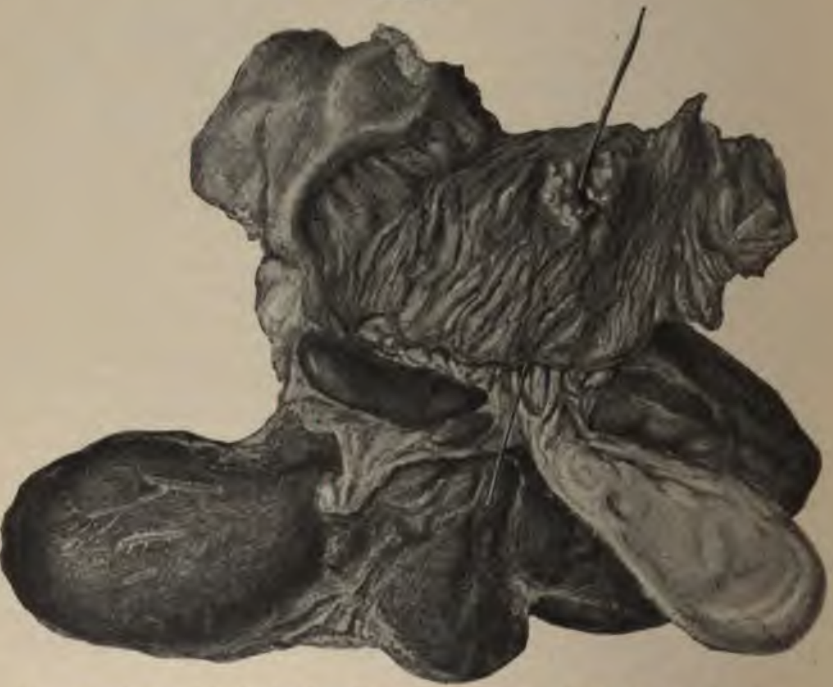
Carcinoma of the gall-bladder may also result in the formation of adhesions, by which it becomes attached to the abdominal wall, the colon, and even the stomach and small intestines. In other instances a suppurative cholecystitis may develop.

Symptoms.—The symptoms of cancer of the gall-bladder are most varied. If the growth is so situated that it interferes with biliary flow, the manifestations of hepatic disease may develop while it is still a very small mass;

whereas, if no pressure is produced, the tumor may reach a very considerable size before its presence is suspected.

As soon as the tumor reaches any size it can be palpated below the border of the last rib in most instances, particularly if it affects the fundus of the gall-bladder, as it does in the majority of cases. Its position is usually along the outer edge of the right rectus muscle and about the neighborhood of the normal gall-bladder. It may extend downward toward the pelvis, or it may be erect and protrude through the abdominal wall, as an aneurysm protrudes from the chest. The tumor feels hard and is usually somewhat pear-shaped. It varies in size from that of a child's head to that of a small walnut.

FIG. 92



Primary carcinoma of the duodenal papilla seen at the upper end of the rod running through the duct. (Kast and Rumpler.)

If the growth is so placed near the neck of the gall-bladder that it prevents the cystic duct from draining that viscus, the resulting distention of the gall-bladder may cause the formation of a greatly distended sac, which may extend far below the ribs, and by its pressure upon the bowel cause intestinal obstruction.

Jaundice and *pain* are very constant symptoms of cancer of the gall-bladder. Musser found jaundice in 69 per cent. of his cases, and pain in 62 per cent. When the growth invades the gall-bladder alone, it does not of itself cause icterus, but the neighboring glands are often involved very early, and by this means, or by secondary growths in the common duct, jaundice is produced. In this connection "Courvoisier's law" is to be recalled, namely, that given an enlarged gall-bladder with jaundice the cause is carcinoma, not gallstone.

Wasting is usually well marked, not only by reason of the malignant growth, but because of the jaundice and gastroduodenal catarrh which are usually present. Ascites may develop (18 per cent. of cases, Musser). Death is due to exhaustion, and sometimes to cholæmia.

Diagnosis.—A growth in the gall-bladder can usually be differentiated from one in the bile-ducts by its size, the readiness with which it can be palpated, and the absence of jaundice until it is of some size. Given a case in which jaundice develops in a woman well along in years, in which the jaundice remains persistent and in which no tumor can be felt, and the diagnosis is in favor of growth in the duct. If metastatic masses can be found in the liver, the primary growth is probably in the gall-bladder, for they rarely develop from a growth in the duct.

An even more important differential point is that between cancer of the gall-bladder with cystic enlargement and enlargement due to hydrops of the gall-bladder. In the latter case the gall-bladder is distended with a clear fluid as the result of the presence of stone or other cause, completely closing the cystic duct. As Courvoisier found this state of hydrops 79 times in 91 cases of impacted gallstone, it is not a very rare condition. The differentiation between these two states is made as follows: In hydrops of the gall-bladder there is usually no jaundice, and the tumor presents a smooth, pear-shaped surface, and there may be a history of gallstone colic. Bile is present in the stools and the degree of wasting is not marked.

In some rare cases the gall-bladder undergoes thickening and presents a small, hard mass, which may be palpated. This has already been described elsewhere as calcification of the gall-bladder. The absence of metastasis and of other signs of cancer also aids us in excluding the morbid growth in these cases.

Other causes of tumor in the region of the gall-bladder are aneurysm, cancer of the pylorus, cancer of the head of the pancreas, and tumor of the kidney. A fecal mass in the bowel may also mislead the physician.

Prognosis.—Life, in a case of cancer of the gall-bladder, usually does not last beyond a year, and in many instances death comes earlier than this.

Death usually comes much earlier in cases of malignant growth in the duct than of malignant growth in the gall-bladder.

Treatment.—The treatment of malignant growth of the gall-bladder, of necessity, does not offer much promise for ultimate and complete recovery, for medicinal measures are, of course, entirely useless, except for the purpose of relieving pain. Operative procedures, even if they result in the complete removal of the diseased gall-bladder, are nearly always followed by the development of secondary growths in neighboring parts, where they are inoperable and where they straightway proceed to destroy the patient. Nevertheless, given a case in which the gall-bladder is greatly enlarged, and in which evidences of the presence of a growth in nearby parts is not marked, it is certainly permissible, and, indeed, advisable, that operation shall be performed, with the hope that the growth can be removed, or that the operation will reveal the fact that the enlargement of the gall-bladder is not cancerous in origin, but dependent upon the presence of gallstone, or other cause, or obstruction of the cystic or common duct.

In all of these cases in which jaundice is present, the very grave influence of this complication upon operative procedure must be taken into consideration, for while, on the one hand, we are learning every year that operations upon the gall-bladder can be performed with a surprising degree of impunity, we have also learned with increasing experience that jaundiced persons withstand operations badly, and that the presence of bile in the blood predisposes the patient to obstinate and persistent capillary hemorrhage during and after operation. This has not infrequently resulted in death, although the mere operative procedure itself was a success.

When the growth involves the bile-duct the outlook is, of course, even more discouraging, for the bile-duct cannot be excised, and the jaundice is usually so extreme that the condition of the patient scarcely justifies operative interference. The most that can be done for a patient with a malignant growth in the bile-duct is to operate with the idea of providing drainage and relieving pressure.

ICTERUS NEONATORUM.

Jaundice of the newborn is a very common condition, and usually appears about the third or fourth day after birth. It is noticeable in the skin and in the conjunctiva, but it is never intense, and in many cases may be so slight as to be overlooked. Holt states that it occurred 300 times among 900 babies, and Kehrer asserts that in his statistics it was present in 75 per cent. The last of these estimates is much too high a proportion for private practice. Hofmeier and others believe that it is hæmatogenous in origin, and results from the destruction, shortly after birth, of a large number of red blood cells. On the other hand it not rarely happens that the urine is slightly bile-stained, and the stools are lacking in bile, which would indicate disordered hepatic action. In some instances the portal vein may have been overdistended in birth, and in this way the finer bile-ducts may have been obstructed. Whatever the cause, in its simple form the condition possesses no evil import whatever.

A severe and rare form of icterus of the newborn may, however, develop and cause the death of the child. It is due to sepsis following infection of the umbilical vein, with phlebitis, to congenital syphilis of the liver, or to congenital atresia of the biliary ducts, so that the bile cannot escape into the bowel.

DISEASES OF THE PANCREAS.

PANCREATITIS.

Definition.—Pancreatitis, as its name implies, is an inflammation of the pancreas. It occurs in three forms, although it must be understood that no hard and fast lines separate these conditions one from the other. These three forms are the *acute*, the *subacute*, and the *chronic*.

Acute Pancreatitis. History.—As long ago as 1672 Tulpius described an acute abscess of the pancreas due to pyæmia. In 1799 Baillie studied what was evidently a case of chronic interstitial pancreatitis. In 1804 Portal recorded an instance of acute suppurative pancreatitis, as did Percival in 1818. In 1879 Balzer reported a case of acute pancreatitis, with fat-necrosis but it was not till the epoch-making paper of Fitz appeared in 1889 that the profession recognized the frequency and importance of lesions of this gland, although in 1886 another American practitioner, Senn, had written a paper upon its surgery. Von Mering and Minkowski made valuable contributions in 1889 and 1890.

Chronic pancreatitis was not recognized till Birch-Hirschfeld described it in 1895. Since that time the literature on pancreatitis has become quite voluminous, and a number of studies have appeared, of which the most notable, from the surgical standpoint, have been those of Robson and Moynihan, of Leeds, and, from the pathological standpoint, one by Opie, of Baltimore, who has done more than anyone else to throw light on diseases of this organ.

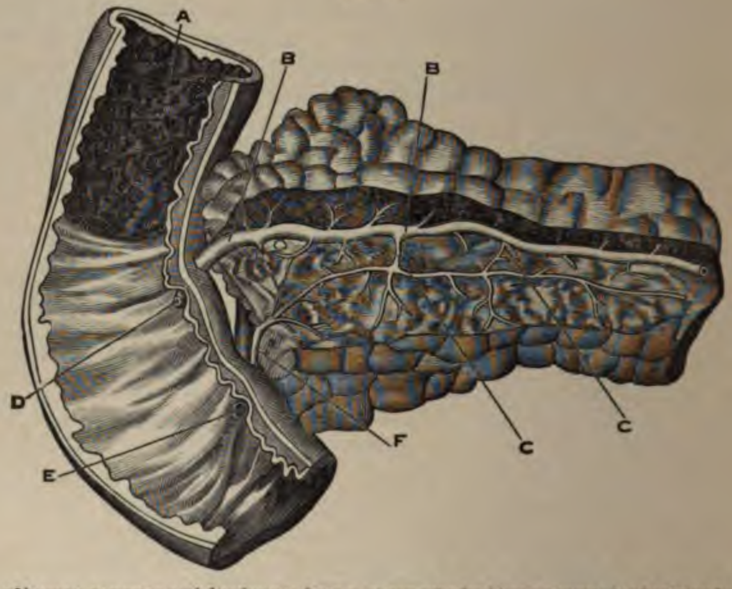
Etiology.—By far the more common cause of pancreatitis is infection of the gland by way of its duct. This results usually from the presence of a gallstone in the ampulla of Vater, which prevents the flow of bile into the bowel, but does not occlude the opening of the pancreatic duct. Again, the presence of a gallstone is prone to result in the development of a septic process in the surrounding mucous membrane, and this results in infection of the bile, which fluid passes along the duct of Wirsung and so enters the pancreas. This is particularly apt to occur if the gall-bladder is so shrunken by disease that it cannot readily expand when the bile is dammed back into the ducts. Further than this, bile alone entering the pancreas, even if it be free from micro-organisms, may cause hemorrhagic pancreatitis and fat-necrosis.

It has been argued that if these causes are active in the production of pancreatitis, this state would be met with much more frequently. The explanation of the fact that so many cases suffer from gallstones and gall-bladder infections without pancreatitis lies in the fact that in a certain proportion of individuals the bile-duct and pancreatic duct do not enter the bowel by one opening, but through separate openings. In a few cases the chief secretion of the pancreas escapes by way of the duct of Santorini. (See Fig. 93.)

Acute pancreatitis may result from several other causes, such as one of the acute infectious diseases, or, again, in the course of septicæmia. Another cause is infection from a neighboring lesion, as in direct extension from a gastric ulcer or cancer, or a subphrenic abscess. Still another cause is injury by blows or by surgical procedure.

Pathology and Morbid Anatomy.—Acute pancreatitis is characterized not only by a primary hyperæmia of the gland, but in some cases by the escape of its ferments into its own tissues, and adjacent tissues as well. As a result of this accident, a condition of "fat-necrosis" develops in the fatty tissues in the immediate region of the pancreas, and often in other parts of the body to which the pancreatic ferment may escape in the lymph. The fat is split up into glycerin and fatty acids. The glycerin is absorbed; the acids, being insoluble, are not eliminated, but combine with calcium salts, and with the necrotic fat form yellowish-white patches in the retroperitoneal fat, and in the fat of the omentum, mesentery, abdominal wall, and it may be in other parts.

FIG. 93



Portion of human pancreas and duodenum from a person who had been executed (after Claude Bernard Physiologie Expérimentale, 1856, tome ii. p. 185), showing a condition in which pancreatic juice may enter the intestine, although the gall-duct is obstructed: A, duodenum showing the projections formed by Brunner's gland; B, B, the large pancreatic duct; C, C, anastomoses between the small pancreatic duct and the large pancreatic duct; D, opening of the superior pancreatic duct into the intestine; E, opening of the small inferior pancreatic duct into the bile-duct.

In a certain number of cases of acute pancreatitis, but not in all cases, hemorrhage into the body of the gland occurs, forming what is known as acute hemorrhagic pancreatitis. (See also Hemorrhage into the Pancreas.) Whether this hemorrhage is the result of the action of the ferment of the gland upon its own vessels is not known.

Acute pancreatitis may proceed to suppuration (suppurative pancreatitis) or to gangrene (gangrenous pancreatitis). The suppurative form may be circumscribed or diffuse. In some cases the pus may be confined to the pancreas or peripancreatic structures, but the lesser peritoneum, or even the general peritoneum, may be involved.

Symptoms.—In acute pancreatitis the patient is suddenly seized by a *severe pain in the epigastric region*, which is associated with *faintness*, or even *collapse*, and it may be *active vomiting*. Inquiry reveals the fact that *constipation* is present, and so the physician may be misled into a diagnosis of intestinal obstruction, but doubt is thrown upon this belief by the fact that gas can be passed by the anus. The pain may be paroxysmal in type, and there is *great tenderness* in the *epigastrium* upon pressure. This area becomes swollen and tense, and as the case progresses the entire abdomen may become distended. The *face is pinched* and *anxious-looking*, and the upper lip drawn as in acute peritonitis. The *vomit* may become *black* and tarry from the presence of altered blood.

If the patient survives more than a few hours, a more or less *marked jaundice* develops, provided that the bile is prevented from escaping into the bowel by swelling of the mucous membrane or the presence of a stone. *Hiccough* may be an annoying symptom, and the *pulse* is *rapid* and *running*. The *temperature* may be *febrile*, *normal*, or *subnormal*. The urine may contain casts and albumin. Death in collapse with profound asthenia usually occurs by the second to the sixth day, but recovery may occur in mild cases, and it sometimes happens that these acute symptoms gradually merge into the subacute type of the malady.

Subacute pancreatitis cannot, of course, be sharply separated from the acute form, yet cases occur in which the symptoms are sufficiently modified to indicate that another form of pancreatitis is present. The malady *is not so sudden* in its onset, the pain is not so excruciating, and the epigastric swelling is neither so great nor so rapid in its development. The pulse is less rapid, and the constipation is more prone to give place to diarrhœa, the stools containing pus, tarry blood, and undigested food. Chills also occur, and after a few days a swelling, due to abscess, may be felt. This abscess may burrow in such a way as to form a perirenal abscess, a psoas abscess, or a subphrenic abscess, or it may burst into the stomach or bowel. The symptoms of sepsis are, of course, present in such a case, and if surgical relief is not given death is due to this cause or to profound asthenia. Such a case must be separated from perforation of a gastric ulcer, from abscess due to caries of the spine, from suppurating cholecystitis, and from perirenal abscess.

Diagnosis.—Fitz gives the following rule for the diagnosis of this state: "Acute pancreatitis is to be suspected when a previously healthy person, or a sufferer from occasional attacks of indigestion, is suddenly seized with violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours by a circumscribed epigastric swelling, tympanitic or resistant, with slight rise of temperature." These symptoms are still more indicative if there is a history of gallstone colic.

Acute pancreatitis must be separated from intestinal obstruction, perforation of the duodenum, and the perforation of a gastric ulcer. It must not be confused with rupture of the gall-bladder, suppurative cholecystitis, and fulminating appendicitis. The differential diagnosis from intestinal obstruction has already been touched upon. If doubt exists an operation will reveal the true cause of the symptoms, for in pancreatitis fat-necrosis may be evident as soon as the belly is opened. The operation is justified, because it ought to be performed in either instance to save life. When perforation of the stomach or duodenum is considered, the history of an old gastric ulcer or of hemorrhage from the bowel, in which dark, tarry blood is passed, will be of importance, and here, again, an operation to save life is necessary, whether the condition be pancreatic disease or perforation.

When suppurative cholecystitis is suspected, it may be confirmed by the discovery that the early swelling is in the neighborhood of the gall-bladder, and a history of typhoid fever or of gallstones will be presented. However, this latter history may be equally suggestive of secondary pancreatic disease. Here, again, an operation primarily for diagnosis and secondarily for relief is indicated.

Perhaps the most important differential diagnosis lies between acute pancreatitis and fulminating appendicitis, because appendicitis is a common disease, because the pain accompanying it is often referred to the epigastrium, and because an incision in the pancreatic region is far removed from that required for appendectomy.

Palpation of the appendix will usually elicit pain over its site, and, perhaps, localized swelling. Examination by the rectum may reveal marked iliac tenderness and a history of repeated attacks of appendicitis may be found.

A valuable aid to diagnosis in these cases is the examination of the urine for leucin and tyrosin, which are not present in healthy urine, although they may be present in other conditions than pancreatitis. The urine is precipitated with basic lead acetate and filtered. The filtrate from which the excess of lead has been removed by hydrogen sulphide is now evaporated almost to dryness and allowed to stand for crystallization to take place, when crystals of leucin and tyrosin may be found. (For a valuable article on the chemistry of the urine in disease of the pancreas see Cammidge, *British Medical Journal*, April 2, 1904.)

Opie has described a test for lypolytic substances in the urine, and Cammidge has advocated a test for glycerin derivatives in the urine as aids to diagnosis; but neither of these methods is readily employed by the general practitioner.

Prognosis.—Acute pancreatitis is always an exceedingly grave state. Death ensues in the majority of cases. Recovery occurs in the very mild cases, as has been proved by instances in which a subsequent operation has revealed the evidences of the disease.

Treatment.—The treatment of acute pancreatitis consists in the prompt institution of surgical proceedings as soon as the shock of the onset of the disease has been overcome. Robson insists that the surgeon should not wait until collapse passes off, as the collapse may be due to septic absorption, which only an operation can relieve. By the relief of pressure, the providing of drainage, and the removal of the cause of the attack, if it exists in the gall-duct, relief may be given the patient and the process arrested before it has proceeded too far. In a certain proportion of cases, however, the state of the patient from the onset is too grave to permit of surgical intervention. Excessive pain is to be relieved by morphine given hypodermically, and collapse is to be treated by the employment of strychnine and atropine.

The treatment of the subacute form of the disease consists in supporting the nutrition of the patient by the use of predigested foods and stimulants, and by surgical intervention for the relief of tension and removal of pus as soon as the diagnosis is made and the patient is strong enough to stand operative procedures.

Chronic Pancreatitis.—This form of pancreatitis has been in the past considered quite rare, but it would seem probable that it exists more commonly than is generally thought. It is usually developed as a result of chronic catarrh of the duct of Wirsüng, which, in turn, is caused by chronic gastroduodenal catarrh or by cholelithiasis, pancreatic lithiasis, or by gastric ulcer.

Of chronic pancreatitis, Opie, of Baltimore, thinks there are two types. One is an interlobular pancreatitis, with increase in the connective tissue between the lobules; the intralobular tissues being unaffected and the islands of Langerhans escaping until very late in the pathological process. This is the type of chronic pancreatitis which follows blocking of the pancreatic duct by a morbid growth or by pancreatic or biliary calculi. Glycosuria is rarely present, because the islands escape. This is the common form. In the second form there is an interacinar pancreatitis, that is, new connective-tissue formation in the lobules themselves. The islands of Langerhans are seriously affected in this type, and hence glycosuria is usually present. This form is not due to obstruction of the ducts.

These pathological facts show why it is that glycosuria does not appear as a sign of pancreatic disease in most cases in which, as the result of gall-stone disease or other sources of infection or obstruction, we suspect the presence of pancreatitis. For a recent and exhaustive consideration of the Relation of the Islands of Langerhans to Morbid Conditions of the Pancreas and Diabetes Mellitus see Finney's article in the *Medical Chronicle* for June, 1903.

The interstitial type, like the more acute forms, may be diffuse or localized. When localized a single area may manifest the change. A somewhat subacute form is also recognized, and in some of these cases there is a conspicuous enlargement of the area involved, so that the head of the pancreas when affected may be mistaken for a neoplasm. Presumably this enlargement represents a cellular infiltration, which later passes on to a fibrosis. The typical fibroid pancreas is small, dense, resists incision, and may creak when under the knife. Fatty infiltration is usually also present, and evidences of a primary acute process may be manifest. On microscopic examination there is a notable increase in the fibrous tissue, which, as already stated, may be perilobular or interlobular, or it may be interacinar. In the latter form the islands of Langerhans show hyaline or fibrohyaline transformation, or they may be absent from certain areas, or, perhaps, cannot be demonstrated in any part of the organ. The ducts may show dilatation, and in some instances be stained with bile.

Symptoms.—Chronic pancreatitis may be divided into those cases in which the symptoms are mild and those in which they are severe. In one case there may be little if any pain, but there is present *persistent loss of flesh,* and in the epigastrium, near the gall-bladder, may be found a hard mass which may be mistaken for a malignant growth in the stomach or gall-bladder. The patient complains of *epigastric distress,* such as that which follows taking food which is difficult of digestion, and attacks of *vomiting* may occur. Sometimes *jaundice* appears. Fever may be present if the infection of the ducts is marked.

In the more severe form of chronic pancreatitis, the symptoms are in close accord with those of the subacute form, in that the symptoms may begin with severe pain in the epigastrium, as if the patient had gallstone colic. There is tenderness in the midepigastrium. *Chills* and *fever* may be well developed.

Diagnosis.—Chronic pancreatitis is to be separated from gallstones in the ductus communis choledochus, from cancer of the head of the pancreas, from cancer of the gall-bladder and of the liver, and from subphrenic abscess. From impacted gallstones in the common duct the differentiation is practically impossible. Tenderness on deep pressure will usually be found over the gall-bladder in the case of gallstones and over the epigastrium in pancreatic disease, and etherization may permit sufficiently deep palpation to feel the enlarged head of the pancreas if chronic pancreatitis is present. The distribution of the pain may be of value in the differentiation. In gallstone cases the pain is in the gall-bladder area and radiates around to the right scapular region; whereas, in pancreatic disease it is central and travels directly backward to a space between the ends of the scapula.

Cancer of the head of the pancreas is a disease of advanced life; its onset is usually painless, and the jaundice that may ensue is gradual in onset and persistent, because the growth primarily, or secondarily, presses continually on the gall-ducts. The presence of great swelling of the gall-bladder in association with jaundice and with the other symptoms of pancreatic disease already named indicates cancer. Sometimes in these cases secondary cancerous nodules are to be found in the liver and in other organs. The liver may be much enlarged from the damming back of bile and from secondary growths. Robson and Moynihan lay stress on the fact that the absence of pancreatic secretion from the stools causes them to be clay-colored, even when bile is present, and this may aid in diagnosis. If on testing the stools bile is found, and the feces are, nevertheless, light in hue, it is a fair supposition that pancreatic secretion is absent. The test of the urine for leucin and tyrosin, already described, should also be used.

There are two other facts which, if present, will decide the diagnosis positively, namely, fatty stools due to the lack of pancreatic juice in the bowel and the presence of glycosuria due to the invasion of the islands of Langerhans in the pancreas by the pathological process. Both of these signs are often absent, but if they are present they are pathognomonic of pancreatic disease. (See Diabetes Mellitus.)

Prognosis.—Patients suffering from chronic pancreatitis in moderate degree may live for months or even for years. If glycosuria develops the prognosis becomes very grave, not only because this symptom is grave in itself, but because the glycosuria indicates that the involvement of the gland in the disease process is well-nigh universal, for as long as but a few islands of Langerhans exist glycosuria does not ensue. Great emaciation, marked jaundice, and the development of a tendency to have multiple hemorrhages are evil signs.

It is in this form of pancreatitis that surgery has given its most valuable results, often producing perfect recovery.

Treatment.—The treatment of the mild form of chronic pancreatitis depending upon catarrh of the biliary and pancreatic ducts may be medicinal for a time in the hope that the condition may be relieved. The measures instituted are practically identical with those advised in cases of chronic cholangitis (which see). It is, however, a vital mistake to permit these cases to drift along in a state of chronic ill health, because the condition is one which will eventually lead to pancreatic cirrhosis, and this in turn results in great emaciation and the development of glycosuria, which causes death. In other words, if the patient does not improve surgical measures should be adopted to give relief.

The treatment of chronic pancreatitis of the severe type is wholly surgical, for it is cured in the majority of instances by abdominal section and drainage of the pancreatic and gall-ducts. Robson gives the mortality of operations in chronic pancreatitis as 12.9 per cent. in 62 cases. Operations in all cases of jaundice are of a grave character, because persistent oozing hemorrhage is prone to follow. Robson and Moynihan are firmly convinced that the danger from oozing hemorrhage after operation in cases of jaundice without pancreatic lesions is less than in those cases in which the pancreas is affected. They strongly advise the use of full doses of calcium chloride in all these cases prior to operation, to increase coagulability of the blood. They advise 20 to 60 grain doses three times a day for one or two days. It is not to be forgotten that the persistent use of this salt finally decreases the coagulability of the blood.

To sum up the subject of treatment, it may be said that all forms of pancreatitis should be operated upon if the condition is such than an operative procedure can be supported. While in acute pancreatitis the operation can do little good directly, it permits drainage and it may remove some provoking cause, and so lead to recovery. In any case it is the patient's only chance. In the hemorrhagic or suppurative cases, the relief of pressure or of pus is, of course, advantageous.

PANCREATIC CALCULUS.

Pancreatic calculus is an exceedingly rare condition. The stones are composed of phosphates and carbonates. They vary in size from two and a half inches in length to fine sand, and they may be single or multiple. As many as 300 have been reported in one case.

Symptoms.—There are no pathognomonic signs of the presence of pancreatic calculus. Pain in the upper zone of the abdomen near the middle line or, as in Minnich's case, near the left costal border may be present. It is often colicky in character, and with the pain there may be vomiting, sweating, and collapse, as in gallstone colic. Fatty stools from the absence of pancreatic juice may be present, and glycosuria may occur. Occasionally pieces of the stone or small stones can be found in the stools.

Treatment.—The treatment consists in removal of the stones from the duct by operation. Hypodermic injections of pilocarpine have been advised to increase the flow of the pancreatic fluid, but this, of course, cannot remove the stone if it is embedded.

PANCREATIC CYSTS.

These occur in four forms, namely, as retention cysts, proliferation cysts, hydatid cysts, and pseudocysts. Such cysts are, however, exceedingly rare. Hale White states that in 6000 autopsies at Guy's Hospital cysts of the pancreas were found only 4 times.

Retention cysts arise from blocking of the flow of secretion by calculi or by a single calculus, by the formation of cicatricial tissue, which narrows the duct, and by pressure from neighboring morbid growths. If no infection accompanies the retention of the fluid a cyst results instead of pancreatitis.

Proliferation cysts occur as cystadenoma, or multilocular tumors, with a lining of cylindrical epithelium, and as cystic epithelioma. Hydatid cysts of the pancreas are very rare indeed, particularly in the United States and England; Hale White has reported one in England.

Hemorrhagic cyst results from the occurrence of a hemorrhage into the gland tissue. Its existence is doubted by many pathologists.

Pseudocysts are not real cysts of the pancreas, but small cystic accumulations of fluid in adjacent tissues. Sometimes a pancreatic pseudo-cyst is due to effusion into the lesser peritoneal cavity. They are thought to follow some injury to the epigastrium.

Pancreatic cysts occur at all ages from infancy to old age, and are met with more frequently in men than in women, but the difference in the two sexes is very slight. The fluid varies very greatly in appearance. It may be as clear as water, or it may be opaque, or yellow, or coffee-colored, and even green or black. Its specific gravity is from 1.010 to 1.020, and it contains albumin. Sometimes it contains all the digestive ferments of the pancreas. The presence of any of them is of value in diagnosis, but their absence does not negative the pancreatic origin of the fluid. Nor does their presence prove that the cysts arose from the pancreas. Pressure upon the pancreas and secondary changes in a cyst wall may cause communication with the pancreas and admit its secretion to the cyst cavity.

Symptoms.—The symptoms of pancreatic cyst are dependent upon the pressure which is produced, and, therefore, more or less discomfort may be the only sign of its existence. If the pressure is great pain is present. If the cyst be large enough to palpate it will be found to fluctuate, to be flat on percussion, and to transmit an impulse as in ordinary *ballottement*. Puncture with a fine aspirating needle may reveal the character of the fluid.

Diagnosis.—Pancreatic cysts must be separated from cystic kidney, horseshoe kidney, ovarian cysts, cysts of the liver, hydrops of the gall-bladder, and cysts of the suprarenal capsules. It must also be separated from mesenteric cysts, omental cysts, and retroperitoneal cysts.

Prognosis.—Pancreatic cyst may last for years without causing discomfort or death. On the other hand, cases occur in which the cyst ruptures into the peritoneal cavity or into the bowel or stomach, and when this happens death ensues, preceded by vomiting and diarrhœa. Rarely hemorrhage takes place into the cyst, and this is accompanied by sudden increase in its size, and by faintness and collapse.

Treatment.—The treatment consists in aspiration, evacuation and drainage, or complete extirpation of the cyst wall. The second procedure is usually that of election

PANCREATIC TUMORS.

These growths are exceedingly rare. Park states that in 53,000 autopsies only 226 showed primary malignant disease of this gland. Tumors of the pancreas consist in carcinoma, sarcoma, adenoma, and gumma. The infrequency of carcinoma is shown by the fact that in 23,581 autopsies made in various parts of the world, in only 29 instances was pancreatic carcinoma present. The relative frequency of these growths is scirrhus, encephaloid and colloid.

Carcinoma is the most frequent primary growth, according to Osler, and sarcoma is a more common secondary tumor because of involvement of the retroperitoneal glands by this disease.

As nearly all cases of pancreatic malignant growth are secondary to disease of the gall-bladder, a tumor in the region of that viscus will usually be found to confirm the diagnosis.

The prognosis in the case of malignant tumors is, of course, hopeless. In benign tumors the outlook depends upon the pressure symptoms. If a gumma is present, antisyphilitic treatment is, of course, indicated.

HEMORRHAGES INTO THE PANCREAS.

This condition is to be distinctly separated in the physician's mind from "acute hemorrhagic pancreatitis." Local hemorrhages may take place into the pancreas without any injury being received and without the patient being a sufferer from hemorrhages elsewhere. The hemorrhage may occur in a person who has seemingly been in excellent health without any other symptoms than collapse, with a feeble pulse and evidences of shock. Sometimes hemorrhage into the pancreas occurs as the result of aneurysm of nearby vessels, or of cancer of the head of the gland. When hemorrhage into the pancreas occurs, it may be a limited extravasation of blood into the gland, from which the patient may recover, or it may be so profuse as to flood the retroperitoneal space, extending back to the kidneys and up to the posterior insertion of the diaphragm.

The differentiation between the symptoms produced by hemorrhage into the pancreas and acute pancreatitis is practically impossible if there is no previous history of hepatic and duodenal disorder. If the condition is due to hemorrhage, an operation to relieve local tension is indicated, as death is due rather to this cause than to the loss of blood.

DISEASES OF THE KIDNEYS.

MALFORMATIONS OF THE KIDNEY.

It happens not very rarely that one kidney is absent, its place being occupied by a little fibrous tissue or by atrophied renal tissue and fat. In order that the blood may be relieved of effete materials the other kidney is often very large, and, as it is usually lower down in the loin for this reason, it may be mistaken for a dislocated or cystic kidney, and removed, which means death to the patient. Not uncommonly one kidney is larger than the other, even if both are functionally active. In other cases one kidney has two pelves and two ureters. Perhaps the most common of these malformations is the so-called "horseshoe kidney," in which the two kidneys are joined across the vertebral column by a mass of renal or connective tissue. Sometimes this horseshoe kidney is displaced into the pelvis, or it may be altogether on one side of the vertebral column.

Again, the kidneys may be fused into a single mass, usually occupying a median position much below the normal level, or even in the pelvis.

MOVABLE KIDNEY.

Definition.—Aside from the renal malpositions incident to malformation, and, therefore, essentially of congenital origin, the kidney may wander from its normal position. Several forms of such displacement are recognized.

The movable kidney lies behind the peritoneum, and usually can be made to assume its normal position, but descends during the erect posture; its malposition may be parallel to the axis of the body, or it may show a lateral movement, or the two may be combined—the "cinder-sifting" kidney. The organ may be pushed from its place and anchored in its new position—the "incarcerated kidney."

When the kidney falls forward and develops a pedicle, consisting of the ureter, vessels, and peritoneum, the last also covering the organ, it then becomes a "*floating kidney*," the pedicle forming the so-called mesonephron.

Kidneys enlarged from any cause are prone to displacement, and displaced kidneys, on account of obstruction of the ureters and veins, are frequently subject to enlargement.

Movable kidney is far more frequent in women than in men, in the proportion of about 7 to 1. Again, the right kidney is the one at fault in the majority of cases, or in the proportion of about 7 to 1. Sometimes both kidneys are movable.

Etiology.—The most important etiological factor is undoubtedly *bodily configuration.* Women of the lean, lank type with floating ribs that slope markedly downward and who are hollow in the flank, like a hunting dog in training, are the most frequent subjects. Their muscles are usually poorly developed, and, if they have been pregnant, as a rule, the abdominal wall is relaxed. If to the natural configuration and these other causes is added the effect of a tight corset and a sudden effort or fall, the needful etiological factors are all present, and a sharp pain in the side may be the sign that the kidney has made its maiden movement from its natural site. In one case the woman says she has had a fall and has wrenched her side in falling; in another she has reached high above her head, as in playing tennis; and in a third case a blow or jar in a railway accident may have been the needed trauma.

Symptoms.—Many patients have no symptoms of this condition for years. If on examining the belly in the region of the gall-bladder a floating kidney is found in a person ignorant of its existence, and anything is said of it, the patient not rarely becomes a hypochondriac on this subject, and goes from physician to surgeon, insisting on relief of symptoms which are often not really in existence. As the condition is, to a large extent, harmless, it should be ignored, unless it is causing symptoms.

When symptoms are present they vary over a wide degree of severity. In some they consist of a sensation of *dragging* in the back and side, in others they amount to *keen suffering*, and in still others they may consist in attacks of *agony* due to twisting or angulation of the ureter by the kidney becoming rotated on its axis or bent by great displacement. These paroxysms of pain have been called *"Dietl's crises."* *Nausea, vomiting, chills,* and *collapse* are present, and after the attack the urine is found loaded with urates and perhaps pus and blood cells. Sometimes even free *hæmaturia* may be present. When by postural change or manipulation the kidney assumes its normal position, a distinct renal pelvis may suddenly be emptied and a gush of urine overdistends the bladder and escapes by the urethra.

Diagnosis.—There are several states that resemble the pain of floating kidney. Violent pain in the epigastrium may be due to this cause or to appendicitis, or gastric ulcer. I have seen a *"Dietl's crisis"* closely resemble a severe attack of acute appendicitis. It also may resemble gallstone or renal colic, or the symptoms produced by abdominal aneurysm. The presence of the mass in the abdominal cavity may lead to a diagnosis of malignant growth or of impacted feces.

The dislocated kidney may be felt on careful palpation in thin women just below the border of the ribs and back of the area in which the tip of the gall-bladder can be felt when that viscus is enlarged, but gentle pressure often causes it to disappear. A floating kidney may, however, be found almost anywhere in the abdominal cavity, and has even been discovered in the pelvis. When found in the belly it is usually so movable as to be easily pushed about, and it is so free and slippery that it is often elusive, being found one moment and lost the next. Not rarely some movement of the patient may cause it to slip into its normal position. This fact often leads the physician to overlook its presence, and only when the patient is repeatedly examined is the kidney found "away from home." In other words, inability to find a floating kidney at one examination does not prove that the patient is not a sufferer from this state.

Pressure upon the kidney gives rise to a sickening sensation, which causes the patient to wince.

To examine the patient properly the woman should be placed on her back with the knees drawn up so that the belly wall is relaxed. If the right side is being examined the left hand of the physician is so placed that the tissues of the right side can be grasped close to the last rib, between the thumb in front and the fingers behind. The patient is then told to take a deep breath, when the kidney may be felt to slip out between the fingers and thumb, as a watermelon seed slips when pressed upon in this way. The left hand remaining *in situ* to block the pathway of return, the fingers of the right hand can now feel the smooth, rounded surface of the organ below the area grasped by the left hand. If the pressure by the left hand be relaxed and pressure upward is produced by the right hand, the kidney may be felt to slip back into its place. While the kidney is wandering deep palpation in the flank may reveal a lack of resistance due to absence of the kidney. When the left kidney is examined the same process is followed, save that the right hand takes the place of the left.

Treatment.—No treatment is needed unless the kidney really causes pain. If it does it can usually be kept in place by the avoidance of tight lacing and of severe exercise, or by wearing a properly adjusted pad and bandage to support the tissues just below the floating ribs. Operative interference, with the object of stitching the kidney in place, is needed in bad cases, but the difficulty is that the relief obtained by operation is not permanent in all cases, the kidney wandering away from where it is sutured. Further than this, the operation is not entirely devoid of danger. My colleague, Dr. Keen, has placed the mortality at 2 to 3 per cent. Out of 137 cases which were operated upon and collected by Watson there were 5 deaths, but 4 were not the result of the operation. In neurasthenic patients operation should be avoided, at least until a rest cure is instituted, when not rarely all signs of renal tenderness may disappear. Even in those cases which have had one attack of severe pain (Dietl's crisis) the kidney may give no further trouble if a belt and pad are worn.

CIRCULATORY DISTURBANCES IN THE KIDNEY.

Changes in the circulation in the kidneys produce very great alterations in the urinary flow, both as to its quantity and quality. If the vessels of the kidney, and particularly those of the Malpighian tufts, are poorly supplied with blood, the urinary secretion is scanty, even though the amount of solids in the urine may be fairly large. On the other hand, any cause which sends a large amount of rapidly flowing blood to the kidneys results in free diuresis. Thus, chilling the surface of the body often causes a profuse urinary flow, and nitroglycerin, by dilating the renal vessels, may do likewise.

Any substance which acts deleteriously upon the delicate cells of the Malpighian tufts causes them to permit not only the transudation of fluid, but of albumin as well.

Acute Hyperæmia.—An acute hyperæmia of the kidneys may follow exposure to cold, or the ingestion and attempted elimination of irritating substances, such as cantharides, turpentine, and a host of other drugs capable of damaging the kidney epithelium. It is also generally accepted that a similar state of these organs may exist in the course of acute infectious diseases, like scarlet fever, but it is exceedingly doubtful if this is the case even in a modified form.

Treatment.—The treatment of this state consists in the use of a few dry cups on the back over the kidneys and in the liberal administration of some water, like Poland water, to flush these organs. Rest in bed is, of course, essential. An old-fashioned and useful remedy is watermelon-seed tea or the infusion of flaxseed. Often the use of an alkaline diuretic, such as 2 drachms of liquor potassii citratis, with 1 drachm of sweet spirit of nitre, is valuable, if given every three hours.

Chronic Hyperæmia.—Chronic hyperæmia is a very much more common condition, and occurs in nearly every case in which the circulation becomes sluggish by reason of cardiac weakness, as in valvular disease with rupture of compensation, in cases of ascites with pressure on the kidneys, and when tumors produce a mechanical interference with the flow of blood in these organs.

Symptoms.—Albuminuria is a prominent symptom of this state, and hyaline casts and tube casts containing blood cells may be found. The amount of urine passed in each twenty-four hours is often very scanty. If such kidneys are seen at autopsy, they will be found to be cyanotic in the early stages, somewhat enlarged and heavier than normal, but the capsule is not adherent and the surface of the kidney is not roughened. Later the connective tissue increases, and when the kidney is incised it is found to be firm and tough, due to overgrowth of interstitial tissue. The pyramids are dark and purple in appearance. A more minute examination will reveal the fact that the capillaries forming the Malpighian tufts are greatly engorged, their walls thickened, and that the vasa recta, the interlobular veins, and the stellate veins of the cortex are also in a similar state.

Diagnosis.—Such kidneys may lead the physician to a diagnosis of nephritis complicating cardiac disease, but the renal symptoms often disappear entirely under rest and proper cardiac treatment, although developed fibroid changes in the organ are, of course, irremediable.

Treatment.—The treatment consists in giving the patient digitalis in full doses if the heart is feeble, and in applying a hot compress over the kidneys; or in the use of dry cups over these organs, and in the employment of gin and digitalis as an alcoholic stimulant to the heart and kidneys. Rest in bed for the tired heart and for the congested kidneys is essential. Another very useful remedy is fluid extract of apocynum cannabinum in the dose of 5 to 10 minims twice or thrice a day. (See Valvular Disease of the Heart.)

ACUTE BRIGHT'S DISEASE.

Definition.—By acute Bright's disease is meant a state in which the tissues of the kidney are involved in an acute inflammatory process, that is to say, it is an acute diffuse nephritis. The condition is not far removed from that of acute hyperæmia already described.

Etiology.—The causes of acute diffuse nephritis are the acute infectious diseases, particularly scarlet fever, and also diphtheria, croupous pneumonia, and septicæmia. Sometimes typhoid fever or malaria act as exciting causes. In other cases an acute infection gains access to the body through the tonsils, and it is remarkable how often evidences of renal irritation follow the onset of tonsillitis. Still another cause is exposure to cold, particularly if the circulation is disturbed by violent exercise, and if the kidneys are irritated by the process of eliminating alcohol and other waste products after a Bacchanalian revel. So, too, the ingestion and elimination of irritant poisons may cause it. Severe burns or scalds may also produce acute nephritis.

Acute and subacute nephritis are exceedingly rare in children, unless produced by one of the acute infections, and by this term is included not only the eruptive fevers, but infections such as bronchopneumonia and the various forms of severe summer diarrhœa, in all of which nephritis is by no means unusual. Thus, in 70 cases of gastrointestinal disorder Morse found signs of renal inflammation in no less than 15 per cent., and Holt states that in every case in which these conditions become severe the kidneys suffer.

Pathology and Morbid Anatomy.—As with all acute inflammatory processes, great variations as to the severity of the alterations in the kidney are met with in different cases. In mild cases the organ may show no gross changes. The essential point to be remembered is that the texture of the kidney is inflamed through and through, although not uniformly so. The glomeruli, the tubules, the connective tissue, and the bloodvessels all share in the process. In typical cases, for this reason, the kidney is more or less congested, enlarged and œdematous, pitting on pressure, and if it is cut it oozes free blood in excess. The capsule strips readily and is often less firmly attached than normal. As a rule, the more œdematous the organ the less adherent is the capsule. Often the pyramids are red and engorged, while the broadened cortex is comparatively pallid.

In some instances (glomerular nephritis) the glomeruli appear to bear the brunt of the process. Under the microscope the vessels forming the tuft are seen to be distended and contain leukocytes and larger cells having large nuclei, which are probably endothelial in origin. Degeneration of these cells ensues, and as this change progresses they are shed into Bowman's capsule with large mononuclear leukocytes, which fill it so completely that the vessels of the tuft are compressed. The epithelium lining the capsule may escape, although it usually undergoes proliferation, degeneration, and exfoliation. Added to these different sets of cells we find an albuminous exudate, due to the acute inflammatory state of the surrounding tissues, mixed with both red and white blood cells.

In the tubules there is also degeneration and necrosis of the epithelium lining their walls, so that the cells become albuminous or fatty, and desquamate. In this way the tubules are more or less distended, not only with these cells, but with red and white cells and granular detritus. Fusion of these materials results in the formation of casts of the tubules, which appear in the urine as blood casts, and as casts of desquamated epithelium.

As with inflammations elsewhere, there is an extravasation of fluid into the tissues, and when this occurs the interstitial portions of the kidney become œdematous, filled with outwandering leukocytes, and, in addition, a considerable number of small spheroidal cells, many of which, in some cases, as shown by Councilman, are of the plasma-cell type. If the inflammation is very severe, there is an actual hemorrhage into the tissues—"hemorrhagic nephritis." When the extravasation of serum and leukocytes is particularly copious, Delafield calls the condition "exudative nephritis." This is the form prone to occur during or after scarlet fever.

Symptoms.—The symptoms of acute diffuse nephritis are usually rather sudden in onset. A child convalescing from scarlet fever, or a man suffering from an acute infection, or after exposure, suddenly suffers from *scanty urinary flow*, and almost before the scantiness is noticed the *face* may be seen to be *œdematous* and the *ankles swollen*. The patient may go to sleep with a normal visage and awaken with a puffy one, the puffiness being particularly marked on the pendent side. In children, if the nephritic irritation be severe, a *convulsion* may develop as one of the early signs. *Fever* may or may not be present.

It is an interesting fact that severe *anæmia* develops with extraordinary speed, so that the swollen face becomes pallid and white in appearance as soon as it is puffy. *Nausea* and *vomiting* are often early symptoms, and are to be regarded with some alarm, as they are indicative of toxæmia.

The *scanty urine* is heavily loaded with solids, and if examined microscopically it shows red blood cells, epithelial cells from the uriniferous tubules, and tube casts composed of blood cells, epithelial cells, and hyaline material. When the heat and nitric acid test is applied to the urine, the amount of *albumin* present is found to be very large, forming a thick and heavy, curd-like mass, which, in the heat test, gradually settles to the bottom of the tube. The *pulse* is usually hard and the tension high, but sometimes the high tension is more apparent than real, the pulse being full, but gaseous in resistance.

Auscultation will reveal, in the cases which have a high arterial tension, an accentuated aortic second sound, and a comparatively feeble first sound at the apex. Sometimes *acute cardiac dilatation* develops, and secondary *pulmonary congestion* aids in the destruction of the patient. In other cases in which œdema is particularly marked a rapid *effusion of fluid* takes place into the *pleural spaces* and into the peritoneal cavity, and *pulmonary œdema* develops with remarkable rapidity. The only vessels which do not leak freely are those of the skin, the kidneys, and the bowels. The *skin* is *dry* and *harsh*, the *urine scanty* or *suppressed*, and the *bowels* are usually *constipated*.

These are the symptoms of what may be called a severe attack of the disease, but it is important to bear in mind that very often no œdema is present, and that in others the urinary flow is not greatly diminished. Often anæmia and pallor may be the first sign during convalescence from an acute infectious malady, to show that all is not well with the kidneys. In other cases some giddiness may be present, and, if the patient is an adult, uræmic symptoms are more prone to develop than if he is a child.

The urine of persons suffering from the infectious diseases should be examined repeatedly, in order that the earliest signs of renal involvement may be recognized. There is no excuse for letting the condition run on unrecognized until it is forced upon the physician by marked objective symptoms.

Diagnosis.—It is evident that this state does not present signs or symptoms which are often presented by other maladies, but, on the other hand, it is important for the physician to recall the fact that certain prominent symptoms may be absent without casting doubt on the diagnosis. Thus the amount of albumin in the urine may be small, the degree of œdema slight, and the urinary flow may not be greatly decreased. Again, the mere presence of large amounts of albumin is not alone indicative, because, as already pointed out in the article on circulatory disorders of the kidneys, it often happens that scanty urine and a large amount of albumin are present in renal congestion.

Prognosis.—The outlook in cases of acute diffuse nephritis, if the patient has previously had healthy kidneys, is favorable, particularly if his habits of life have been satisfactory. This statement holds true of the acute condition following exposure in young adults rather than in children and in young adults who have nephritis from acute infectious maladies, particularly scarlet fever. The younger the child the more grave the danger in scarlet fever. (See Scarlet Fever.) Evil symptoms are drowsiness from toxæmia, a tendency to pulmonary œdema, a feeble heart, and a free transudation into the subcutaneous tissues. Suppression of urine is, of course, a most serious symptom.

The renal lesions in those who survive the acute stage of the inflammation vary greatly in their persistency. In some cases all signs of renal trouble clear up in a fortnight, but in others albuminuria in some degree persists for months and returns whenever the patient is chilled or takes excessive exercise. When anæmia is persistent and resists fresh air and tonics, even if the albuminuria is scanty, suspicion of subacute or chronic lesions following the acute stage is aroused. In other cases the acute nephritis is but an exacerbation of a hitherto unrecognized chronic parenchymatous nephritis, in which case the outlook is most grave. Sometimes, just as the most encouraging progress is being made, a terminal pneumonia occurs, and death ensues.

Treatment.—The treatment of acute diffuse nephritis consists in putting the patient to bed at absolute rest and in the ordering of a liquid diet containing nothing which can irritate the kidneys, such as condiments like pepper or mustard. Counterirritation over the kidneys in the shape of frequently renewed hot compresses are of value, but, as a rule, it is unwise to add any irritating drug to these compresses, for it may be absorbed and cause renal irritation. If the pulse is quick, small doses of tincture of aconite (1 minim every one or two hours) may be given, with a teaspoonful of sweet spirit of nitre in cool water.

During convalescence tincture of the chloride of iron and tincture of nux vomica may be used as tonics and to combat anæmia. Great care should be taken that the patient is not exposed to cold and dampness, and that woollen garments are worn next the skin to protect it from being chilled, since chilling of the surface may produce secondary renal congestion.

CHRONIC BRIGHT'S DISEASE.

Definition.—The term "chronic Bright's disease" is applied to several types of slow, persistent, inflammatory process in the kidney which result in very definite alterations from the normal in these organs. Each type is also so distinct from the other in its rapidity of progress and results that it is difficult to regard them as related in any way, yet in each instance we find inflammation and degeneration forming the chief pathological change.

The two chief forms of chronic Bright's disease are called "chronic parenchymatous nephritis" and "chronic interstitial nephritis," because in the first the pathological process is chiefly concerned with the parenchyma of the organ, and in the second form the conspicuous changes are in the connective tissue.

There are also cases in which these two forms of nephritis exist simultaneously, that is, the kidneys present the changes found in both types. Indeed, these cases are much more numerous than is generally thought, because physicians, having been taught in student days that nephritis is capable of being divided into two forms, are continually trying to force cases that come to them into one of these categories, it being forgotten that while classification and division are artificial methods devised for teaching purposes, nature does not adhere to any such boundaries, but presents cases which may partake of more than one type at the same time. It is perhaps well, then, to consider that chronic Bright's disease is a chronic diffuse nephritis, although sometimes the parenchyma and sometimes the interstitial tissues suffer chiefly.

On the other hand, the clinical pictures afforded by the two classes of cases constitute adequate grounds for recognizing chronic parenchymatous nephritis as different from the chronic interstitial form. There can be no doubt that we have been laying too much stress on the results obtained from an examination of the urine in differentiating the two conditions. Sometimes a urinary examination at once settles the diagnosis, but Cabot is clearly right in his contention that the urine, in many cases, offers no information as to the exact type of change going on in the renal cortex. Differentiation, when possible, must rest upon symptoms considered with the results of urinary examination.

Etiology.—Chronic nephritis may follow acute nephritis, but this is very rarely the case. Its most common causes are alcoholism and exposure, and in the upper classes alcoholism and overfeeding, with lack of exercise. Gout and syphilis, chronic lead poisoning, and chronic digestive disorders are also causes, in all probability. While the latter are not as yet proved to be definite causes, there is good reason to believe that the continued absorption of toxic materials from the bowels for long periods of time may cause renal lesions in the effort of these organs to eliminate the noxious substances. A very important cause, in all probability very closely allied to those just named, is arteriosclerosis, it being considered that the changes in the bloodvessels are responsible for renal changes, although, on the other hand, the renal lesions are often the cause of the vascular degeneration. In many cases it is probable that the same causes produce both the arterial and the renal changes simultaneously. In some cases, indeed, it may be said in the majority, the exciting cause of the renal changes is undiscoverable, and perhaps may depend upon some congenital defect, which as yet we do not understand. This defect may be localized in the kidneys, or lie in other organs whose imperfectly performed labor results indirectly in renal disease.

Chronic parenchymatous nephritis is a disease of early and middle life, while chronic interstitial nephritis is observed in patients of more advanced years, but exceptions to this rule, of course, occur.

Frequency.—Chronic renal disease is one of the most common maladies affecting man, a large proportion of the deaths of all persons over thirty years of age being due to this cause. Not only do the mortality statistics prove the correctness of this statement, but it is now becoming a well-recognized fact that many cases which die of acute pneumonia are in reality cases of chronic nephritis, in which the pneumonia acts as a terminal infection and destroys the patient when his powers of resistance have diminished as a result of his renal state. The United States census for 1900 shows that disease of the kidneys stands sixth in the list of diseases causing death, pneumonia, tuberculosis, heart disease, diarrhœal affections, and unknown causes only leading it.

Of 41,924 medical cases treated in five large Philadelphia hospitals, 1395, or 3.32 per cent., were affected with nephritis, and of 24,624 medical cases treated in four large Philadelphia hospitals in which the form of the lesion was stated, 797, or 3.23 per cent., were affected with chronic nephritis. On the other hand, of 228,232 cases treated in the medical dispensaries of four large Philadelphia hospitals, only 1902, or 0.9 per cent., were affected with some form of nephritis. This remarkable difference in frequency in the wards and in the out-patient departments is probably due chiefly to the fact that most of the cases of renal disease presenting themselves for treatment were so ill that they became in-patients, and are so recorded.

From this point on it will be best to consider the two chief forms of chronic renal disease separately.

Chronic Parenchymatous Nephritis.—Chronic parenchymatous nephritis is sometimes called "chronic desquamative nephritis," because of the desquamation of the epithelial cells lining the tubules; "chronic tubular nephritis," because the uriniferous tubules are involved, or in certain cases, "chronic glomerulonephritis," because the glomeruli of the kidney are chiefly affected in this malady. It is also called "chronic diffuse nephritis."

Pathology and Morbid Anatomy.—In typical cases the kidney is found to be large, pale, its subcapsular veins conspicuous, and the capsule easily detached. The organ offers little resistance to incision, and inspection of the incised surfaces shows that the marked enlargement of the organ is due to broadening of the cortex. The medullary pyramids, while often lighter in color than normal, never attain the pale yellowish hue of the cortex, and may be dark from associated congestion. In this form the microscope shows the epithelium, especially that of the convoluted tubules, granular or even fatty, desquamating, and coalescing to form casts, which may readily be recognized in position, or, having been passed, these casts leave tubules which are imperfectly lined by epithelial cells. There is a less conspicuous, but fairly constant, change of a similar character involving the glomerular epithelium, and also the epithelial cells of some of the pyramidal tubes. Hemorrhages in the interstitial tissue are sometimes present. This is the form called "large white kidney."

In some cases the interstitial tissue is but slightly involved, but in others it is notably increased and irregularly distributed, with unequal contractions, giving rise to a more or less bossed or granular surface, not unlike that seen in typical interstitial nephritis. In this form (small white kidney) the organ may not be enlarged, and may be even smaller than normal. It resists incision, due to the increase in fibrous tissue, which, with the unaided eye, may be seen as grayish patches in the yellow cortex. Hemorrhages may be present, mottling the incised surface with reddish or reddish-brown areas.

Microscopically, the changes in the epithelium are similar to those seen in the large white kidney, but the increase in fibrous tissue constitutes a conspicuous difference. It has not been definitely decided whether the small white kidney is a later stage of large white kidney, an independent affection, or a form of nephritis which having been primarily interstitial has had parenchymatous disease superimposed.

The cardiovascular changes of chronic parenchymatous nephritis are similar in kind, but do not approach in degree those found in chronic interstitial nephritis. With the small white kidney, cardiac hypertrophy and slight arteriosclerotic changes are not of infrequent occurrence. There is usually an excess of fluid in the serous cavities, a varying quantity of œdema in the subcutaneous fat, and also in the lungs and meninges. Retinal hemorrhages are occasionally observed.

Symptoms.—When chronic diffuse nephritis of the "large white type" is well developed there are few clinical pictures which are so typical. The more or less *swollen visage;* the *greasy, pallid skin;* the *stupid facies,* and the *dyspnœa* on exertion strike the eye at once. Not rarely the partly buttoned waistcoat and the loosely laced shoes show that sufficient *anasarca* exists to cause the patient some discomfort.

If the pretibial tissues are pressed upon, they *pit on pressure.* Percussion may show the presence of fluid in the pleural and peritoneal cavities, although large effusions are not commonly met with in these cases.

If the *heart* is examined, its sounds are found to be altered, so that the *first sound lacks good quality,* and the *second sound* is usually *accentuated.* The *pulse* is *rapid,* and the arterial tension higher than normal. The are also causes, in all probability. While the latter are not as yet proved to be definite causes, there is good reason to believe that the continued absorption of toxic materials from the bowels for long periods of time may cause renal lesions in the effort of these organs to eliminate the noxious substances. A very important cause, in all probability very closely allied to those just named, is arteriosclerosis, it being considered that the changes in the bloodvessels are responsible for renal changes, although, on the other hand, the renal lesions are often the cause of the vascular degeneration. In many cases it is probable that the same causes produce both the arterial and the renal changes simultaneously. In some cases, indeed, it may be said in the majority, the exciting cause of the renal changes is undiscoverable, and perhaps may depend upon some congenital defect, which as yet we do not understand. This defect may be localized in the kidneys, or lie in other organs whose imperfectly performed labor results indirectly in renal disease.

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When the function of the kidneys is seriously disturbed, *apathy*, *stupor*, and finally *coma* may ensue from *uræmia*, and in some cases *convulsions* occur. These may vary from a slight twitch followed by stupor to a severe general convulsion, in which all the voluntary muscles are involved. Sometimes a *fleeting monoplegia*, *aphasia*, or *hemiplegia* comes on as the result of the uræmic poisoning and without any connection with an actual apoplexy. (See Uræmia.)

Patients suffering from chronic parenchymatous nephritis are sometimes seized by a *severe serous diarrhœa*, which is an effort at elimination on the part of the economy, and should be cautiously checked only when it becomes severe enough to be dangerous in itself.

Comparatively rarely in the course of chronic parenchymatous nephritis an *albuminuric retinitis* develops, but when the nephritis complicates pregnancy this ocular symptom is very common.

The combinations of symptoms just recited are met with in the well-developed cases which present characteristic manifestations of the disease. It is to be distinctly recollected that in many cases few or none of these signs develop until the patient is suddenly overwhelmed by the climax of his malady, and that of all the chronic and grave maladies which affect man none advance and develop as insidiously as does chronic parenchymatous nephritis in many cases. In one instance a *persistent indigestion* which fails to yield to appropriate digestive remedies is found to be due to renal disease; in another *anæmia* fails to yield to ordinary chalybeates and is found to be renal in origin; in still another a persistent failure of health without apparent cause has its origin in bad kidneys. In yet another group of cases a persistent *bronchitis* is founded on this cause. If consulting physicians could be polled, I feel confident that they would universally state that the average case of grave ill-health seen by them in consultation, when the diagnosis is obscure, is not really difficult of diagnosis if the state of the kidneys is carefully studied. It is not sufficient to examine the urine once. It should be done repeatedly before deciding that it throws no light on the case.

An important clinical fact to bear in mind is that a latent chronic parenchymatous nephritis may exist for a long time, and finally be recognized by the sudden development of uræmia due to an acute congestive nephritis coming on as a complication.

Diagnosis.—The diagnosis of chronic parenchymatous nephritis is reached by the following symptoms and tests: The face is puffy and pallid; there is often dyspnœa, even without exertion, and the second sound of the heart is usually accentuated. If the case is well developed, general anasarca may be manifest. The chief diagnostic factor, however, is the state of the urine. It is less than normal in quantity, of high specific gravity, and contains very considerable quantities of albumin. (See Albuminuria.) A microscopic examination of its sediment reveals fatty, granular, and hyaline casts, red blood cells, and large amounts of desquamated epithelium from the uriniferous tubules. The gravity of the disease is usually in direct proportion to the quantity and quality of the casts, the large, dark, granular casts being indicative of grave disease. Quantitative analysis reveals marked decrease in the total elimination of urea in each twenty-four hours.

Prognosis.—The prognosis of chronic parenchymatous nephritis is inevitably fatal, and the patient's life is rarely prolonged over a few months, when once the disease is well developed. In those cases of chronic parenchymatous nephritis characterized by early dropsy which rapidly develops into general anasarca, Senator states that the duration of life varies from a few months to a year. In those cases in which the development of dropsy is gradual and irregular, varying in degree from time to time, he says that death ensues in from one to two years after the beginning of the malady. Strümpell gives the average duration of life in chronic parenchymatous nephritis as from one-half to one and one-half years. Every clinician of experience will, however, agree with Senator in recognizing another class of cases which extend over several years, and which are characterized by mildness of symptoms. In these cases there is not much albumin in the urine, but slight headache, and little swelling of the lower extremities or of the face. Gradually these patients become worse, until they finally fall into one of the well-defined classes already described. They are probably representative of that form of the disease characterized by moderate changes in the parenchyma of the kidney with associated interstitial disease.

Treatment.—It has been generally held that diet is a very important factor in the treatment of this form of renal disease. While it is somewhat iconoclastic to say that this general belief is untrue, it is, nevertheless, a fact that a generous diet which does not strain the digestion and the eliminating organs can usually be allowed in most cases. We have before us a patient who is bound to die within a few months, and the question arises as to whether we can so regulate the diet that we will obtain results which compensate for the discomforts and unhappiness of the rigid milk diet, which is usually ordered, or one which causes the patient to regard his food with loathing and which is a constant reminder that he is ill.

Although skimmed milk is theoretically capable of maintaining nutrition, it is practically incapable, because such enormous quantities must be consumed, to provide an adult with a sufficient amount of nutriment. Again, milk lacks the quantity of iron which ordinary food contains. It presents to the patient the same quantities of calcium, magnesium, potassium, and phosphorus as is found in the ash of newborn animals, but it contains six times less iron; this lack of iron being made up in the young by the

[illegible]

[illegible] the quantity of water [illegible] Bright's disease. Since [illegible] as possible, on the ground that [illegible] and increase the labor of the [illegible] important one we doubt, but as [illegible] water-drinking increases nitrog- [illegible] the kidneys in Bright's disease are unable to fully deal with the products of normal metabolism, it would seem evident that excessive quantities of water must be harmful. On the other hand, there can certainly be no good results from depriving the patient of water to the extent of making him suffer.

The remedial measures other than diet consist in the use on each alternate day, if the heart is strong enough, of a moderate Turkish bath, in order that the skin may aid the kidneys in eliminating fluids and solids from the body. Fresh air and sunlight are essential, and severe exercise is to be prohibited.

Drugs are of little value, except to relieve symptoms which may be annoying. It has been the custom of physicians for many years to prescribe iron, usually in the form of Basham's mixture, for the purpose of combating the anæmia of chronic parenchymatous nephritis. This method of treatment does not possess the advantages with which it has been credited. The anæmia depends upon the toxæmia of the disease, and this, of course, is not removed by the administration of iron. Further than this, iron has a tendency to produce constipation, and constipation is prone to increase anæmia, and, again, constipation is a particularly undesirable condition in Bright's disease, since it prevents the bowels from aiding the kidneys in eliminating impurities.

The administration of very large doses of Basham's mixture is, therefore, unwise. It should be borne in mind that iron has no curative effect upon the renal condition, and therefore it is useless to administer more than the system can utilize for the relief of the secondary anæmia in the blood. Small doses of Basham's mixture are, therefore, as useful as large ones, so far as the anæmia is concerned. If the effect of Basham's mixture as a diuretic is desired, the liquor ammonii acetatis of the United States *Pharmacopœia* may be added to a teaspoonful of Basham's mixture and given three or four times a day, as in this way the diuretic effect is obtained without an excess of iron being given.

Should evidence of cardiac dilatation develop digitalis is indicated, and may be given in the dose of 5 or 10 minims of the tincture three times a day until some evidence of its physiological effect is obtained, when the dose should be cut down one-half. While the infusion of digitalis has the reputation of being more diuretic than the tincture, it is so much more prone to disorder the stomach that the tincture is usually preferable.

A useful formula in place of digitalis will be found on page 519.

If uræmic symptoms develop, the patient should be given a hot pack, and if there is any reason to believe that pulmonary œdema is threatened, or that the heart is too feeble to endure the hot pack, a hypodermic injection of strychnine, $\frac{1}{20}$ of a grain, should be given before the pack is begun. Sometimes a cup of strong black coffee is also useful at this time. Pilocarpine should not be employed, as it is too depressant and prone to produce pulmonary œdema.

The question as to whether the bowels should be thoroughly purged by one of the hydragogue cathartics is debatable. On the one hand, it is claimed that by this means the intestines are unloaded and a large quantity of liquid and toxic material is removed from the body, and, on the other, that the purging may cause concentration of the blood, and so increase toxæmia. Probably the best rule to follow is to administer a hydragogue cathartic only when there is reason to believe that the bowels are confined and are consequently loaded with fecal matter. Hypodermoclysis, which is so useful in the uræmia of chronic contracted kidney, is worse than useless in chronic parenchymatous nephritis, owing to the presence of œdema.

A valuable drug for the purpose of diminishing arterial tension and so decreasing the work of the heart, and also because it diminishes the loss of albumin through the kidneys, is nitroglycerin, which should be given in the dose of $\frac{1}{100}$ of a grain three or four times a day. This drug often increases the urinary flow when it is scanty.

In regard to the treatment of uræmic convulsions, it is commonly held that the administration of morphine hypodermically for this purpose is dangerous, although there are some active practitioners who believe that it is a useful drug. It is probably more dangerous in the parenchymatous than in the interstitial form of the disease. If the convulsion is severe chloroform or nitrite of amyl should be given by inhalation. (See Uræmia.)

Comparatively recently it has been suggested that cases of chronic renal disease should be treated by decapsulation of the kidney. This plan of treatment is more indicative of surgical enthusiasm than of a clear conception of the pathology of the disease. A knowledge of the pathology and the results of experiments on animals show its futility, if not its danger. These views will be found in detail in the editorial columns of the *Therapeutic Gazette* for January, 1904, and June 15, 1904.

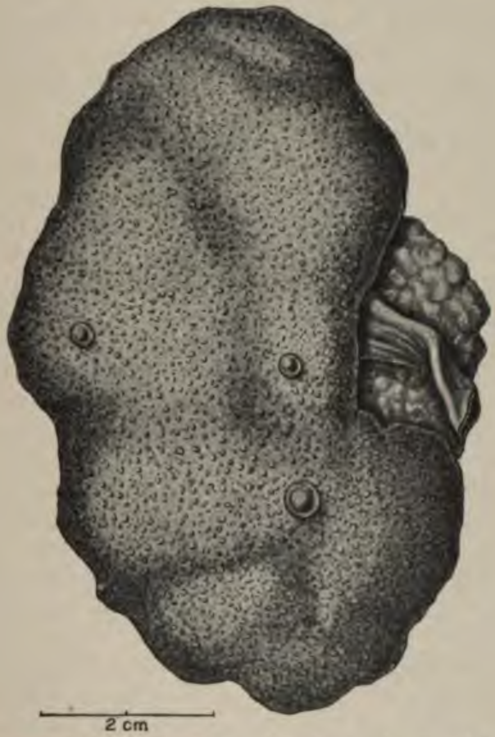
Chronic Interstitial Nephritis.—To this form of chronic renal disease the terms "contracted kidney," "granular kidney," "cirrhosis of the kidney," and "sclerotic kidney" are applied.

Pathology.—Chronic interstitial nephritis is usually a primary process, although the small white kidney is really a combination of the fibroid and the chronic parenchymatous form. In this type the overgrowth of the connective tissue of the kidney is the dominant part of the pathological process, and the degeneration of the parenchyma, as represented by the glomeruli and the tubules, plays a secondary role.

When kidneys affected by this state are examined, it is found that they contain large masses of fibrous tissue extending through the organ, which by contraction cause a shrinkage in size and a puckering of the surface. The capsule becomes thickened and exceedingly adherent, and the tissues beneath it are torn if it is stripped off. The surface of the cortex is roughened by rounded granules of varying size and cysts may appear at various points on its surface (Fig. 94). When an attempt is made to cut through the kidney, it is found to be tough and difficult to incise. After the organ is split open it is seen that the cortical portion is very much wasted.

When the renal tissues are placed under the microscope, they show an irregularly distributed increase in the connective tissue, involving in particular the cortex and the interlobular vessels. There is also an associated atrophy of the epithelium lining the uriniferous tubules. Casts are seen in the tubules, but not to the degree in which they are met with in the parenchymatous form of nephritis. The glomeruli in advanced cases may be converted into thick, fibrous bulbs; the walls of the bloodvessels forming the tuft may be thickened, and the capsule is seen to be dense and fibrous. Nor does the fibroid process affect the finer bloodvessels and the connective tissue alone. It extends to the large bloodvessels, and even to the renal arteries and veins.

Fig. 94



Kidney of chronic interstitial nephritis. The surface is granulated and irregular, and contains numerous cysts. The contraction is quite marked, the organ being but little more than half the normal size.

It is also a noteworthy fact that while cardiac hypertrophy and arteriosclerosis are often met with to some degree in parenchymatous nephritis, they are constantly found in a well-developed degree in the interstitial type of the disease. This cardiac hypertrophy is not limited to the left ventricle, as it is in the parenchymatous form. It affects the whole heart and it is often very great. The cause for the hypertrophy has been the subject of much debate, but the conditions present in the bloodvessels seem to offer an adequate explanation of the state. These vessels are always fibroid and lacking in normal elasticity, and this, of course, offers greater resistance to the flow of blood through them and into the capillary networks. As the process is a gradual one, there is a gradual increase in the demands made upon the heart, and this is met by an increasing hypertrophy. The difficulty in forcing blood through the renal vessels also aids in producing this effect, but such influence has no doubt been overestimated.

This is not the place to dilate upon the relationship of these vascular changes to the renal lesions. Many persons think that the renal changes are the cause of the vascular lesions, and others hold that the vascular degeneration causes the renal state. A most striking illustration of the relation between arterial disease and contracted kidney was shown by Welch at the meeting of the American Medical Association in 1904. He presented a specimen which consisted of a kidney supplied by two renal arteries, one of which was sclerotic. The area of the kidney supplied by this vessel was typically fibroid, while the other pole of the organ nourished by the uninvolved trunk was but slightly changed.

Again, it is held by many that the overgrowth of connective tissue takes place to fill gaps made by the atrophy of the parenchyma, and by others the view is taken that the overgrowth of the connective tissue destroys the parenchyma by pressure. The latter view seems the more probable, but the former opinion is adhered to by many pathologists, who believe the primary change is in the parenchyma.

Symptoms.—This is the type of renal disease which is found in the iron-master or stock broker who boasts that he has never had a sick day in his life, and who begins to find himself, at forty or fifty, lacking in initiative, and who suffers from vertigo or dizziness, which he thinks are due to a club dinner or a strong cigar. This is the disease of the hard-working, "high-nervous-tension" individual who has lived hurriedly, and perhaps quieted himself between periods of great exertion by a drink or two of whiskey, repeated it may be many times. Often it develops in those who have not used alcohol, but given a man who takes little exercise, some whiskey, and who is managing one or more large business interests, and he is the individual who is paving the way for or has already developed chronic contracted kidney. Very rarely, indeed, the disease develops in early life, but cases have been reported by a number of clinicians in children as young as from two to seven years. Most of the instances have occurred about puberty. Sutherland and Walker have reported 2 cases, aged eight and sixteen months, respectively, who had contracted kidney due to congenital syphilis.

The symptoms of contracted kidney are, in a large proportion of cases, absent until the disease progresses so far that grave secondary changes take place. Indeed, it not rarely happens that the patient continues in what he considers good health until an acute attack of *uræmia* or *cardiac failure* sends him to the hands of the physician, who may be misled into the diagnosis of acute syncope, due to overexertion, by the fact that the heart seems to be feeble, and because the urine shows little or no albumin. The number of diagnoses that have been wrecked upon the shoal of "no albumin" is a multitude, because it is a peculiarity of chronic contracted kidney that albumin is often absent for brief periods, or present in such minute amounts that it is overlooked. It is only when the heart begins to fail so that some congestion of the kidney develops that the albumin becomes more copious. The *albuminuria* of contracted kidney is, as a rule, as scanty as it is profuse in the parenchymatous form. Again, the scanty urine of the parenchymatous type is replaced by a *profuse flow* in the contracted type, and the patient in consequence is disturbed in his sleep by having to get up at night to empty the bladder. The specific gravity of the urine is low, about 1.005 to 1.012, and it is clear and lacks color. If the urea is estimated this ingredient is usually much reduced, not only relatively, but actually, so that the patient in many cases does not excrete half the normal output per day. Casts may not be found and rarely are abundant, and often the centrifuge has to be employed to reveal them. Further, these casts are readily overlooked, for they are chiefly hyaline and so transparent that if careful focusing and illumination are not resorted to they are not seen. In some cases granular casts are only periodically demonstrable.

The *circulatory symptoms* of contracted kidney are as important in reaching a diagnosis as the renal signs. The *pulse* is *hard* and *tense*, and so *high* is the *blood pressure* that it may be almost impossible to occlude the vessel by pressing upon it. If the radial artery is rolled under the fingers, it feels like a piece of thick rubber tubing, and it is easily recognized as being distinctly fibrous when it is palpated carefully. In other words, the blood tension is high and the vessel is thick. If the heart is examined, there is found, as a very constant symptom, a *sharply accentuated aortic second sound* at the second right costal cartilage, which is due to the high arterial tension. On inspection the *apex of the heart* is found to be displaced downward and outward because of the *cardiac hypertrophy*. At the apex a more or less distinct systolic murmur is heard in many cases, due, as a rule, to stretching of the mitral orifice under the stress of high pressure in the ventricle, resulting from great arterial tension. When compensation fails, either because the heart becomes exhausted or because of fibroid or other myocardial degeneration, these symptoms may be replaced by weak heart sounds and by a feeble pulse. It is only while the heart has vigor that high tension can exist.

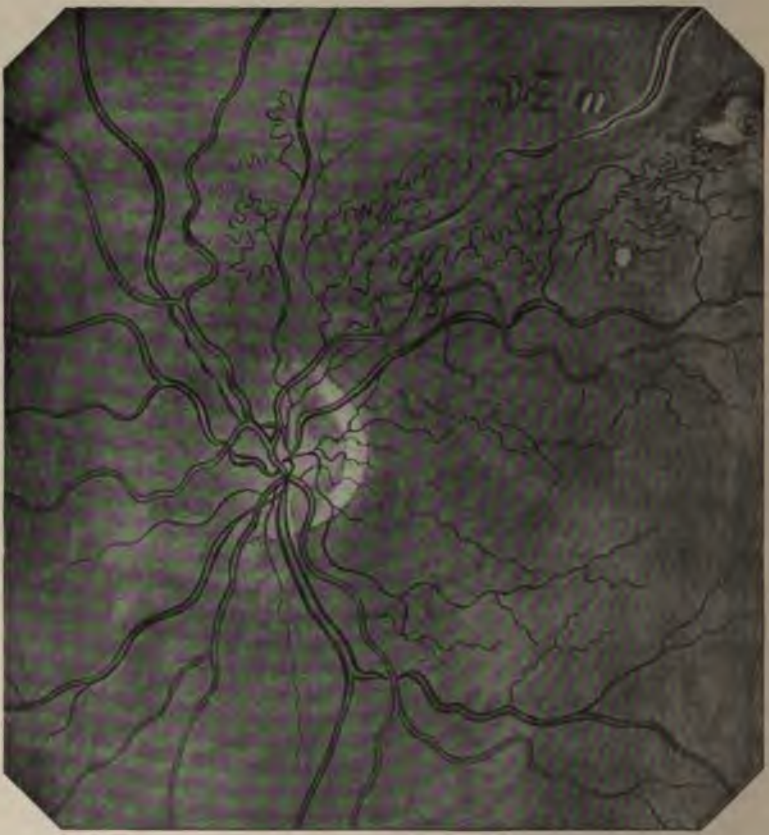
The *respiratory system* does not offer much that is characteristic, but complicating lesions often develop in these parts. One of the most common is *pneumonia*, which finds a fair field for its development in all cases of renal disease. Indeed, in every case of acute pneumonia the physician should study the renal condition. Often the routine examination of the vessels and of the urine in a case of pneumonia is the first evidence that chronic contracted kidney is present. Perhaps the most common respiratory manifestation is *difficult breathing* resembling asthma, which, coming on in persons not previously asthmatic, should always raise the suspicion of renal disease. *Effusions* into the *lung* or *pleural spaces* may occur with suddenness and cause death. When the toxæmia is well marked, Cheyne-Stokes breathing may develop.

The *cerebral symptoms* consist in *vertiginous attacks*, *migraine-like seizures*, and *persistent*, *dull*, or *throbbing headache*. *Apoplexy* due to the degenerative arterial changes may take place.

It is a most interesting fact that *œdema is as rare* in contracted kidney as it is common in the large white kidney. When it occurs it is not renal in origin, but due to the failure of the heart. The skin in this type of renal disease is usually dry, and is chalky in hue.

Next in importance to the examinations of the urine and the study of the peripheral circulation in these cases is the observation of the *state of the retinal vessels.* They very commonly reveal the renal condition.

FIG. 95

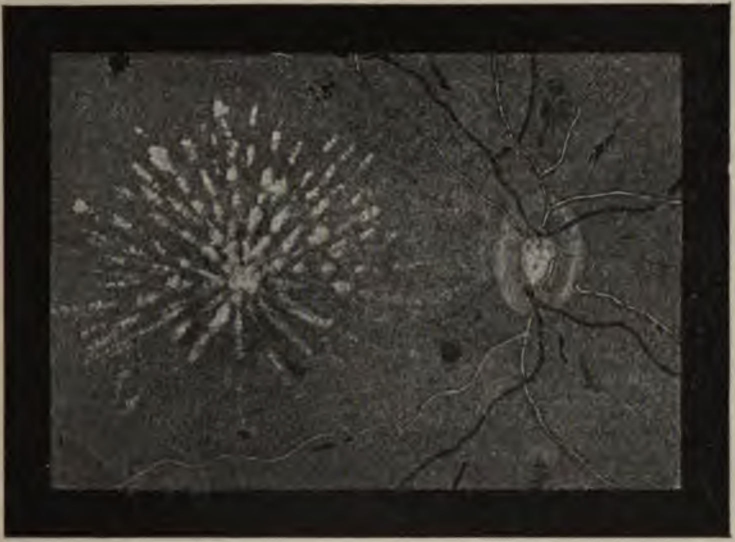


Retina showing Hirschberg's vessels. The high tension in the arteries can be seen to be narrowing the veins by pressure. (de Schweinitz.)

Different observers give varying percentages of occurrence of retinal lesions. Out of 935 cases of renal disease, Groenouw found retinal lesions in 209, or 22.4 per cent. The age at which they most frequently are met with is from fifty to sixty years, but they have been seen in adolescents.

Five types of these lesions are recognized by ophthalmologists: (*a*) *typical albuminuric retinitis;* (*b*) *degenerative albuminuric retinitis;* (*c*) *hemorrhagic albuminuric retinitis;* (*d*) *albuminuric neuroretinitis,* and (*e*) *albuminuric* *papillitis.* In the first form *irregularly shaped white dots* or spots appear in and *around the macula*, and may take a stellate form. When the condition is well developed a zone of whitish-yellow may surround the head of the optic nerve. *Flame-like hemorrhages* may also appear. The condition is at first one of hyperæmia, then of degeneration, and finally one of atrophy. In the second form the *white spots* are *small*, and *hemorrhages* are more *limited*, and the white zone about the nerve head is not well developed. The third form, as its name indicates, is *chiefly hemorrhagic* in type, and the *hemorrhages* are *large* or *profuse*, while the other changes are insignificant. Only when the hemorrhages are absorbed do these areas become whitish. The fifth form shows that the process has been confined to the optic nerve, so that a *papillitis* or *choked disk* is present, the retina being but little involved.

FIG. 96



Albuminuric retinitis. Granular kidney. Note hard-edged "asterisk" exudation and the silver-wire condition of the arteries, and the punctate and linear hemorrhages.

In some cases *detachment of the retina* or *hemorrhagic glaucoma* develop as complications.

Of even greater importance than the states just described in the early diagnosis of renal and cardiovascular disease is the tortuosity of the retinal veins and their narrowing by the pressure of the retinal arteries wherever these vessels cross the veins—the so-called "Hirschberg's vessels" (Fig. 95).

Because of the fact that the symptoms of chronic contracted kidney are so often insidious in their development, the ophthalmologist is often the first to recognize the existence of the disease, because a man who considers himself in perfect health asks for glasses for failing vision or seeks relief for blindness in one eye. Not rarely these patients have repeated attacks of rupture of subconjunctival vessels, as well as hemorrhages into the retina.

Flexner states that the terminal dysentery of Bright's disease is often due to the *Bacillus dysenteriæ*.

Prognosis.—Here, again, chronic contracted kidney presents a widely different aspect from that of the parenchymatous form, for, while in the latter lesion death, as a rule, occurs inside of eighteen months at best, these cases often live for many years, if the process is not already far advanced when the case is first seen. The points governing prognosis are the state of the heart and the vessels, the ability of the kidneys to approximate the normal daily task, and the life which the patient can or will lead. It is manifest, from what has been said as to its pathology, that the affection is incurable, but patients often live as long as ten or fifteen years after undoubted signs of the malady are present. The development of signs of uræmia, of feeble heart, or of pulmonary congestion, œdema, or pneumonia is, of course, alarming.

By far the most important factor in determining the probable duration of life is the state of the retinal vessels already named. De Schweinitz has studied this matter most carefully from the standpoint of the ophthalmologist, and we have followed a number of cases together.

These facts are well emphasized by the following figures, which illustrate the duration of life in chronic interstitial nephritis after the occurrence of retinal changes: Belt collected 419 cases, of which 72 per cent. died within one year and 90 per cent. within two years. The cases reported from Haab's practice by Possauer showed that none of the men applying to the clinic for treatment lived more than two years; of the women, 68 per cent. died within the same period of time. Of private patients who could live comfortably, only 59 per cent. of the men and 53 per cent. of the women had died at the end of two years. Gruening collected 100 cases, none of which survived more than two years after retinal changes began, and Bull found that 69 out of 103 cases died within two years. Of the remaining 34, 17 died after a longer period, and 17 were alive at the time his paper was published. Harlan analyzed 40 cases with the following results: 33 ended fatally at various periods, averaging four months; 3 lived a year after the discovery of retinal changes; 3 recovered, and 1 regained his eyesight, although the urine was albuminous at the end of two years. Miley traced 45 cases, and found the average duration of life to be less than four months from the time eye changes were first observed. One of his patients lived eighteen months and two fourteen months, but all the others died within a year. Webster mentions the case of a clergyman suffering from chronic interstitial nephritis, in whom retinal changes had been recognized ten to fifteen years before, and "who is still living," and Wert had a woman under observation in whom retinal changes had been noticed more than four years before he reported the case. Her general condition was much the same as when she came under his charge. I have under my care at this time a man who has been under observation four years, during which retinal hemorrhages have repeatedly occurred, and whose arterial tension is astonishingly high. He is now taking ½ a grain of nitroglycerin a day with the iodides, to lower his blood pressure and relieve his heart. Such a result is most rare.

Most of these cases with very high tension and retinal changes die from apoplexy or an acute myocardial failure soon after retinal changes develop, the patient often dropping dead without warning symptoms. These extreme instances are interesting, but they are rare in the history of the malady.

Treatment.—There are few diseases of an incurable character in which the patient can be so greatly benefited by treatment.

The question of diet in cases of chronic contracted kidney is to be decided along pretty much the same lines as those which have been drawn in the article upon the treatment of chronic parenchymatous nephritis. W. Hale White has expressed the belief, in which we coincide, that, as a rule, this disease is treated too zealously, and that in the desire to spare the kidneys the patient is starved, with the result that the only means by which the degenerative process can be retarded, namely, the maintenance of general good health, is impaired.

As we do not know of any articles of ordinary diet which can be considered really harmful in granular kidney, it is best to give the patient ordinary plain digestible foods containing the normal proportions of proteids, fats, carbohydrates, and salts, just as it is necessary to give a person in health a similar mixed diet. It need hardly be stated that highly seasoned foods, or foods which are difficult of digestion, should be interdicted. Again, we are glad to note that Dr. White is in accord with us in believing that the limitation of these patients to a diet of chicken and fish without any red meat is entirely unnecessary. Not only does such a limitation do no good, but it is often harmful in the sense that it makes the patient consider himself seriously ill, and also diminishes his appetite. Patients with interstitial nephritis, however, should especially eschew all forms of alcohol, since it is imperfectly oxidized in these cases, and tends to increase arterial tension.

It is of vital importance in chronic contracted kidney that arterial tension should be kept at a point as near the normal level as possible, since by this means danger of rupture of a cerebral vessel is diminished and the work of the heart is decreased. A sufficient quantity of nitroglycerin to accomplish this result should always be administered. It is not a question of the size of the dose, but of the quantity required. It is also to be remembered that patients rapidly become accustomed to nitroglycerin, so that ascending doses are nearly always necessary. Very large doses may be taken by many of these patients without any disagreeable symptoms of the full action of the drug. Beginning with $\frac{1}{100}$ grain three times a day, I have seen as much as $\frac{1}{2}$ grain a day taken with no other than excellent results.

Another valuable drug in chronic contracted kidney, both from the standpoint of arterial tension and of the fibroid changes in the arteries, is the iodide of potassium. There can be no doubt that this salt diminishes arterial tension, and if any remedy exercises an influence for good in arresting the pathological changes in the kidneys and in the bloodvessels, that remedy is certainly one of the iodides. Usually doses of from 10 to 20 grains, three or four times a day, are quite sufficient. A useful substitute for the potassium salt is the sodium or strontium salts or the syrup of hydriodic acid given in ascending doses, beginning with 30 drops three times a day. It goes without saying that the iodides have not as powerful an influence in decreasing arterial tension as have the nitrites.

Digitalis is rarely needed in chronic interstitial nephritis, as the cardiac hypertrophy is usually adequate, and digitalis tends to raise tension in the vessels which is undesirable, but in some cases there comes a time when the blood pressure falls largely because the advancing myocardial degeneration and cardiac fatigue prevent the heart from pumping the blood with normal energy. In such cases digitalis and strophanthus do good, and nitroglycerin may do harm. (See Arteriosclerosis.)

Uræmia is to be treated by the use of the hot pack, if the patient's heart is strong; by the employment of hypodermoclysis, and if the patient is fairly full-blooded by venesection as well. (See Uræmia.) Cups may be placed over the kidneys, if there is any condition of renal congestion, and three or four cups should be placed over the base of each lung, if any signs of pulmonary œdema develop. When evidences of pulmonary difficulty arise, full hypodermic doses of strychnine are advisable, and if any tendency to urinary suppression occurs, $\frac{1}{4}$ to $\frac{1}{2}$ of a grain of cocaine may be given hypodermically every four to six hours for several doses.

Many practitioners have thought it wise to employ full doses of morphine hypodermically in the treatment of uræmic convulsions. I have recently made a collected investigation in regard to this matter, and have obtained the opinion of physicians of experience in both this country and in England. This opinion is almost universally adverse to this use of morphine.

The convulsions should be controlled by nitrite of amyl and by chloroform given by inhalation. (See Uræmia.)

The question of renal decapsulation has already been discussed under Chronic Parenchymatous Nephritis.

If the progress of the disease is slow, it is advisable to send the patient to some warm climate during the winter months, if his home is in a northern latitude. The object is to avoid rapid changes in temperature in the atmosphere and consequent chilling of the surface, with secondary congestion of the kidneys and decrease in the activity of the skin.

The best climate is to be found in the neighborhood of San Diego, California, or in Egypt.

AMYLOID DISEASE OF THE KIDNEYS.

Definition.—Amyloid disease of the kidneys is part of a general process affecting many organs. The renal manifestation is characterized by the deposit of lardacein in the subintimal stratum of the bloodvessels, in the glomeruli, and, to a lesser degree, in the connective tissue of the tubules.

Etiology.—Amyloid disease of the kidney is usually the result of a prolonged suppurative process, such as hip-joint disease, chronic pulmonary tuberculosis with cavity, or any cause whereby the system is simultaneously sapped by suppuration and poisoned by the absorption of toxic substances. It may also be a sequence of one of the prolonged fevers or occur as an associated change with chronic diffuse nephritis of the parenchymatous type. Syphilis is a common cause, and malaria may be the only demonstrable antecedent.

Pathology.—The kidneys are usually enlarged, and when incised the cut surface is shining or polished in appearance. When the condition is combined with interstitial nephritis these organs may be small. The surface of the organ is paler than normal, particularly in the cortex, but the pyramids are deep red in hue, and the glomeruli are readily seen.

If an aqueous solution of iodine is painted over the cut surface of such a kidney, the areas most affected by the amyloid change stain a deep mahogany brown, and if to these areas is applied a dilute aqueous solution of sulphuric acid, the brown hue is changed to blue.

Microscopically the kidney structure when examined reveals the fact that the capillary vessels forming the tufts in the capsules are parts of the parenchyma most affected, the tuft being transformed into a waxy mass. The disease process also involves the afferent and efferent bloodvessels of the tuft and the straight or direct vessels of the kidney. In marked cases the connective tissue of the tubules is infiltrated, the interstitial tissue increased, and the epithelial cells may be granular or fatty, as in parenchymatous nephritis. In some instances all these types occur together.

Symptoms.—There are no symptoms which, taken by themselves, may be considered indicative of amyloid disease of the kidneys. It is only when certain urinary signs develop in a case which is the subject of those maladies which predispose to amyloid change that we can say that a diagnosis is assured. The patient is usually *pallid*, may be well covered by unhealthy waxy fat, and the heart is not rarely somewhat enlarged, although no less an authority than Dickinson contradicts this view. The urine is passed more freely than is normal. It is clear and has a *low specific gravity*, about 1005 to 1010. The quantity of *albumin* which it contains varies, but it is usually present in *considerable quantity*. There is a distinct increase in the quantity of serum globulin in the urine. Under the microscope the tube casts are found to be hyaline, fatty, or waxy. Occasionally the amyloid reaction already named may be demonstrated in the urine. The degree of œdema and the state of the bloodvessels and heart depend more upon the presence of associated nephritis than upon the amyloid change itself. If nephritis is well developed, anasarca and high arterial tension may be present as an associated state.

Prognosis.—The prognosis depends upon the gravity of the causative process and the degree to which the secondary change in the kidneys has progressed. Then, too, it must be remembered that amyloid disease is not a condition limited to the kidneys, but affects such organs as the liver, spleen, and even the heart, and, therefore, the patient is peculiarly handicapped in his struggle for health. If the casts are fatty or waxy, and are present in large numbers, if the albuminuria is copious, and if anæmia is marked, the outlook is bad. Indeed, in every case it is anything but good, and the longer the suppurative process continues the worse the outlook becomes.

Treatment.—This consists in the same measures as have been recommended for chronic parenchymatous nephritis, such as iron, arsenic, cod-liver oil, and fresh air and sunlight to combat the underlying cause. If possible, the suppurative process, if such is the cause, should be removed.

URÆMIA.

Definition.—Uræmia is a condition in which as the result of faulty activity of the kidneys a patient develops a series of symptoms of which the most notable are stupor, coma, convulsions, or paralysis, or in other instances gastrointestinal disorders.

Etiology and Pathology.—The causes of uræmia are not clearly understood. It is well known, and universally recognized, that the condition is due to perverted renal activity, both as to the elimination of the ordinary products of metabolism and the effects of renal disease upon the tissues of the body, but beyond this the actual cause is as yet undetermined.

At one time, under the leadership of Traube, the idea prevailed that the symptoms of uræmia were dependent upon changes in the circulation in the brain produced by the constriction of its arteries by the vascular changes associated with nephritis or by cerebral œdema. In other words, that cerebral anæmia due to arterial constriction was the cause of the symptoms. This view has been cast aside because it has been shown that ligature of the carotid arteries does not cause uræmic symptoms, because it has been proved that the high arterial tension of renal disease results in dilatation of the cerebral vessels, since they are poorly endowed with muscular fibres, and finally because it is impossible to cause active contraction of the cerebral vessels by any drug or measure used for raising arterial tension.

The theory, that uræmia is due to the retention of the ordinary effete materials of the body owing to renal disease, also cannot be accepted as a complete explanation of the condition, because it has been found that ligation of the renal arteries in animals and ligation of the ureters fail to produce uræmia, although the function of the kidney is by these means completely arrested. Again, suppression of urinary secretion by the presence of stone in both kidneys, and even by necrosis of the cortex of both kidneys in man, does not cause typical uræmia. Further than this the injection of urea and even of healthy urine into the blood does not cause any symptoms of true uræmia. Again, in certain cases of renal disease, as in chronic contracted kidney of the aged, when the renal excretion is seriously impaired, uræmia is rare. All forms of deficient renal activity do not, therefore, cause uræmia.

A third theory is that as a result of the renal disease peculiar poisons are made in the body which when they accumulate cause uræmia, or that the condition of the system in renal disease permits certain micro-organisms to grow and produce a toxic substance.

A fourth opinion is that the kidney secretes when in health an "internal secretion" which governs metabolism and so prevents the formation of certain poisons. The last theory falls to the ground because ligation of the renal vessels does not result in uræmia, as it would do if these symptoms were caused by the lack of some internal secretion.

We are left, therefore, with the fact that uræmia is due to the presence in the body of peculiar poisons arising in Bright's disease, either as the result of the growth of micro-organisms or perverted metabolism, and with the knowledge that the kidneys are unable by reason of disease to be active in the elimination of any poisons. It would seem probable that this combination of extra poisons and deficient renal activity are the two factors necessary to the development of uræmia. This is further supported by the fact that if the labor of the kidneys is increased by gastro-intestinal fermentation or putrefaction, an attack of uræmia is very prone to occur. Finally, there is additional proof of the development of extra poisons in the body in renal disease. This is found in the marked loss of weight in patients suffering from nephritis, the wasting showing that metabolism is seriously impaired and that tissue breakdown is marked. Manifest loss of weight may not be present because of dropsy, but, if this is removed by purging, the wasting is manifest. The toxicity of the urine is increased.

Symptoms.—Uræmia occurs in several forms. The most common manifestation of uræmia is that in which the patient passes into *coma*, which may be *preceded by delirium* and *drowsiness*. In certain cases there is associated with the development of the comatose state twitchings and contractions of widely separated muscles, and particularly the extensors and flexors of the forearms.

The most startling, but by no means the most frequent, form is the *convulsive type*. In this condition the patient, with or without any preliminary indications of nervous disturbance, is seized with a more or less severe epileptoid attack, which usually involves the muscles of the face and hands, and then spreads rapidly to the whole body. No sooner is one seizure over than another comes on, and with the repetition of the attacks, or it may be with the development of the first fit, the patient becomes unconscious, or has very distinct mental impairment. The body temperature usually falls unless the convulsions are so severe as to temporarily cause a slight rise. The knee-jerks are usually markedly exaggerated and the pupils are contracted. Because these symptoms are so extraordinary, the idea has gained ground that convulsive seizures in uræmia are commonly met with. This is incorrect, as they are not common except in that peculiar form of eclamptic convulsion due to toxæmia which is encountered in pregnancy.

A third form is that in which there is marked *respiratory disorder* of a dyspnœic type. The patient finds it exceedingly difficult to breathe, and feels as if suffocated. Not only is the respiration wheezing, as it is in asthma, but it is peculiar in that it is accompanied by a hissing sound, the patient very frequently ending each expiration with a puffing hiss. Associated with these symptoms there may be some duskiness of the skin, but cyanosis is not marked. The patient's mind is usually clear, and he not infrequently complains of his great difficulty in getting sufficient air. As this condition proceeds, the respirations may become "Cheyne-Stokes" in type. Although it is generally held that the development of Cheyne-Stokes respirations under any circumstances is indicative of a fatal result, it not infrequently happens that patients with this symptom arising during the course of uræmia recover from that particular attack. Sometimes the Cheyne-Stokes breathing occurs only during sleep, and it may be the only manifestation of uræmia, the mind remaining clear.

There is still a fourth form in which the patient develops *mania* or *acute insanity*. He is restless, very excited, and may be extremely violent. As a rule, after these symptoms have lasted for a short time, the mental excitation is followed by gradually increasing drowsiness which finally passes into coma.

In the so-called *paralytic form* of uræmia, either hemiplegia or monoplegia may come on suddenly, as does hemiplegia in cases of cerebral hemorrhage. But the paralysis is not due to rupture of a bloodvessel, to a formation of a thrombus, or the plugging of a vessel by an embolus. So far as is known, it depends upon intoxication of the nervous centres controlling the parts involved in the paralysis. It is of course possible for cerebral apoplexy to complicate uræmia, and for this reason it may be difficult to immediately make a differential diagnosis between the hemiplegia of uræmia and the complicating hemiplegia of cerebral rupture.

There is still another form of uræmia which manifests itself in *persistent insomnia* with muscular irritability or cramps and hiccoughs.

In some cases of uræmia *violent gastrointestinal disorders* suddenly assert themselves, *vomiting* may be *persistent* and *severe*, and *nausea intense*. Not rarely *profuse serous purging* comes on, which may be an effort on the part of the body at elimination.

In some cases *uræmic amaurosis* develops. This consists in sudden, bilateral, and complete blindness. Rarely one eye suffers before the other, and in some cases the perception of light may be preserved, although ordinary vision is destroyed. In the greater number of these cases the ophthalmoscope reveals no changes in the retina, although it may be found to be œdematous and there may be an appearance of the optic nerve like that of choked disk. This condition develops more commonly in those cases of acute nephritis associated with the eruptive fevers, as scarlet fever, than in ordinary chronic nephritis. The amaurosis lasts for a few hours to a day or even longer than this, and vision often returns as suddenly as it was lost. The prognosis is favorable as to vision.

All of these forms of uræmia differ very materially from that type which has been called "latent uræmia" by the late Sir William Roberts, and of which mention may be found in connection with the article upon Nephrolithiasis. In these patients life is maintained for periods varying from one to two weeks in the presence of total urinary suppression. They remain conscious almost to the moment of death, and the uræmic symptoms just described in their various forms are never present. There may be some headache and nausea and weakness and drowsiness. The temperature is subnormal and the pupils are contracted. In some instances vomiting is a prominent symptom in this type of uræmia. It is unfortunate that the term "latent uræmia" should be applied to this condition, as the condition is really not one of latency nor of uræmia as that term is generally understood.

A very important symptom of uræmia is the *peculiar odor* about the patient, which is quite characteristic and which may be due in part to urea which is being eliminated by the skin or to the presence of some toxic substance as yet not isolated.

Diagnosis.—The presence of albuminuria with casts of the uriniferous tubules, of somewhat thickened bloodvessels, and of an accentuated aortic second sound in association with the development of any of the symptoms which have just been described, makes the diagnosis of uræmia practically certain. If the patient is bled for the purpose of relieving symptoms of venous engorgement, it is wise, if opportunity offers, to make a determination of the urea in the blood if the physician is sufficiently skilful to perform the necessary manipulations. The most difficult differentiation lies between uræmic monoplegia and hemiplegia due to rupture of a bloodvessel, or to an embolus, or thrombus. In some cases such a differentiation is impossible because these vascular lesions may be present as a complication of the uræmic state. The presence of the urinary changes just described and of the other signs and symptoms mentioned in the article upon Bright's Disease will serve to aid in the differentiation to some extent.

At times, if the uræmic poisoning is not of such a character as to produce convulsions, but merely semi-consciousness, the patient may live in a state of stupefaction for several days or weeks, and because of the mental condition, of the feeble pulse, of the slight fever, of the coated tongue, be considered a case of typhoid fever or general tuberculosis.

Opium poisoning is to be separated from uræmia by the presence of an odor of laudanum on the breath if laudanum has been used instead of morphine, by the fact that the pupils are contracted to a pinpoint, and by the examination of the urine. From alcoholism uræmia is separated by the examination of the urine, by the odor of alcohol in the breath, and by the history of the patient. But it must not be forgotten that many alcoholics have chronic renal disease and that the ingestion of considerable quantities of alcohol may precipitate an attack of uræmia, and so an alcoholic history may be present which will mislead the physician.

As a rule, sudden fulminating uræmic symptoms develop in patients with chronic interstitial nephritis, whereas the types of uræmia with headache, vertigo, and other warnings of toxæmia are seen most frequently in the parenchymatous form.

In hot weather, when men are exposed to great heat in rolling mills and furnaces, the distinction between heatstroke and uræmia may be difficult, since in both conditions violent convulsions with cyanosis may be present. In heatstroke, however, the temperature is usually much higher than in uræmia and the cyanosis is usually more intense. It is, however, quite possible for heatstroke to complicate nephritis.

Prognosis.—The prognosis is always grave, but not necessarily fatal by any means. A professor in one of the medical schools of Philadelphia had a moderate uræmic seizure after nearly every lecture for a whole winter course of lectures before a final fatal seizure came on. In uræmia due to acute nephritis the prognosis is good if the patient can but survive the attacks long enough for the kidneys to regain their function. In the cases due to chronic renal disease the outlook depends to some extent upon the general state of the patient and particularly the condition of the lungs. If any tendency to pulmonary œdema or congestion is present, the outlook is much more serious.

Treatment.—The treatment of uræmia depends to some extent upon the variety of nephritis which has produced it and the peculiarities of the individual who is suffering from the attack. When uræmia comes on as a complication of acute nephritis, such as that complicating scarlet fever, the patient should have hot compresses placed across the small of the back, and, if diarrhœa is not already present, one of the saline purgatives, such as the citrate of magnesium or the sulphate of magnesium, should be given in sufficiently concentrated form to produce several watery movements. After this has been accomplished 5 to 10 grains of the citrate of potassium dissolved in Poland water should be given three or four times a day.

If the symptoms of uræmia persist, it will be necessary to place the patient in a warm pack. This may be given in one of two forms, the choice of the form depending upon the condition of the patient's skin and the presence of an eruption resulting from the disease. The choice also depends to some extent upon the temperature of the patient. If the rash has to some extent disappeared, the skin is dry and hot, and the temperature high, it is well to wrap the patient in a sheet wrung out of water at 70° or 80°, and then to immediately surround him with a dry blanket. The primary effect of this cold sheet is to aid in the dissipation of heat over the body, but it very rapidly becomes warmed by the heat of the body so that the patient at first is under the influence of cold, and very shortly afterward is surrounded by a warm pack. The primary cold drives the blood from the surface, and the secondary heating fills the peripheral capillaries so that the temperature is lowered by an improved peripheral circulation, and the skin is thoroughly supplied with blood so that it has a better opportunity to eliminate poisons. If no fever is present, and the rash has not faded, or if for any reason it is considered inadvisable to use cold primarily, the hot pack may be given, the patient being quickly wrapped up in a blanket which has been wrung out of water as hot as the skin can bear. Outside of this is placed a dry blanket and on the patient's head is placed an ice-bag to prevent cerebral congestion. Every few moments the patient is given a few sips of cold water to drive the blood from the internal portions of the body to the skin, the object being to flush the peripheral circulation, and to cause a sweat which will relieve internal congestion and eliminate impurities from the body. If the arterial tension is high, nitroglycerin may be given in the dose of $\frac{1}{300}$ of a grain to a child, or $\frac{1}{100}$ to a man, every three or four hours; or, in its place for a child $\frac{1}{2}$ drachm to a drachm of the sweet spirit of nitre may be given.

In the uræmia of chronic parenchymatous nephritis a plan of treatment identical with that which has just been described for that of acute nephritis may be carried out. As a rule, the patient is already too anæmic to permit of bleeding, and his tissues are so œdematous that hypodermoclysis is impossible.

In the uræmia of chronic contracted kidney with high arterial tension, the measures already indicated for the uræmia of acute nephritis may be instituted, the nitroglycerin being particularly useful, and being given hypodermically in order that it may act promptly. It also has a beneficial effect in that it relaxes the spasm of the renal bloodvessels and so produces diuresis. If there is much engorgement of the venous system, venesection is exceedingly useful, particularly if it is accompanied by free hypodermoclysis, or, in urgent cases, by an intravenous injection of normal saline solution. Sometimes in these cases if the heart seems strong, small doses of pilocarpine, $\frac{1}{8}$ of a grain, may be given hypodermically to aid in producing the sweat which is caused by the hot pack, and, with the object of preventing cardiac depression, it is usually wise to combine with it $\frac{1}{40}$ of a grain of strychnine. The lungs and the heart should be carefully watched, and if any signs of pulmonary œdema or cardiac failure develop, strychnine should be given freely, and 1 or 2 drachms of Hoffmann's anodyne should be administered as a rapidly acting diffusible stimulant. In some cases in which there is a tendency to suppression of urine, not only nitroglycerin but cocaine in the dose of $\frac{1}{8}$ to $\frac{1}{4}$ of a grain may be given hypodermically twice or thrice a day. Should convulsions occur, they should be controlled by chloroform, if they are exceedingly severe, and by the use of a drachm of bromide of sodium and 20 grains of chloral by the rectum. Morphine, which has been largely used to control uræmic convulsions, is not regarded with favor by most practitioners at the present time. If the arterial tension is exceedingly high, full doses (5 to 10 minims) of the tincture of veratrum viride, repeated every half-hour until some evidences of circulatory depression are produced, may be advantageous.

PYELONEPHRITIS AND PYELITIS.

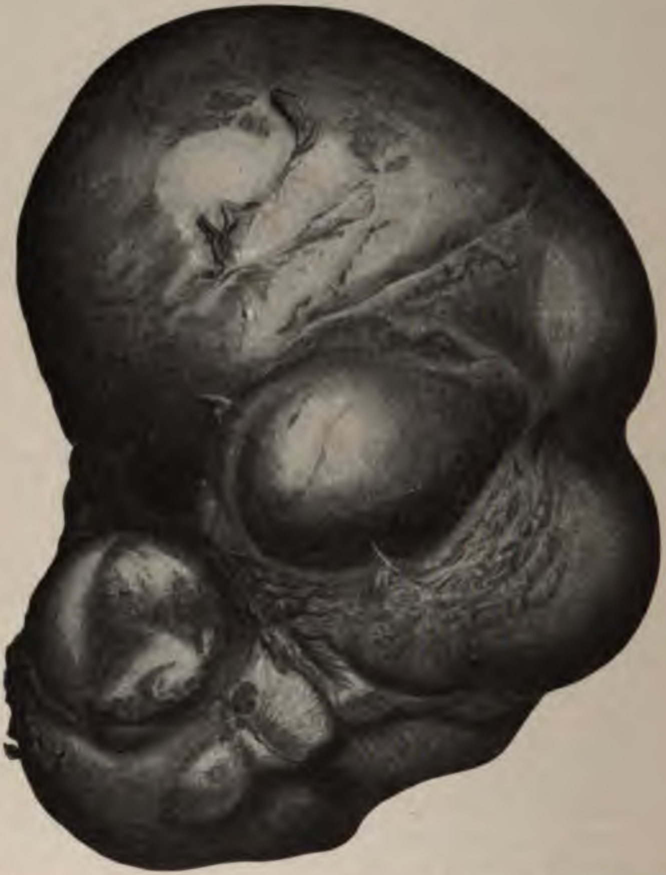
Definition.—Pyelonephritis signifies an inflammatory process involving both the pelvis of the kidney and the kidney texture itself. The term is usually applied to that form in which the condition is suppurative. Sometimes it is called suppurative pyelonephritis. Pyelitis is an inflammatory state of the pelvis of the kidney without involvement of the kidney proper. As synonyms to pyelonephritis we may use the terms pyonephritis, pyonephrosis, and caseative nephritis.

Etiology.—These conditions are nearly always the result of infection from below; that is, they are secondary to infection of the lower urinary tract, viz., the bladder or urethra. Very rarely infection of the kidney may take place through the blood, but this is only when the vital resistance of all the tissues is greatly impaired, or when the infection is very virulent, for the healthy kidney quite readily eliminates micro-organisms brought to it by the blood stream. It is possible, too, in cases of floating kidney, in which the ureter becomes twisted or obstructed so that the vital resistance of the pelvis is impaired, that infection through the blood may ensue. Although stones in the kidney are now attributed to bacteria, it is conceivable that a renal calculus by damaging the pelvic wall may prepare the way for infection, so acting as a direct cause of pyelitis. Infected emboli may also produce this state.

The micro-organisms which most frequently cause pyelitis and pyelonephritis are the *Bacillus coli communis*, the *Streptococcus pyogenes*, the *Staphylococcus pyogenes aureus*, the tubercle bacillus, the typhoid bacillus, the gonococcus, and the *Bacillus proteus vulgaris*. Brown has shown that the *Bacillus coli communis* is the most frequent cause in women, probably because of the near relationship of the anus and the meatus urinarius. Brown also asserts that, whereas some devitalizing cause is usually necessary to permit infection, a constantly ammoniacal urine is sufficient cause in many cases.

Pathology and Morbid Anatomy.—Pyelonephritis may be catarrhal, pseudomembranous, gangrenous, or suppurative. The first two forms usually depend upon, and are overshadowed by, associated diseases such as typhoid fever, and rapidly assume the suppurative type. In pyelonephritis the mucous membrane lining the pelvis of the kidney is thickened and coated with pus. A fibrinous exudate may also be present. The kidney structure may be involved in two ways: either small abscesses are scattered through the parenchyma of the kidney or in long white streaks which project themselves along the tubules. The renal tissue in and near these areas is, of course,

FIG. 97



Brewis' case of pyonephrotic kidney. Girth, forty-eight inches; weight, forty-five pounds.

necrotic. If the suppurative process proceeds, the calyces of the kidney become enlarged, the renal tissues waste and suppurate, and the kidney structure is largely replaced by a large single or multiple abscess. Finally, if the patient survives so long, the liquid drains off through the ureter, and the pus becomes inspissated so that a cheesy mass remains which may become infiltrated with lime-salts. This process may extend to the tissues surrounding the kidney causing paranephritis. When one kidney is involved the cause is nearly always primary disease in the bladder, and to this type the term " surgical kidney " is given.

Symptoms.—The symptoms of pyelitis and pyelonephritis may be in many cases so masked by the conditions which produce this disorder in the renal pelvis that they are overlooked. Thus the urinary picture is commonly obscured by an *associated cystitis*, which may either precede or follow the renal lesion. The most definite symptoms are *pain* and *tenderness in the back* over the kidneys, with or without *frequent urination*. The pain is increased by jarring the body or by coughing, and it is often felt in the testicle or inside of the thigh on the affected side. The quantity of urine passed is usually scanty in acute pyelitis but profuse in the chronic form. With the development of suppuration *septic fever* develops, vomiting may come on, and occasionally a *profuse septic sweat* follows a *chill* and *fever*. The *urine* is *acid* and contains *pus*, *blood cells*, and *degenerated epithelium*. At times the urine may appear perfectly normal, but soon returns to its earlier state. This variation is due to the ureter becoming blocked by a plug of putty-like pus, so that for several hours only one kidney, and that the healthy one, drains into the bladder. Such a variation in the urine therefore proves the difficulty to be unilateral. This plugging of a ureter may give rise to attacks of pain, somewhat like those due to a renal calculus becoming engaged in the ureter, but the pain is rarely so severe.

Diagnosis.—Pyelonephritis is sometimes taken for malarial fever, as are other septic processes, because of the chills, fevers, and sweats. An examination of the patient and of his blood and urine readily excludes malaria and reveals the renal disease.

In other cases the dry tongue, loss of weight, diarrhœa, and abdominal tympany may mislead one into a diagnosis of typhoid fever.

From suppurative cystitis the condition is to be differentiated by the fact that the pain is felt chiefly in the renal region, by the greater quantity of pus in the latter state, by the greater alkalinity of the urine in vesical disease, and finally by the use of the cystoscope and the ureteral catheter.

Usually there is more albumin in the urine in pyelonephritis than in cystitis, and more discomfort in the suprapubic area in the latter condition than in the former.

From perinephric abscess pyelonephritis is separated by the greater tenderness over the kidney in the former condition, by the fact that this area is not bulging. Most important of all is the absence of pus in the urine in the first condition and its presence in the second. In some cases, however, of pyelonephritis very distinct bulging over the kidney is manifested.

A valuable sign in this state is that the swelling occasionally disappears or diminishes as the pus and urine escapes through the ureter, when an obstruction is removed. A bulging or swelling in the renal area may also be due to hydronephrosis, but there is usually no fever in this state.

The possibility of a painful swelling in the region of the kidney being due to an aneurysm must always be excluded before operation is resorted to.

Prognosis.—Prognosis depends largely upon the cause of the malady and the state of the kidney. If the condition is one of simple pyelitis, occurring during the course of one of the infectious diseases, the outlook is not necessarily bad. (See Typhoid Fever.) If the suppuration is marked the prognosis is not good, and if the kidney structure is involved to the extent of pyonephrosis the prognosis is bad, and death may come from the exhaustion of prolonged septic fever, from the extension of suppuration to other parts, or because of amyloid degeneration in other organs.

Treatment.—The treatment of pyelitis in its milder phases consists in the use of mild diuretics if the urine is concentrated, of counterirritation by cups or heat over the loins, and rest in bed. No highly seasoned foods are permissible. The reaction of the urine must be determined. If the urine is acid, alkaline diuretics and salol are useful. If it is alkaline, then we may give 5 grains of uritone or urotropin three or four times a day in a glass of sparkling water. The diet should be hearty and easily digested. Bitter tonics and iron are useful, but quinine is contraindicated because of the state of the bladder, upon which it acts as an irritant. When hectic fever is developed and remains persistent the patient should be subjected to nephrotomy or nephrectomy before he becomes exhausted by sepsis. Opium or morphine may be needed to control the pain. Recently several observers have reported cases in which benefit was derived from lavage of the renal pelves, by means of ureteral catheterization.

HYDRONEPHROSIS.

Definition.—Hydronephrosis is a condition in which because of obstruction in the ureter there takes place in the pelvis of the kidney an accumulation of fluid which is not purulent. This fluid as it increases in quantity stretches and dilates the pelvis and the calyces until very large amounts of fluids are retained and a good-sized cyst is formed.

Etiology and Pathology.—Hydronephrosis may be acquired or congenital, constant or intermittent. It arises from permanent or intermittent closure of the ureter so that the urine cannot escape into the bladder. When congenital the ureter may never have been patulous or it may have a stricture or be abnormally inserted into the bladder wall. When acquired it arises from stricture of the ureter, or from plugging by a clot or a fragment of calculus. It may result from twisting of the ureter in floating kidney, but calculus is the most common cause of unilateral hydronephrosis. When either of these causes are responsible for the retention of fluid, the hydronephrosis may be intermittent, because, when the twist is undone, or when the calculus slips, the fluid can escape. The patient may remain free from trouble for years unless the obstruction forms again. The obstruction may be at the bladder, as in tumor of that organ, or consist in a paracystitis. In the female pelvic adhesions, neoplasms, and cysts may press upon the ureter and impede the urinary flow.

The secondary effects of this condition upon the kidney are disastrous if it is long continued and severe. The pressure acting upon the renal tissues causes atrophy and wasting so that finally the kidney structure largely disappears and in its place only a large collection of fluid, surrounded by fibrous tissue and remnants of renal tissue, remain. So large may the tumor grow that it projects downward into the abdomen, and it has even been mistaken for ascites. The other kidney may be similarly affected, but often it undergoes hypertrophy to compensate for the inability of its mate.

Symptoms.—The presence of symptoms depends largely upon the rapidity of the accumulation of fluid and the size of the renal pelvis. If it develops slowly and if a previous attack has enlarged the pelvic capacity, much fluid may be present without the patient presenting any symptoms. If, on the other hand, the fluid rapidly accumulates, *pain* may be very *severe*. When stricture is the cause the accumulation is usually slow, but when a twist or a calculus closes the ureter it is speedy and painful. In the slow cases *sharp pain* may be replaced by a *sense of weight* and *dragging*. A very characteristic sign in some cases, when the obstruction is suddenly removed, is a *profuse flow of urine* which fills the bladder rapidly, although it may have been emptied but a short time before. The tumor which may have been present in such a case disappears with the flow.

Diagnosis.—Palpation of the abdomen reveals in some cases a mass projecting from beneath the floating ribs, in which fluctuation may be detected. When the history of the causes and symptoms is as clear as has just been detailed the diagnosis is not difficult, but the history is frequently not clear. In children such a mass has been mistaken for an enlarged spleen and for a sarcoma of the kidney or of the retroperitoneal glands. (See Tumors of the Kidney.) In other cases, if the kidney is floating and hydronephrotic, the tumor may be taken for an ovarian cyst. In still other instances the tumor may so fill the abdomen as to lead to a diagnosis of ascites. Thus, Sutton has recorded a case in which the cyst held no less than thirty gallons! Aspiration of the fluid may reveal that it contains some urea or that it partakes of a urinous odor.

Prognosis.—The prognosis depends entirely upon the cause of the difficulty and the state of the other kidney. When the closure is congenital and complete, death ensues in a few days. When the closure is due to a twist of the ureter or to a calculus, much depends upon whether the flow is entirely stopped and how long it is arrested. A single attack followed by sudden relief may never be repeated. When the disease is bilateral the gradual involvement of the kidneys may result in uræmia, or if infection of the kidneys ensues suppuration may develop.

Treatment.—It is evident that no medicinal treatment can be curative in hydronephrosis. Morphine and atropine hypodermically to allay pain and relieve spasm may be useful at a time when the obstruction is complete and the accumulation of fluid rapid. If the condition is due to a floating kidney with twisting of the ureter, the replacing of the kidney in its normal position may give relief. In other instances the temporary assumption of the knee-chest position is curative. If the attacks occur very frequently, it may be wise to suture the kidney in place.

When hydronephrosis occurs in a woman, ureteral catheterization is of value: first, because it withdraws the accumulated fluid, and, second, because if a stricture is present a catheter may dilate the stricture and so exercise a curative influence. But catheterization of the ureter is a much more delicate procedure than catheterization of the bladder, and there is greater danger of infecting the ureter and kidney, so that the greatest possible caution in regard to asepsis must be secured. In certain cases where the accumulation of fluid is very rapid, and where the symptoms are urgent, aspiration has been practised, but this is not devoid of danger, and gives relief in only about 50 per cent. of the cases. Morris directs that the needle is best inserted at the most bulging point, but that if no such point is manifest it should be driven in half-way between the last rib and the crest of the ilium and between two and two and a half inches behind the anterior superior spine of the ilium, if it is the right kidney which is in trouble. An aspirating needle should be used instead of a trocar and cannula. When repeated aspirations are required for relief, nephrotomy is necessary.

CYSTIC DISEASE OF THE KIDNEY.

Cysts of the kidney occur as congenital malformations and are acquired in late life. There is perhaps no more striking object to be found in a collection of pathological specimens than a congenital cystic kidney. This condition not rarely affects both kidneys, which is an important point to bear in mind if any operation on one kidney is thought of. These cystic kidneys are not composed of one large cyst, but of a multitude of cysts massed together regardless of shape and size, and separated by fibrous bands or by strands of atrophied renal tissue. The contents of the cyst is usually a clear yellow fluid with an acid reaction and containing urinary salts, but occasionally the fluid is opaque and may contain small amounts of blood. The causes and processes by which these cysts are developed are not definitely known, but it is thought they are formed by extraordinary dilatation of the tubules or of Bowman's capsules. Shattock believes they are due to mal-development of the mesonephron. Such kidneys often weigh several pounds. Although congenital in origin, it is to be remembered that life may be continued far into adult years before they give any trouble.

Congenital cystic kidney (Fig. 98) may project well below the ribs and give rise to a diagnosis of sarcoma, hydronephrosis, or of enlarged spleen.

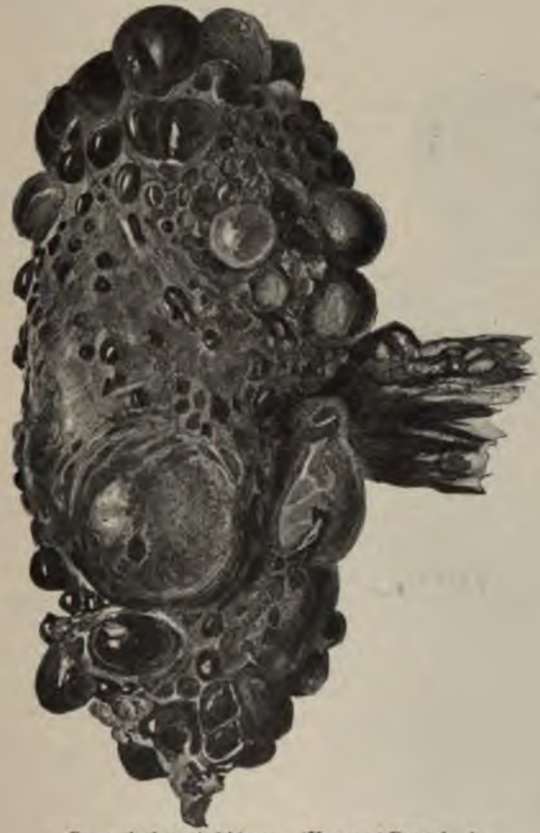
Cysts of the kidney, single or multiple, may be present in kidneys which otherwise show no abnormalities, and these cysts may be smaller than a pea or larger than an orange. Their contents may be clear or brown in color, and may be gelatinous in character.

Attention has already been called to the small cysts which are seen on the surface of the kidneys in chronic interstitial nephritis. At times it is difficult to determine whether the kidney is the site of acquired or congenital cysts when these cysts become large and multiple.

Echinococcus cysts of the kidney also occur. (See article on Parasitism.)

The *symptoms* of congenital cystic kidney are in no way peculiar unless the kidney be large enough to project in the manner described. Aside from this sign the patient presents no signs of renal disease until the cyst, by increase in growth and resulting decrease in renal tissue, develops renal failure, and the signs of chronic nephritis ensue. In some cases a sudden attack of uræmia may be the first symptom of renal difficulty. When the condition persists and adult life is reached, there may be a *high arterial tension* and *hypertrophy of the heart*, as in ordinary chronic nephritis. Often they interfere but little with the life of the patient. In the museum of the Jefferson Medical College is a cystic kidney weighing seven pounds, diagnosed during life by the late Dr. J. M. Da Costa, and carried by a busy

FIG. 98



Congenital cystic kidney. (Kast and Rumpler.)

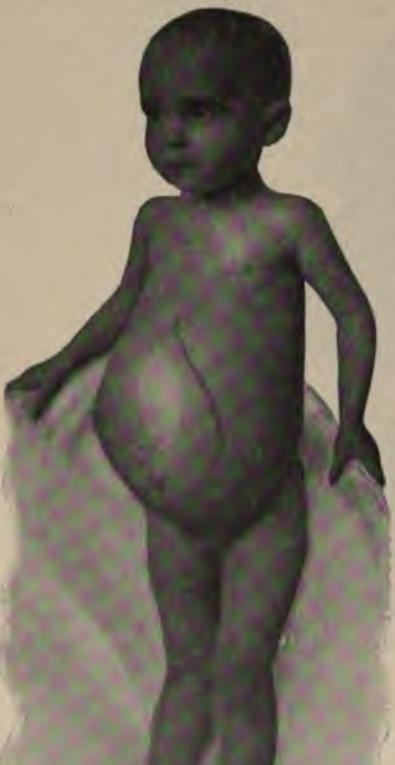
practitioner of medicine for over two years afterward. The other kidney was but slightly affected.

Beyond the use of pain-relieving drugs, there is nothing that can be done for these cases. Operative procedure is contraindicated in the sense of nephrectomy, because the other kidney is so often diseased that it is unable to carry the burden of elimination if left by itself.

TUMORS OF THE KIDNEY.

The kidney is not rarely the seat of morbid growths. They may be *benign* or *malignant*. The benign growths are the fibromata, which chiefly affect the pyramids of the kidney, and less commonly the lipomata and angiomata. Occasionally papilloma of the mucous membrane of the pelvis of the kidney develops. The most common malignant growth in the kidney is sarcoma, which is by no means very rare in young children, and often grows to a very great size. Sarcoma of the kidney secondary to sarcoma elsewhere is also met with in adults. Endothelioma may develop. They are all vascular and often bleed, producing hæmaturia. Adenoma of the kidney usually springs from the cortical tissues, but it may grow to so large a size that it takes the place of most of the renal tissue. They occur frequently in children and are walled off from the rest of the kidney by a fibrous sheath. Adenoma is found in two forms, the papillary and alveolar. Not rarely considerable areas of necrosis develop in these tumors. Cancer of the kidney as a primary growth is rare. As a secondary growth it is more common.

FIG. 99



Sarcoma of the right kidney. The dark line on the abdomen is a blue-pencil outline of the tumor. (Le Conte.)

Of all the tumors affecting the kidney those arising from ectopic adrenal tissue are probably the most frequent. Such tumors, called "hypernephromata," vary in size from almost microscopic masses to growths larger than an adult head. In their earlier development such neoplasms are benign, but later tend to involve adjacent structures, and by metastasis the lungs. If they occur on the right side, the liver is often affected. These tumors are soft, vascular, yellowish, or blood-tinged masses developing in the kidney, and often cause hæmaturia.

The symptoms of renal tumor, if the growth is benign, are not marked, unless it grows large enough to produce pressure.

When the tumor is malignant, free *hæmaturia*, with clots moulded to the shape of the ureter, may be present. *Pain* develops only when the growth presses on neighboring parts or on adjacent nerve trunks, or when the weight of the growth is such as to cause a *sense of weight in the loin*. Severe attacks of colicky pain may, however, be present when a clot is being forced through the ureter. In some cases marked loss of flesh takes place, but children with renal sarcoma often remain remarkedly well nourished. The tumor, if large, may project well forward in the belly and give rise to the belief that it is an enlarged spleen or liver (Fig. 99). This error is frequently made. The colon may give tympany on percussion over the growth, showing that it springs from behind that part of the bowel. Sarcoma of the kidney must be separated from sarcoma of the retroperitoneal space. Care should be taken that it is not confused with cystic kidney, or hydronephrosis, for the malignant growth may be nodular and very elastic, or even give a sense of fluctuation on palpation.

There is, of course, no medical treatment of this state. In some cases nephrectomy of the sarcomatous kidney has been performed in young children with good results, but there is usually a metastasis elsewhere, which ultimately takes life.

NEPHROLITHIASIS.

Definition.—This condition is often called "stone in the kidney," or "renal calculus." It is due to the formation in the tissues of the kidney, or in its calyces or pelvis, of concretions composed of solids derived from the urine.

Etiology and Pathology.—The concretions, when in the pelvis of the kidney, may be single or multiple, and very great variations in their size may be met with. In some instances they are so small as to be scarcely larger than grains of sand; in other cases they may be as large as a pea or bean, and in still other instances a large calculus may form which completely fills the renal pelvis and projects itself into the ureter and into the calyces and infundibula, forming what is called a "coral calculus." The latter form is, of course, never passed from the kidney, but the smaller stones often become engaged in the ureter, and in their passage through it to the bladder cause intense pain. Not rarely the fine renal sand passes so readily that it attracts no attention until it is seen in the urine.

Not only are concretions found in the calyces and pelvis, but also in the tissues of the kidney. Thus, formations of uric acid may take place in the tips of the pyramids after birth and cause much pain in the first month of life. Again, accumulations of sodium and ammonium urate are not rarely found in adults at the tips of the pyramids, particularly in gouty invalids, and in very old persons a deposit of lime-salts is found in streaks in the pyramids.

The concretions just spoken of may be formed of a number of urinary solids, such as uric acid, calcium oxalate or phosphate, urate or carbonate. Cystin and xanthin also are ingredients. The mere existence of these substances in the urine is not the cause of the formation of stone, however, for if this were true everyone would have calculus. There are at least two additional factors present, one of which is the presence of an albuminoid substance, which serves to glue together tiny particles of these solids, and a condition in which there is an abnormal tendency to crystallization of these bodies. The bacterial origin of gallstones and the presence of micro-organisms in the nuclei of renal calculi suggest a similar origin for both.

The most common ingredient of stone is uric acid or the urates. Stones of this character are met with in people who, because of small quantities of fluid ingested, have a scanty urinary flow, or who, by reason of great activity of the sweat glands, have little urine. As a consequence of concentration and high acidity of the urine, the uric acid and urates are readily separated in solid form and held together by the albuminous matrix. Stones of this character are quite hard, and their surface is smooth and reddish.

Phosphatic calculi are of the most common occurrence after those formed from uric acid and the urates. They are composed of calcium phosphate, ammoniomagnesic phosphate, or both, but they are rarely found in the kidney, being generally developed in the bladder. They are usually formed when the urine is persistently ammoniacal.

Next to phosphatic calculi, those formed of calcium oxalate are most commonly met with. They are peculiar in respect to their great hardness and their roughened surface (mulberry calculi). Sometimes when they are small they are smooth and rounded, "hemp-seed calculi." They are dark in hue and not infrequently, on being split, they are found to be formed about a nucleus of uric acid. Oxalate stones are only met with in those who, because of digestive or metabolic disorders, pass considerable amounts of oxalates in the urine.

The effects of the presence of stone in the renal pelvis are not always marked. Indeed, calculi may be present for years without causing any discomfort whatever. Sometimes they suddenly cause trouble if the patient suffers from a fall which causes the stone to damage the lining membrane of the pelvis, and as a result hæmaturia may ensue, or the stone may be started from its nest, and, proceeding to travel down the ureter, cause an attack of colic. In still other cases the stone may cause a hydronephrosis by plugging the orifice of the ureter. Again, the damage done by the sudden movement of a stone against the tissues may open a path for infection and consequent pyelitis or even pyelonephritis.

Frequency.—In certain parts of the world stone is very prevalent, notably in some counties in England. This is probably due to certain mineral ingredients of the water which is taken for drinking purposes. Stone is also very commonly met with in China and in India. The late Dr. Kerr, a Chinese missionary, removed hundreds of vesical calculi during his residence in China. So far as I have been able to discover, stone is not much more prevalent in one part of the United States than in another.

Prognosis.—The prognosis of nephrolithiasis depends entirely upon the question of the state of the kidney tissues about the stone or stones. In many cases the stone produces no trouble for years. If, as the result of an injury or infection, the surrounding tissues become diseased, the state of the patient may become serious from pain or from sepsis.

Symptoms.—As just stated, stones may be in the kidney for years *without causing any signs*. When they escape into the ureter they cause *renal colic*, which is due to three causes: first, blocking of the ureter results in obstruction to urinary flow which causes distention; second, the pressure of the urine on the stone forces it forward through the narrow canal, often wounding its lining, and, finally, the walls of the ureter are spasmodically contracted because of the presence of the stone. The pain is often so severe as to be a horrible agony. I have seen a strong and brave man grovel on the floor groaning with anguish, and vomiting because of its severity. The *pain extends into the pelvis* and the *inner side of the thigh* on the affected side, and even *into the testicle* and *penis*. It also radiates into the back of the chest. These symptoms may persist for an hour or for several hours. In the latter instances there are often temporary remissions in the pain. Not rarely the *bladder* is exceedingly *irritable*, and the patient continually passes small quantities of urine which contains traces of blood from the affected ureter, but most of the urine comes from the normal side.

In cases of suspected renal calculus the urine should be examined microscopically for blood cells, both during and between attacks. They are practically never absent when a calculus is lodged in a ureter.

When both kidneys are affected, total *suppression of urine* due to obstruction of the ureter or to reflex irritation may ensue, and the resulting toxæmia produce death. It is, however, a noteworthy fact that this state is rarely rapid in onset or rapidly fatal. The patient often lives for many days, unless there has been renal disease present for some time with some degree of toxæmia. I have recently seen a case in consultation in which no urine had been passed for a week, and the catheter obtained nothing from the bladder, yet the patient was conscious and alert when spoken to, appearing drowsy only when left alone. This is a state quite separate from ordinary uræmia, and has been called "latent uræmia." (See Uræmia.)

After an attack of renal colic has passed, *pain* and *soreness* are felt for some hours or days in the *affected loin*, and tenderness on pressure may be elicited.

Diagnosis.—The pain of renal colic must be separated from that of acute appendicitis, that of gallstone colic, and from neuralgia. It must also be distinguished from the pain due to hydronephrosis resulting from a twist in the ureter. Sometimes a diaphragmatic pleurisy may mislead us. The peculiar radiation of the pain into the groin, penis, and inside of the thigh is diagnostic. The pain of gallstone is radiated into the back, and is often associated with jaundice. Neuralgia does not cause bloody urine. Twist of the ureter can be predicated by finding a floating kidney. Pleurisy is defined by the area of the pain, by fixation of the diaphragm, and by a friction sound in some cases. A valuable, but by no means absolutely reliable, method of diagnosis is the use of the Roentgen rays, which may or may not reveal a stone, and which sometimes has caused the surgeon to operate when no stone has been found.

Treatment.—The treatment of nephrolithiasis may be divided into two parts: that devoted to the relief of the patient at the time of the attack of renal colic, and that devoted to the prevention of the formation of new stones or an increase in the size of those already present. For the relief of the attack of renal colic, a hypodermic injection of $\frac{1}{4}$ grain of morphine, with $\frac{1}{150}$ grain of atropine, should be given at once; or, if atropine is known to be disagreeable in its effects upon the patient, nitroglycerin may be used hypodermically, for the double purpose of aiding in relaxing the spasm and because it tends to prevent the after-disagreeable effects of the opiate. If the heart is in a satisfactory condition, chloroform may be given by inhalation, and if the patient will lie quietly enough to permit it, hot applications may be made over the painful kidney.

In the intervals between the attacks the patient should be instructed to drink large quantities of some pure water like Poland water, or one of the Lithia waters, which depend chiefly for their effects upon their purity rather than upon their lithia. If the urine is alkaline, lithia waters are contraindicated, and under these circumstances it is well not only to use copious draughts of water, but to direct the patient to take uritone, urotropin, or benzoate of ammonium, for the purpose of making the urine acid.

When the urine is excessively acid, it is advisable for the patient not only to drink large quantities of water, but also to take 15 or 20 grains of bicarbonate of potassium three or four times a day. In other instances the citrate of potassium may be given. Sometimes good results follow the use of Celestins Vichy water when the urine is acid. If an examination of the urine reveals the presence of a large number of urates, it must be borne in mind that these have their origin in disordered gastrointestinal functions, and the diet must be carefully regulated, and nitromuriatic acid given in full doses. Sometimes, too, these cases are benefited by the administration of such intestinal antiseptics as salol, or aspirin, in the dose of 5 or 10 grains three times a day.

The patient should be forbidden to drink any sweet wines or beer, and the only form of alcoholic stimulant permitted should be rye or Scotch whiskey. If the patient can do without any alcohol at all, it is much better for him to be content with non-alcoholic drinks.

If the patient is one who is accustomed to leading a sedentary life, he should be instructed that an amount of exercise which is sufficient to produce healthy fatigue is absolutely essential; but if he exercises he should also drink copiously of water.

When pain in the kidney is continuous or so frequent in its recurrence that the enjoyment of life is impaired, or if there is any evidence of the tissues of the pelvis of the kidney or of the kidney itself being irritated or infected, the question of operative interference must be carefully considered, and the patient advised to seek surgical relief.

PERINEPHRIC ABSCESS.

An abscess sometimes forms around the kidney by the extension of infection from the pelvis of this organ, because of transmitted lymphatic infection from a suppurative appendicitis, from injury to the tissues by a blow or fall, by extension of infection from spinal disease, from perforation of the stomach or bowel followed by subdiaphragmatic abscess, and rarely after acute infectious fevers, as typhoid fever. The pus may cause *pain* and *bulging over the kidney;* it may burrow upward, and escape into the thorax, or downward and resemble a psoas abscess. The pus is usually very foul and distinct septic symptoms may be present. On the other hand, I have seen at least two cases in which there was little pain and no fever. There was nothing more than some discomfort, with swelling, over the kidney. Not rarely the spine is fixed, and the leg on the affected side is drawn up when the patient lies down.

Treatment.—The treatment is, of course, operative. The patient's vitality should be supported by food and stimulants.

DISORDERS OF URINARY SECRETION.

Anuria.—Anuria is a condition in which there is a total suppression of urine. It arises as the result of thrombosis of the renal vessels, and an intense acute nephritis such as that which follows the ingestion and elimination of very irritant poisons as cantharides and turpentine. Sometimes complete anuria also follows the administration of ether when this anæsthetic has been given over a long period of time and in too large quantities. It is particularly prone to occur if the kidneys are already in a state of irritation. In other instances total suppression of urine results, reflexly, from the irritation produced by nephrolithiasis. In most instances when this occurs both kidneys are affected. Partial or complete anuria also sometimes occurs after operations upon the genito-urinary tract. A careful distinction should be made between anuria when no urine is *secreted* and retention of urine in which no urine is *passed,* but in which the bladder is found to be well filled.

Treatment.—The treatment of anuria varies somewhat with the cause. When the arrest of secretion is due to the presence of stone, an operation is theoretically demanded, but usually the diagnosis as to the cause is not completed before the patient's condition has become so grave that operative interference is of questionable propriety. When the suppression of secretion is not due to stone, but to reflex irritation or spasm of the renal vessels, I have known full doses of nitroglycerin given every three or four hours, hypodermically, to relax the bloodvessels and cause free urinary flow. This is particularly apt to occur if, simultaneously, the patient receives a large injection of cool water by the bowel. If saline solution is used, care should be taken that it is not of more than 0.6 per cent. strength, since strong saline solutions abstract liquids from the tissues instead of being absorbed. In these instances our desire is that fluid shall enter the tissues, find its way to the bloodvessels and so flush the kidneys. Usually about one quart of liquid should be given by gentle hydrostatic pressure. Hot compresses, or poultices, may be laid across the loins, and if there is any reason to believe that renal congestion is present, three or four dry or wet cups may be applied over each kidney. In some instances moderate doses of the bromides and the vegetable salts of potash, like the citrate, are advisable. If the heart is strong the patient may be subjected to a hot pack or may be given a Turkish bath by elevating the bed-clothes and allowing hot air to surround his body. Small doses of pilocarpine may be given as a diuretic, particularly if the heart is guarded by strychnine. The difficulty in using large doses of pilocarpine is that it is prone to produce pulmonary œdema.

Hæmaturia.—Bloody urine, or hæmaturia, is a condition in which there is found in the urine not only the coloring matter of the blood, but red blood corpuscles as well. It sometimes occurs with the stage of onset in acute fevers and in certain cases of leukæmia. It is also met with in certain forms of malarial infection of the æstivo-autumnal type, and in cases of infarction of the kidney arising during an attack of endocarditis or from other causes. When there is stone in the pelvis of the kidney or any part of the conducting tract, and the patient is jarred or jolted, the stone may cause sufficient local damage to produce bloody urine. Parasites such as the *Filaria sanguinus hominis* and the *Bilharzia* may produce the same condition. Blood also appears in the urine as the result of papilloma, cancer, or other neoplasm of the bladder, ureter, renal pelvis, or kidney, ulceration of the urethra, and of injuries to the genito-urinary tract by falls or blows. Sometimes, too, it develops in the course of scurvy and purpura hemorrhagica. In acute, and even in chronic parenchymatous, nephritis the urine sometimes contains small amounts of blood, but they are visible only under the microscope.

The appearance of the urine when it contains blood is quite characteristic. It is not only dark red in hue, but it is opaque and contains considerable sediment, which is chiefly composed of fibrin and blood corpuscles. In pure renal hæmaturia the voided urine is more of a smoky hue, the red cells are of the shadow or phantom type and clots are rarely if ever present. If the urine is very alkaline, the red blood corpuscles may be dissolved rapidly or become colorless and difficult to see. Clots formed before the urine is passed nearly always arise from hemorrhage in the bladder, but occasionally moulds of the ureters may appear. An examination by means of the cystoscope or urethroscope may be necessary to determine the source of the hemorrhage, and if the blood comes from the kidney it may be necessary to catheterize the ureters to determine which kidney is damaged.

There are occasional instances of hæmaturia in which, even at autopsy, no sufficient cause can be found. These include the angioneurotic hæmaturia of Klemperer, the renal hæmophilia of Senator, and Gull's "renal epistaxis."

Treatment.—Treatment of hæmaturia depends largely upon its cause. The patient should be put absolutely at rest. If the blood comes from the kidney there is no treatment which can be relied upon as being efficacious.

Chloride of calcium in the dose of 5 grains three or four times a day may be given well diluted with water to increase the coagulability of the blood. Gallic acid, tannic acid, and sulphuric acid have been largely used by some practitioners, but it is doubtful if they really exercise any definite influence. If any one of them does act as a styptic it is probably sulphuric acid. Ten drops of the aromatic acid may be given every three or four hours, well diluted, and counterirritation may be applied in the shape of cups or hot compresses over the kidneys. Counterirritants like mustard, turpentine, and cantharides should be avoided, as they may be absorbed and increase renal irritation. The patient should be protected from cold. If the hemorrhage comes from the bladder, an injection of a pint of normal salt solution containing ½ to 1 ounce of adrenalin chloride solution 1:1000 should be injected.

When the hemorrhage comes from the urethra a similar plan of treatment can be resorted to, simply instilling the adrenalin into the portion of the urethra which is bleeding. The objection to the use of substances which cause a coagulation of the blood is that they produce clots, which may become septic or give rise to obstruction.

Hæmoglobinuria.—In all probability hæmoglobinuria occurs most frequently as a complication of malarial infection. There is much doubt as to the actual cause of this condition. In some instances it is probably due to the destructive action of the malarial parasite on the blood, but it would seem probable that in other instances it is due to an associated infection or a condition which quinine cannot be expected to remedy. Indeed, a large number of cases are now on record in which the administration of quinine has been followed by hæmoglobinuria or hæmaturia. This condition is to be distinctly separated from hæmaturia, for in this case no blood corpuscles are present, but only the hæmoglobin or coloring matter of the blood. Its presence does not indicate any lesion in the genito-urinary tract. Strictly speaking true hæmoglobinuria is not present, but methæmoglobinuria. The urine is clear, but may be quite dark in hue, and deposits on standing a heavy, reddish-brown sediment. It usually gives the reaction for albumin.

Hæmoglobinuria arises from the ingestion of a large number of poisons, such as poisonous mushrooms, chlorate of potash, pyrogallic acid, and some of the coal-tar products. Not long since I had under my care a physician who suffered from repeated attacks of hæmoglobinuria whenever he tinkered with his automobile, which was stored in a small, tightly closed shed in which the fumes of gasoline were quite concentrated. He never suffered with hæmoglobinuria before these exposures, and since avoiding them has had no return of his trouble. Hæmoglobinuria sometimes follows severe burns. It may also develop in Raynaud's disease. The discoloration of the urine produced by carbolic acid is not due to hæmoglobinuria, but to a dark, oxidized educt, which is in part hydrochinon.

Hæmatinuria.—Under the name of paroxysmal hæmatinuria, sometimes called *hæmoglobinurie a frigore,* a condition rarely occurs in which the urine varies in color from a port-wine to a chocolate-brown, or almost black hue, the alteration in its appearance lasting, however, for but a few hours. Its specific gravity usually ranges from 1.025 to 1.027. The urea is increased. The quantity of blood which is represented has been estimated as equivalent to from seven to twelve parts in one hundred of urine. Blood corpuscles are very rarely found in the fluid. The urine not only contains hæmoglobin, but considerable quantities of albumin and globulin. Not infrequently hyaline and granular casts are present. This condition is chiefly provoked, apparently, by exposure to cold—that is, by chilling of the surface of the body. Aside from exposure to cold and chilling of the surface of the body, severe muscular exercise seems to be a causative factor, and not infrequently there is also a tendency to vasomotor disorders. Indeed, it is probable that a large part of the disorder lies in an abnormal vasomotor condition. Gilman Thompson has recently reported two cases and summarized the literature. It appears that during the past forty years only 206 cases have been reported, and that most of these have appeared in England, Germany, and France, and that very few indeed have been reported as occurring in the United States and Canada. The condition affects males very much more frequently than females, there being only about 4 per cent. of females in the 206 cases so far reported. The period of life at which it commonly occurs is between thirty and forty years, but cases have been reported as late as the sixty-fourth year. At the time of the attack there is usually a sharp rise of temperature, amounting to 102° or 103°, but this falls to normal almost as rapidly as it rises, the febrile period lasting only a few hours. Chills are often present, and may be the first symptom of the attack. Jaundice, which is hæmatogenous in origin, develops. Some persons have thought that the jaundice is hepatogenous, but this is unlikely in view of the fact that the urine is not bile-stained and the stools are not lacking in bile.

Paroxysmal hæmoglobinuria is to be separated from the hæmoglobinuria met with in some cases of malaria by the fact that the latter is a disease of tropical or semi-tropical regions, whereas paroxysmal hæmatinuria usually occurs in cold climates. In one case the malarial organism is present and in the other it is absent.

Paroxysmal hæmatinuria is also associated with a neurotic condition, with urticaria and with localized areas of cyanosis.

Treatment.—The treatment consists in avoidance of exposure to cold and to causes which produce nervous excitement. Tyson has suggested the use of suprarenal gland. Thompson suggests the use of thyroid extract. Saundby commends calcium chloride and Chvostek believes that inhalations of nitrite of amyl may be useful to abort an attack. If the circulation is feeble, rapidly acting diffusible stimulants like Hoffmann's anodyne, aromatic spirit of ammonia, and small doses of spirit of chloroform are advantageous.

The treatment of hæmoglobinuria cannot be direct. Copious draughts of water to flush the kidneys, careful attention to the state of the bowels, protection from exposure of the surface of the body to cold, and the use of foods which are not highly seasoned and irritating to the liver and the kidneys are the only measures which the physician can institute. When the cause of the condition is malaria the debatable question of administering quinine must be discussed and decided. (See Treatment of Malarial Fever.)

Albuminuria.—Albuminuria is a term applied to a condition of the urine in which serum albumin, serum globulin, and, by some writers, other urinary proteids, including nucleo-albumin, albumose, peptone, or fibrin is found in it. While it is true that delicate chemical tests will frequently reveal traces of albumin in the urine, it is also a fact that any quantities which can be appreciated by the use of heat and nitric acid or by the potassium ferrocyanide or mercuric iodide tests are to be regarded as abnormal.

As albumin is a colloid substance and therefore does not readily diffuse through animal membranes it does not pass through the bloodvessels of the kidneys and renal tubules unless these structures have undergone some degenerative change, or are subjected to a pressure which they cannot withstand. Sometimes, too, albuminuria may be due to changes in the blood itself, whereby its albuminous ingredients are altered or the renal texture is secondarily affected. The presence of albumin in the urine when disease of the conducting apparatus can be excluded (accidental albuminuria may occur through contamination of the urine by vaginal discharges) is therefore indicative in the vast majority of instances of some renal lesion, and as chronic parenchymatous nephritis and chronic interstitial nephritis are the most common renal diseases, it is usually indicative of one of these maladies or of a subacute nephritis complicating one of the acute infectious diseases. Twenty years ago albuminuria was considered as pathognomonic of Bright's disease. We now know that albumin often appears in the urine when Bright's disease is not present.

As a general rule it may be stated that the quantity of albumin is in direct ratio to the severity of the renal lesion, but there are certain notable exceptions to this, as in the case of chronic contracted kidney, in which disease the kidney is seriously affected, yet the albumin is always in small quantity and may be absent at times.

Albuminuria is not rarely met with in cases of congestion of the kidney due to cardiac failure. Under these circumstances the quantity of albumin present may be very large, almost as great as that which is found in chronic parenchymatous nephritis. It is due under these circumstances to a structural and probably nutritive alteration brought about by passive renal congestion, and the use of cardiac stimulants usually results in its disappearance, at least to some degree. So, too, albuminuria may develop in certain persons after severe and prolonged exercise, as in soldiers after a long march, or in athletes after a long run. One cause of this, at least, is feebleness of the heart from exhaustion.

In still other cases what is known as "Cyclic Albuminuria" comes on, which is sometimes due to exposure to cold, and at other times seems causeless. Cyclic albuminuria is sometimes called the "albuminuria of adolescence," and is, as its name implies, intermittent and is usually not present when the patient rises in the morning, but appears as the day progresses. The upright posture is in some cases sufficient to induce the condition—"orthostatic" albuminuria. Usually the urine is above the normal specific gravity and contains no casts. As its name indicates, it occurs at puberty, in easily fatigued, overgrown, pallid children. It usually disappears when puberty is passed and the system is established on an adult basis.

In certain persons the ingestion of excessive quantities of albumin in food also produces albuminuria.

A very high arterial tension due to cardiovascular disease may also cause this symptom. While it is true that the cardiovascular disease usually results in some impairment of the kidney, it is also a fact that reducing arterial pressure in these cases by the use of nitroglycerin often stops the albuminuria.

Albuminuria may be due, as already stated, to changes in the condition of the blood itself, met with in certain cases of anæmia, and in diseases like purpura, scurvy, and other conditions which cause marked changes in the circulating fluid.

TESTS.—Albumin is best detected in the urine by the general practitioner by the use of the so-called heat and nitric acid tests, which may be used separately or in conjunction. A test-tube is two-thirds filled with urine, which if cloudy should be filtered, and if alkaline acidified, and the upper part of it is held over a lighted alcohol lamp so that the fluid in this portion of the tube soon boils. Under these circumstances if albumin is present the upper portion of the urine becomes clouded from coagulated albumin, but the portion below remains clear until the coagulated albumin is precipitated. If earthy phosphates are present some cloudiness of the fluid develops, but the addition of a few drops of nitric acid disperses the cloud if it is due to the phosphates, but does not do so if it is due to albumin. Many physicians use a somewhat less accurate test, which consists in placing one-half to one drachm of nitric acid in a test-tube, and allowing an equal quantity of urine to trickle down the side of the tube so that it overlies the acid. If albumin is present a layer of albumin appears at the point of juncture of the two fluids. Sometimes, if marked intestinal fermentation is present, a reddish-brown, but transparent zone, appears at this level also.

For the purpose of making a delicate test the potassium ferrocyanide method may be employed. The writer has found it most convenient to use for this purpose the so-called urinary test tablets which are now placed upon the market. Into 30 minims of urine is placed a citric acid tablet for the purpose of acidification. To this is then added a tablet of potassium ferrocyanide, and the tube is shaken or allowed to stand still until both tablets are completely dissolved, when if albumin is present tiny flocculi may be seen floating in the fluid, which settle to the bottom of the test-tube when it is placed at rest. This is a delicate test for albumin, and has the advantage that it does not precipitate mucin, peptones, phosphates, urates, or vegetable alkaloids. If the physician does not wish to use these tablets, he may add to a test-tube half-full of urine 5 or 6 c.c. of a freshly-prepared solution of potassium ferrocyanide of the strength of one in twenty, adding 10 to 15 drops of acetic acid. In other instances the physician may, if he chooses, employ potassiomercuric iodide test tablets in the same manner, with equally good results. When the potassium mercuric iodide acid test is used, the cloudiness due to albumin does not break up into flocculi, as it does when potassium ferrocyanide is employed.

The following facts in regard to these tests should be remembered. If the specimen of urine is very alkaline, more than one citric acid tablet, or an extra quantity of acid solution should be added. If cloudiness is produced by the acid, it is due to mucin, uric acid, or some oleoresin, as, for example, when copaiba or cubebs have been taken internally. If the urine is warmed the urates dissolve, but the mucin remains. The precipitate produced by the oleoresins clears up by boiling, but returns as soon as the urine cools slightly. When the tablet, or solution, of potassium mercuric iodide or potassium ferrocyanide is added to the acidulated urine, and cloudiness is produced, the urine must be heated. If the reaction is due to albumin the precipitate remains undissolved, but if it clears up it may consist of peptones or derivatives of vegetable alkaloids if the mercury test has been employed. When the potassium ferrocyanide test is used peptones are not precipitated, and may therefore be excluded.

For the quantitative estimation of albumin Esbach's method is most commonly employed. It consists in using a graduated test-tube which is called an albuminometer. This test-tube is marked with the letter "U," and higher up with the letter "R." Below the letter "U" are graduate lines from 1 to 7. Urine is placed in the tube to the level of the letter "U," and the following solution is then added till the fluid in the tube reaches the letter "R." The solution used consists of 10 grams of picric acid; 20 grams of citric acid; 1000 cm. of distilled water. The tube is now corked and inverted several times until the test solution and the urine are completely mixed. It is then allowed to stand on a rack in a perpendicular position for twenty-four hours. At the expiration of the twenty-four hours the albumin is found to be at the level of one of the numbers cut on the side of the tube, and this represents the number of grams of albumin per litre. If it is desired to know the percentage of albumin, a decimal point is placed in front of the figure. In cases where the quantity of albumin is so great that it cannot be measured by the ordinary Esbach tube, the urine should be diluted with water, and the result in grams multiplied by the number of times the urine has been diluted.

There is no excuse for neglecting examination of the urine for albumin. In the absence of other apparatus and reagents the urine may be acidified with vinegar, boiled in a spoon or cup, and if necessary poured into a glass for inspection.

From what has been said it must be evident that the significance of albuminuria varies. Usually, except in the case of chronic interstitial nephritis, its importance from a prognostic standpoint is in direct relation to its quantity; its cause is also an important factor concerning prognosis. The presence of tube casts with the albumin is also of great importance, particularly if these tube casts are granular and contain fatty globules. Tube casts should always be sought for in the urine, if need be with the aid of the centrifuge, but, on the other hand, it should be remembered that if the centrifuge is thoroughly employed, there are few specimens of urine which will not reveal an occasional cast.

No one perhaps has studied more carefully than has Leube this question of albuminuria. His view is that while in many cases physiological albuminuria does occur, particularly after severe exercise, we should nevertheless regard all such instances with suspicion. Washburn, in studying the records of life insurance cases who were supposed to have physiological albuminuria, found that the death rate among them was 17.5 per cent. instead of 9 per cent., as it should have been.

A careful study must be made of the heart, kidney, lungs, and other organs before the patient is given a favorable prognosis.

Wright and Ross have shown that it is sometimes possible to differentiate the albuminuria of renal disease and physiological albuminuria by increasing the coagulability of the blood through the use of calcium lactate. (For dose see article on Purpura.) If the use of this drug arrests the albuminuria it does not depend upon actual renal disease.

While casts may be present in the urine of patients who are thought to be healthy, but who have albuminuria, these casts should disappear if the patient rests in bed, and they should not be epithelial casts. If they are present, then it is not physiological albuminuria. It is perhaps best to say that albuminuria ought not to be present, and that its existence at least excites suspicion of some renal change. But it does not necessarily mean Bright's disease, and I have seen more than one case in which distinct albuminuria was present without the association of casts for more than fifteen years.

Pyuria.—Pus in the urine may arise from pyelitis, pyelonephritis, cystitis, urethritis, vaginitis, or the rupture of an abscess into the urinary passages from contiguous parts. When pus is in urine, it gives it a peculiar opacity, and on sedimentation the bottom of the vessel contains a somewhat ropy mass, presenting a wavy surface. It is to be distinguished from the phosphatic deposits mixed with mucus by the fact that it is not so white, and does not so closely resemble white or pinkish powdered chalk. Further than this, the phosphates are usually cleared up by boiling or by the addition of acid, but urine containing pus is not so altered.

The treatment of pyuria depends upon the cause of the presence of pus. If there is an abscess in the kidney surgical measures are required, but if the pus is due to a pyelitis or cystitis the use of substances which exercise a mild antiseptic influence is to be resorted to, at least for a time. For this purpose the patient may receive 5 grains of uritone or urotropin three times a day in sparkling water, or 10 grains of benzoate of ammonium three times a day in capsule. For the methods and drugs to be employed for irrigating the bladder the reader is referred to works on genito-urinary diseases.

Chyluria.—Chyluria is a condition in which the urine presents a milky appearance owing to an admixture of fat. It may occur in some cases of pregnancy and during lactation. In other instances it follows injury to the lymphatics of the abdominal cavity. The most common form of chyluria is that which comes on as a complication or symptom of infection by the parasite *Filaria sanguinis hominis*. This condition, as pointed out elsewhere, is met with most commonly in India, China, and in the Straits Settlements, and its cause is the obstruction of the lymphatics produced by the presence of the parasites within them. Not rarely urine, when chylous, coagulates in the vessel holding it or becomes gelatinous in appearance.

Phosphaturia.—This term is applied to a condition of the urine in which it contains an excess of phosphates and is supposed by some to be associated with unusual activity of the nervous system, particularly in connection with that degree of excessive nervous strain which is often productive of neurasthenia. Whether this view is correct is debatable. There can, however, be no doubt of the fact that in certain of the diseases characterized by great loss of flesh, such as tuberculosis, an excess of phosphates is present in the urine. Such a condition also arises in acute atrophy of the liver and in certain forms of grave anæmia. On the other hand, acute diseases running a febrile course, and supposed to be characterized by a great amount of tissue breakdown, are not accompanied by this manifestation. In some instances in which there is an excess of phosphates present in the urine the patient is also diabetic, and in still others the patient, while suffering from polyuria and phosphaturia, and who has such diabetic symptoms as thirst and loss of flesh, nevertheless does not develop a glycosuria, sugar being constantly absent. It has been thought by some that these cases of so-called "phosphatic diabetes" represent an early stage of true diabetes, for in some of them glycosuria ultimately develops.

The best remedy for the purpose of cleaning the urine of an excess of phosphates is benzoate of ammonium in doses of 10 to 20 grains three times a day.

Oxaluria.—Oxaluria consists in a condition in which urine of high specific gravity contains on standing, when decomposition is absent, an excess of calcium oxalate crystals. The condition is an important one in that it frequently points the way to the correct diagnosis and treatment of patients who are suffering from dyspepsia, nervous irritability, melancholy, and mental depression with a general condition of wretchedness. It is said to be present in those cases in which there is lack of free hydrochloric acid in the secretion of the stomach. It also develops in patients who eat pears, cabbage, tomatoes, and, occasionally, in those who take coffee to excess. In many patients it is an evidence of faulty metabolism due to lack of fresh air and exercise. The condition is of interest from a therapeutic standpoint because of the fact that these patients often gain great benefit if they receive moderate doses of nitrohydrochloric acid and take a fair amount of physical exercise and lead an out-door life.

Indicanuria.—Traces of indican, or, to speak more correctly, indoxyl sulphate of potassium, are present in normal urine, being derived from the indol which is formed in the intestine by the decomposition of proteids through the action of bacteria. If this indol is absorbed from the intestine into the blood, it is oxidized and forms the indoxyl sulphate of potassium just named.

When indicanuria is marked, it is an evidence of an excessive amount of intestinal putrefactive change, and the discovery of indicanuria in a patient who is suffering from the symptoms of autointoxication is, therefore, of value from a diagnostic standpoint. An estimation of this substance in the urine is also useful to differentiate intestinal obstruction from ordinary severe constipation, for in the former indicanuria is usually marked, and in the latter the trace of indican which is present is usually not above the normal.

In rare instances, owing to decomposition of the indoxyl sulphate of potassium before it escapes from the body, the urine is blue when it is passed, but in the majority of cases in which a blue urine has appeared it has been found that the patient has taken methylene blue or some similar aniline dye, either as a medicine or in foodstuffs. The presence of indican in the urine is determined by heating to the boiling point 5 c.c. of nitric acid in a test-tube and adding 5 c.c. of urine. If indican is present in excess, a bluish ring develops at the point of contact between the two fluids, and if 2 c.c. of chloroform are added and the liquids mixed by shaking, and the test-tube then set aside to stand, it will be found that the layer of chloroform which soon separates has a violet color.

Lithuria.—Under this heading is mentioned a condition in which an excess of uric acid occurs in the urine, chiefly in association with sodium and ammonium, and sometimes with potassium, lithium, and calcium. An examination of the urinary sediment under the microscope may reveal small, reddish grains or crystals, looking under the microscope like particles of red pepper. There is no condition which is so little understood at the present time as is this one. Almost every layman, and a multitude of doctors, continually speak of being "full of uric acid," meaning by this that they have muscular stiffness, or that the urine shows an excess of urates, or even uric acid crystals. In the majority of instances this excessive deposit of urates, or uric acid, depends not upon any abnormality in bodily metabolism, but upon conditions of the urine which cause the precipitation or deposition of these solids. There is either a condition of acidity or a minimum quantity of mineral salts, and as a result precipitation takes place. For this so-called "uric acid diathesis," physicians prescribe large quantities of lithium and copious draughts of water. There can be no doubt that the water is advantageous, but the lithium only does good until a certain degree of alkalinity is reached, when it is of little value, and if the doses are large it acts as a depressant to the general system. It is quite true that persons who eat heartily, drink alcohol, and take no exercise are not infrequently overloaded with effete materials representing imperfect metabolism, which cause disagreeable symptoms. It is also perfectly true that exercise, a proper diet, and the use of plenty of drinking water will overcome these symptoms, but this does not prove that the patient is a sufferer from the "uric acid diathesis."

Melanuria.—Melanuria is a condition in which the urine at the time it is passed, or shortly after its exposure to the air, becomes intensely dark in hue, owing to the presence in it of melanin. It is found in certain conditions in which this substance is produced in the body by pathological processes, such as melanotic growths. If a solution of ferric chloride is added to the urine, it becomes inky black. If caustic potash is added, it becomes at first violet and then claret colored, and if acetic acid is added to this mixture it may become blue. The test most commonly employed is the solution of ferric chloride mentioned.

Myelopathic Albumosuria.—The presence in the urine of the so-called Bence-Jones albumose or proteid has by a number of observations been shown to be most suggestive of the rather rare tumor of bone known as multiple myeloma or medullary osteosarcoma, "Kahler's Disease." The test is quite simple. The urine, if not distinctly acid, should be made so with acetic acid and then heated. At 50° C., if it contains this albumose, it becomes milky; at 60° C. it deposits a thick precipitate which clings to the sides of the tube or collects on the surface. Further heating to 100° C. causes the almost complete disappearance of the precipitate, which, however, re-forms as the urine cools.

DISEASES OF THE DUCTLESS GLANDS AND LYMPHATIC SYSTEM.

DISEASES OF THE THYROID GLAND.

GOITRE.

Definition.—Under the term *goitre, bronchocele, thyreocele,* or *struma* is included almost all enlargements of the thyroid gland, but the application of so general a term is not to be regarded with favor. It is better, therefore, to consider the various diseases of the thyroid as inflammatory (traumatic strumitis), infectious (tuberculous or syphilitic strumitis), and parasitic (echinococcic) strumitis. Of the remaining so-called hypertrophies of the gland it may be said that some of them at least are apparently not of the nature of neoplasms, and to these the term "*simple*" or "*benign goitre*" is applied; whereas, in the case of those enlargements which are due to the development of tumors within the gland, we apply the term "*neoplastic goitre,*" as, for example, endotheliomatous, sarcomatous, and cancerous growths (malignant goitre). (See Tumors of the Thyroid.)

With regard to some of the simple or benign goitres, it is still unsettled as to whether they are neoplastic, that is, adenomatous, in every instance. In some cases they are undoubtedly adenomatous. In still other cases the greatly enlarged gland may, under the microscope, have a histological formation like that of the normal organ. To this type of goitre the name "hyperplastic goitre" is applied, including those varieties characterized by reproduction of the parenchyma of the gland and called by some writers "parenchymatous goitre." When the enlarged gland contains cysts developing from its acini, the mass is called a "cystic goitre." Cystic goitres result from the distention of the gland spaces, with absorption of the intervening walls, thereby giving rise to cavities of various sizes. The contents of these cysts may be gelatinous or colloid. In some instances the enlargement depends upon dilatation of bloodvessels, and apparently upon the overgrowth and dilatation of new bloodvessels, forming the so-called vascular goitre, and not rarely there is a blending of two or more of the types already mentioned. Such conditions are known as "mixed goitres."

In ordinary goitre there takes place an increase in the size of the alveoli in the thyroid gland and a simultaneous formation of new glandular tissue. Side by side with this increase there is often colloid degeneration, and when this degenerative process is marked the state is called colloid struma or colloid goitre. Not uncommonly still other degenerative changes take place, in which the walls of the alveoli break down, and in this way several cavities are thrown together, forming cysts, which may hold colloid matter and blood derived from the vessels in the alveolar walls. This is called cystic goitre. In still other cases the bloodvessels of the gland become dilated, so that a telangiectatic state develops. Finally, it sometimes happens that all of these changes take place in the same gland, and upon them may be superimposed acute inflammation and even malignant growth. The increase in size may be limited to a single part of the gland or be widely diffused.

Etiology.—The cause of ordinary enlargement of the thyroid gland of the fibroid and cystic type is unknown, but there can be no doubt that it depends, at least in part, upon the character of the drinking water used. My colleague, Professor Keen, has just investigated a very remarkable prevalence of the disease in the interior of the State of Pennsylvania, in which the relationship of water supply and goitre is extraordinary, a very large number of the people on one side of a mountain ridge being affected, and those across the divide escaping. The disease is also said to be very common in some parts of Michigan and in Switzerland. The suggestion of Grasset that goitre is of protozoal origin has not been favorably received. In support of his view he calls attention to the fact that, like malaria, goitre is endemic in certain areas, and he is inclined to believe that the thyroid enlargement is analogous to the splenic tumor of chronic malarial infection. The disease is more frequent in women than in men, and in adults than in children, in which class it is very rare.

Symptoms.—The symptoms of goitre are usually of no consequence until the growth is large enough to be seen or until, by its pressure on the adjacent tissues, it causes difficulty in breathing and interferes with swallowing or with the function of the vagus nerves. The goitre may involve all of the gland, its isthmus, or either one of the lateral lobes. Rarely aberrant goitres arise in ectopic or misplaced thyroid tissues, and they may be intrathoracic or occur at the base of the tongue (lingual goitre). In still other instances they have been known to develop along the course of the thyroglossal ducts or in adjacent areas. The degree to which the cervical tissues are displaced in those cases in which the growth is chiefly in one lobe is remarkable. Not long since I sent to Dr. Keen for operation a patient whose hyoid bone was pushed to one side so that it rested nearly under his right ear.

Treatment.—There is no medicinal treatment of much value in goitre. Painting the part with iodine and the use of various counterirritant ointments have been resorted to, but they have no real effect over the growth. If it becomes very large, it must be excised if the pressure symptoms are severe.

SWELLING OF THE THYROID.

This occurs from two chief causes, namely, from inflammation and from hyperæmia or congestion. Some writers have described an angioneurotic form. When the swelling is due to inflammation it may arise from infection of the gland, as in typhoid fever or other acute infectious diseases, and may follow vaccination or sepsis; or, again, it may be due to tuberculosis of the gland, or to syphilis with the formation of gummata. Occasionally marked swelling arises from trauma, and this may be acute or chronic.

It is asserted by Fothergill that there are recorded five cases of enlargement of the fetal thyroid due to the administration of potassium chlorate to the mother.

Some years ago I reported the case of a woman who, in stooping in a dark room, struck her neck against the edge of a chair and at once felt violent pain in the thyroid gland. The gland rapidly became swollen, and the patient presented all the symptoms seen in persons to whom large doses of thyroid gland have been given, such as headache, a rapid pulse, and a tendency to syncope. In still another case, seen by me, an army surgeon on the fighting line received a severe blow in the thyroid, and developed a chronic enlargement of the thyroid gland, with some tachycardia.

The thyroid gland is also found enlarged by hyperæmia in young girls, particularly at the menstrual period, and in young women in their first pregnancy. It also occurs in young persons who suffer from cardiac disease. Such an enlargement usually passes away when the cause is removed.

Sometimes thyroiditis occurs in the insane, but its etiology and symptomatology need further study.

TUMORS OF THE THYROID GLAND.

The tumors of this gland are adenoma, in which state the condition is practically that of goitre as already described, carcinoma, sarcoma, and endothelioma. The sarcomata are usually primary. Morf has been able to collect but 39 instances of carcinoma of the thyroid; he himself adds 1 case. Of the 173 cases of cancer of the thyroid collected by Orcel, 14 invaded the trachea.

Carcinomata are also usually primary and undergo metastasis to nearby tissues or even to distant structures. The growth may develop in the parenchyma or in the connective tissue of the gland.

An interesting form of tumor of the thyroid is the so-called carcinosarcoma, or mixed tumor of this gland.

Occasionally old, quiescent goitres may become malignant, undergoing either sarcomatous or carcinomatous degeneration.

An anomalous condition in this group of affections is thyroid metastasis, consisting in the growth of typical thyroid tissue at points distant from the glands. These growths are quite commonly in bones and are seldom malignant. The thyroid itself may show no change.

EXOPHTHALMIC GOITRE.

Definition.—Exophthalmic goitre is often called "Basedow's disease," "Parry's disease," or "Graves' disease." Parry described it (1825) ten years before it was described by Graves (1835) and fifteen years before it was described by Basedow (1840). Exophthalmic goitre is an entirely different disease from ordinary goitre or simple enlargement of the thyroid gland. It is a malady in which, as its name implies, there is protrusion of the eyeballs, and, in addition, palpitation of the heart, with a very rapid pulse. There are fine tremors in the hands, arms, and head, and disordered vascular tone. A tendency to abnormal sweating of the palms of the hands, and great mental depression is often present.

Satterthwaite recognizes acute, subacute, and chronic forms of Graves' disease, and says we may speak of acute or temporary and essential or chronic forms. A secondary form is that in which an old goitre takes on the symptoms of Graves' disease.

Frequency.—In 10,603 cases admitted to the Jefferson Hospital, there were 11 cases of exophthalmic goitre, or 1 in 964. In the University Hospital, out of 35,076 cases, there were 48 cases of exophthalmic goitre, or 1 in 730. Of 7270 cases treated at the Dispensary for Nervous Diseases at the Orthopædic Hospital and Infirmary for Nervous Diseases, Eshner found that 30 were exophthalmic goitre, or 1 in 242. It is therefore by no means a rare disease.

Etiology.—The cause of exophthalmic goitre is not known, but, as is the case with most diseases of obscure causation, a multitude of factors have been named as possible causes for its development, varying all the way from rheumatism and tonsillitis to fright and traumatism. The disease is very much more frequent in women than in men. Thus, out of 1839 cases collected from various sources, 1553 were females and 286 males, a proportion of about 6 to 1, and its average age incidence is between sixteen and forty years. Cases are on record in which it has affected children as young as two and a half years. In some statistics, collected by me some years ago, it was shown that there is a very distinct hereditary influence present in many cases. There can be little doubt that the symptoms which are present in exophthalmic goitre are dependent upon excessive internal secretion of the thyroid gland, or, if not upon excessive secretion, to the entrance into the general system of more of the active principle of the thyroid gland than is normal. It has been claimed that the cause lies in disorder of the sympathetic ganglia, but there is no adequate proof of the correctness of this view.

Pathology and Morbid Anatomy.—So far as the morbid changes are concerned, there is an excess of fat in the orbit as compared to the quantity of fat in other portions of the body.

The heart may be normal or dilated. Not rarely there is some undue relaxation of the sphincteric fibres around the mitral orifice of the heart. The thyroid is enlarged, the veins covering it are dilated and numerous, and the arteries supplying its tissues are enlarged and tortuous. On the other hand, certain observations have shown that in many instances there is no remarkable change in the vascularity of the organ.

A microscopic examination of the thyroid gland reveals, in many instances, however, a very marked increase in the development of its parenchyma, and the epithelium lining its vesicles is changed in appearance. The colloid substance in the vesicles partly or entirely disappears and is replaced by mucoid material which takes a stain badly. The epithelial cells lining the vesicles frequently desquamate. Greenfield has shown that additional tubules lined by a layer of epithelium are also developed. After the disease has lasted for a considerable period of time, there is not infrequently an overgrowth of connective tissue in the gland. MacCallum finds that, as a whole, the changes resemble most closely those found in experimentally produced compensatory hypertrophy of the thyroid.

The thymus gland is enlarged in a very considerable proportion of cases, but a microscopic examination of it does not show pathological changes in most instances.

The parathyroids are often found to be atrophied and the thymus gland is persistent and often hypertrophic.

Certain alterations have been described in the sympathetic system and in the central nervous system, but it has not been proved that these have any close relationship with the disease.

Symptoms.—The *protrusion of the eyeballs*, even when it is present to a moderate degree, is so striking that attention is directed to this symptom almost as soon as the patient is seen. It varies greatly in degree. In some cases the eyeballs may be so prominent that it is impossible for the lids to completely close, whereas in others the exophthalmos may be very slight indeed. While it is true that the exophthalmos may not be equally developed on both sides, it is nearly always bilateral, although a few instances of unilateral exophthalmos have been reported.

Under the name of *von Graefe's sign*, a condition is found, in which, if the patient is directed to look at the floor, the upper eyelid does not follow the eyeball in its downward rotation as rapidly as it should. This symptom is not always present. *Stelwag's sign* consists in a widening of the palpebral fissure, with retraction of the lids, to such an extent that the sclerotic coat of the eye is seen above and below the iris. This very distinctly increases the exophthalmic effect. With this symptom there is also diminished reflex excitability, so that winking is delayed when a sudden movement is made toward the patient. Under the name of *Möbius' sign* is described a condition in which there is a lack of power of convergence, so that if a pencil is brought near the patient's face the eyes cannot converge, as they do in a normal individual.

Notwithstanding the exophthalmos, vision, as a rule, is not interfered with, but ulceration of the cornea sometimes occurs as the result of the inability of the eyelids to protect the eye. (See Fig. 100.)

The increase in the size of the thyroid gland is usually not very great, and it never becomes as large as in many cases of ordinary goitre.

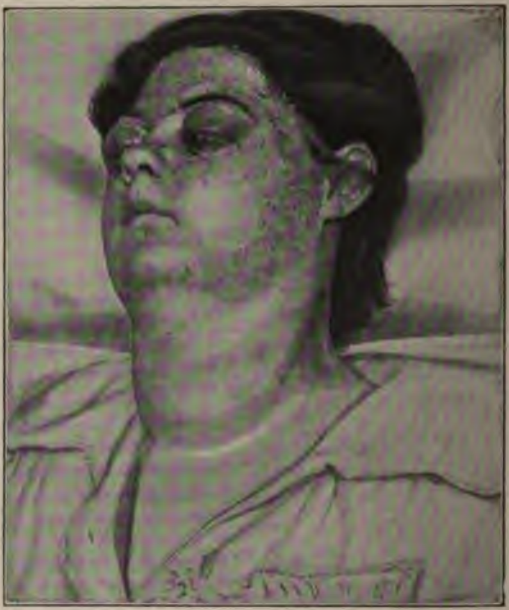
On inspection the gland may seem to pulsate, and on palpation it often transmits a *thrill* to the finger-tip. If the stethoscope be placed over the gland, a distinct *humming murmur* can be heard. This same murmur is also detected, even more clearly, if the stethoscope be placed over the carotid arteries.

The gland is never very hard, but may be soft and fluctuating, and is apt to vary in size considerably from week to week or from day to day. *Tachycardia* is practically always present in these cases. The *pulse* varies from 90 to 100 or even 200 beats a minute, the ordinary rapid pulse being very much increased on exertion or excitement.

If the finger-tips of the patient are rested upon the finger-tips of the physician, there can be felt not infrequently a *fine tremor*, sometimes called "railroad bridge tremor," owing to its resemblance to the sensation produced in one's feet when standing upon a railroad bridge during the time a train is passing over it. In other instances the tremor is very marked and coarse in character.

The patient nearly always complains of *feebleness* and *mental depression*, and in some cases *melancholia* may be so profound as to result in suicide, as in a patient recently under my observation. An irritating *nervous cough* and occasional attacks of *dyspnœa* may occur, and cases are on record in which the patient has suddenly died from urgent dyspnœa, which has had its origin in intense swelling of the thyroid gland, so that it has pressed upon the trachea in much the same manner as does the enlarged thymus in *status lymphaticus*. Another symptom which, when it develops, must always be regarded with alarm, is excessive and *obstinate vomiting*. This condition may speedily pass by, but in some cases it has persisted until death has ensued. Sudden attacks of *diarrhœa* are not rare. The digestion is fairly good, but the appetite is uncertain, and the patient craves abnormal things as she does in hysteria. Oftentimes a constant "rifting up" of wind gives great annoyance. The *nervousness* is so *intense* that in many cases the patient's life is almost unbearable because of the irritation produced by noises and other sources of irritation which ordinarily would not be noticed. Nervous chills or tremblings are often a source of great annoyance to the patient, and the palms of the hands

FIG. 100



Exophthalmic goitre. The illustration shows the enlarged thyroid gland. The exophthalmos was so great that the lids had to be sewed together to protect the eyes.

are prone to be wet with excessive perspiration. Patches of pigmentation on the skin often develop. There is usually distinct loss of weight and strength.

Prognosis.—A certain number of cases of exophthalmic goitre undoubtedly recover, but they are always liable to relapse. Rapid loss of flesh and strength, marked tachycardia, persistent vomiting, and diarrhœa are all of them symptoms which would cause us to give a guarded or unfavorable prognosis. Whereas, if the symptoms are mild, we have a right to feel correspondingly encouraged. Sometimes exophthalmic goitre may last so short a time as a few days or weeks. In other instances it may continue for many years.

Treatment.—The treatment of exophthalmic goitre is not very satisfactory. When the thyroid gland was first used as a therapeutic agent it was given to a considerable number of persons suffering from exophthalmic goitre, with the idea that it might do good, but from the first it must have been manifest that it could not be of value because the patient is suffering from too much thyroid secretion, and the addition of more of the thyroid substance must in consequence be disadvantageous.

Lépine has recommended antithyroid serum in the treatment of Graves' disease. This serum is obtained from animals immunized against hyperthyroidism. Under the name of thyroid serum Moebius has given us serum derived from sheep from which the thyroid gland had been removed six weeks before. The dose is 1 to 5 c.c. three times a day, given by the mouth. A similar preparation is prepared in this country by Parke, Davis & Co., and is called "Thyroidectin." The dose is 5 to 10 grains t. i. d., in capsules.

In most instances the patient should receive a carefully carried out rest cure, extending over a period of from four to six weeks, with regulation of the diet and massage and electricity. Sometimes a course of hydrotherapy is advantageous, and change of air and scene is particularly valuable if the patient has been subjected to nervous stress. All forms of exercise or gayety which tend to exhaust the nervous system should be forbidden.

The drugs which seem to be of most benefit are those which belong to the class of sedatives. When the heart's action is very excessive, I have known full doses of tincture of veratrum viride to be most advantageous, in that they quiet the heart not only by depressing its muscle, but also by stimulating the pneumogastric nerve. In other instances the bromides may be employed. In still other cases I have seen gelsemium employed to advantage.

The disadvantage of opium or morphine, for the production of nervous quiet, is the danger of establishing the "habit." This is a very real danger in these patients, because of their lack of nervous equilibrium. Sometimes belladonna may be given with advantage to quiet the circulatory and nervous excitement.

Within the last twenty years a very large number of operations have been performed upon patients with exophthalmic goitre, with the object of curing the disease. In some instances the thyroid arteries have been tied. In others the capsule of the gland has been stripped from it and made fast in the wound, so that the tissues will shrivel. In still another class of cases the cervical sympathetic has been cut, and Jaboulay has strongly advocated this measure. The value of operative procedure is, however, well summed up by my colleague, J. Chalmers Da Costa, who says:

"Treat most cases medically and by rest; if medical treatment fails, consider the advisability of surgical treatment.

"Do not operate if there is great hysteria; if the gland is very large, thyroidectomy will fail; if the gland is very small, it will do no good to remove it.

"If the symptoms are urgent, if the goitre is distinct, but not excessively large, if it has relapsed under medical treatment, or if the patient refuses to submit to the necessary restrictions of medical treatment, perform thyroidectomy.

"Take Kocher's advice, and do not promise cure, but realize that the patient may die or there may be a partial cure.

"When thyroidectomy is performed do not remove the entire gland. Remove one lobe only, or one lobe and a half or two-thirds of the remaining lobe. Even so-called complete thyroidectomies are not often really complete, as a remnant of the processus pyramidalis is usually left behind. In addition to removing a part of the gland, take Kocher's advice and tie three of the four thyroid arteries.

"Do not give a general anæsthetic, but produce local anæsthesia (Kocher). A general anæsthetic is very dangerous in goitre operations."

In advanced cases with threatened suffocation, it may be necessary to do tracheotomy. If the goitre is small, if the symptoms are marked, and do not yield to rest and medical treatment, and the patient refuses thyroidectomy, we can perform bilateral resection of the cervical sympathetic ganglia.

MYXŒDEMA.

Definition.—Myxœdema is a disease in which extraordinary nutritional changes take place in the body as the result of absence, atrophy, removal, or inactivity of the thyroid gland. It is characterized by a peculiar swelling of the subcutaneous tissues, by falling of the hair, by mental failure, and by feebleness of the circulation. Myxœdema is closely related to cretinism in children. It is sometimes called "Athyrea" and "Gull's disease."

Etiology.—The cause of myxœdema is failure of the body to receive the normal quantity of secretion from the thyroid gland. In this sense it may be considered the antithesis of exophthalmic goitre. The cause of the atrophy or inactivity of the gland is unknown.

Myxœdema occurs more frequently in married than in single women, and it appears most commonly after thirty years of age. It affects women and men in the proportion of 6 to 1.

Pathology and Morbid Anatomy.—When the thyroid gland undergoes atrophy, when it is removed by surgical operation, and when, as the result of a specific infection, as actinomycosis or morbid growth, its function is destroyed, myxœdema ensues. In most cases the atrophy of the thyroid gland can be readily recognized, but in others the gland may seem larger than normal. This increase in size may be due to infiltrations, tumors, cysts, subacute or chronic inflammatory processes leading to an overgrowth of the connective tissue of the gland, and is not a sign of any actual increase in glandular structure.

The state of the subcutaneous tissues is very remarkable. They are puffy and swollen, and if incised are found infiltrated with a mucoid or jelly-like material. This material is present in such excessive quantities that the cutaneous glands are pressed upon and their nutrition interfered with, so that the skin becomes dry and harsh, and the hair falls out. Nor does this process of infiltration cease with the involvement of the subcutaneous tissues, for in the liver and in the kidneys the cells are pushed apart and compressed. The kidneys are larger than normal, and considerably toughened in texture.

Frequency.—Myxœdema is a rare disease, particularly in the United States. Physicians connected with large hospitals may see a case only once in many years.

Symptoms.—The symptoms of myxœdema are *infiltration of the tissues* of the entire body, so that they appear at first glance to be dropsical, but they do not "pit" on pressure and are quite firm and resistant. The *skin is dry*, *pallid*, and *poorly nourished*, the *hair falls* till only a few strands are left, and the eyebrows disappear. The *expression is altered* by the obliteration of the facial lines, and by the *stupidity* from which the patient suffers, for a form of *mental inertia* develops. As the disease advances *muscular feebleness* often arises, so that the patient falls, and there may be difficulty in holding the head erect. The *temperature* is slightly *subnormal*, the *heart* is *feeble*, and *albuminuria* is sometimes present.

Prognosis.—The prognosis depends entirely upon the treatment. If no specific treatment is resorted to death invariably ensues as a result of general asthenia or from some intercurrent malady. If specific treatment is adequate recovery usually takes place, provided the patient is not seen until after the disease is very far advanced.

Treatment.—Aside from the use of antitoxin in diphtheria, there is no therapeutic measure which produces such extraordinary results and acts in so specific a manner as the administration of thyroid gland in myxœdema and in cretinism. The dried thyroid gland of the sheep should be given the patient in gradually ascending doses, beginning with 2 grains in capsule twice or thrice a day, and gradually increasing the amount until 10 to 15 grains are taken daily, provided the patient does well on these doses and seems to need large quantities. When the extract of the thyroid gland is used, the dose is ½ grain three times a day to start with. Meltzer states that the extract and dried gland prepared by Parke, Davis & Co. have given him the best results. When overdoses are taken symptoms of cardiac weakness develop. These are dangerous and should be controlled by the use of strychnine and by insisting that the patient remain in bed for several days. Indeed, rest in bed is the safer plan whenever ascending doses are being employed. After thyroid gland has been given long enough to cause great improvement, so that the patient is practically well, it is essential that about one-half the dose be continued indefinitely or at certain periods, in order to prevent a relapse, for the sheep's thyroid must take the place of the wasted gland in the neck.

As these patients are very susceptible to cold, they should be carefully clothed and sent to a warm climate in the winter months, if possible.

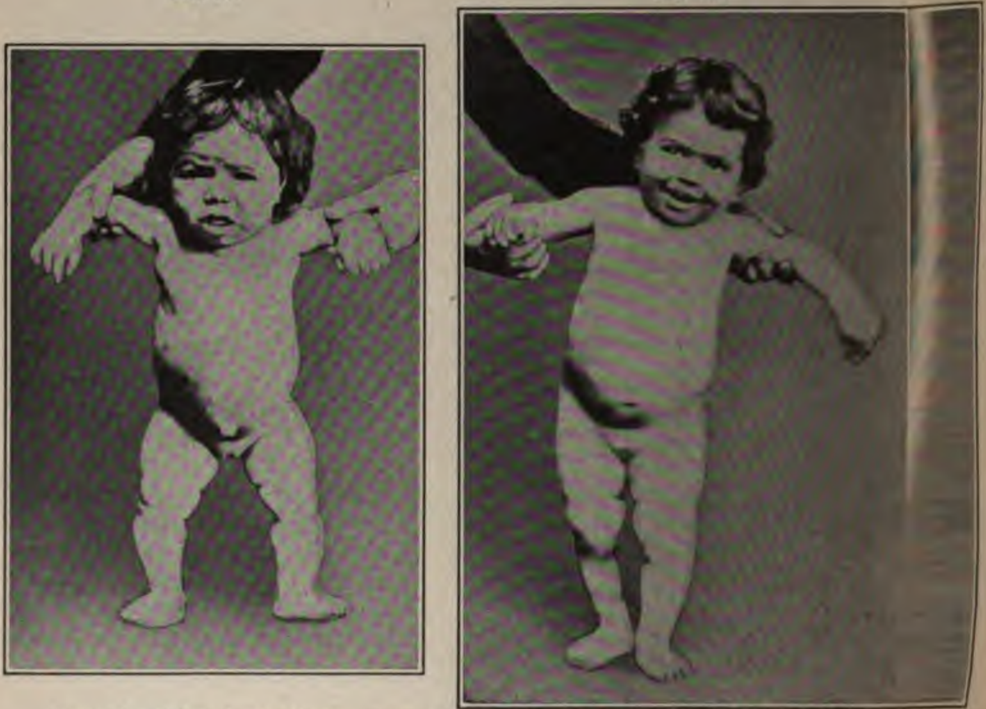
CRETINISM.

Definition.—Cretinism is sometimes called congenital myxœdema, or the myxœdema of childhood, and depends upon the same causes as does myxœdema, in that the curious systemic changes which develop in the patient are the result of an absence of the secretion of the thyroid gland.

Cretinism occurs in two forms, as endemic cretinism and sporadic cretinism.

The conditions leading to thyroid absence or inadequacy are not known. It is true that in some instances the results of the marriage of near relatives or the presence of a tuberculous history has seemed to indicate that there might be some connection between these factors and the development of the disease, but in most cases these causes are absent.

Fig. 101 Fig. 102



A case of cretinism, showing the improvement produced by the administration of thyroid gland. (Davisson's case.)

Symptoms.—The symptoms of cretinism rarely develop before the end of the second year, but the symptoms may be noticeable from the time the child is twelve months of age, when the parents usually consider that the child is somewhat "backward." At this time it is found to be *stunted* and *mentally dull*. The *head* and the *hands* and *feet* may seem *unduly large* in proportion to the size of the trunk and limbs. The face is *stupid* and *heavy*, and the *eyes dull*. The *palpebral openings* are *narrow* and *elongated*, and the *nose* is *broad* and *flat*, with heavy nostrils. The *lips* are *coarse*, are apt to protrude, are usually held apart, and not infrequently there is a good deal of *dribbling of saliva*. The *tongue is swollen*, and there seems to be some weakness of the cervical muscles, so that the head is not well carried on the shoulders. An *anteroposterior curvature* of the spinal column is often present, so that the abdomen is very much protruded. The *legs* are *short* and *bent*, as in rickets, and the *skin* is *sallow* and *greasy*. The hair is scanty and brittle, and the skin is badly nourished. The *temperature* is *subnormal*, but there are no important changes in either the urine or the blood. The most marked alterations from normal in the blood are a diminution in the quantity of hæmoglobin. In many instances the child has little more intelligence than that which is needed to take its food. In other cases it is vicious and dirty.

An autopsy in a case of cretinism usually reveals an absence of the thyroid gland, its place being taken by a few fatty granules, or by a fibrocystic growth. On opening the skull there is found to be an excess of intraventricular and interarachnoid fluid.

Diagnosis.—There is no difficulty in diagnosticating cretinism if a typical case is presented. Given a patient suffering from rickets and idiocy, some resemblance to true cretinism may be present, but the state of the skin and hair and the absence of the thyroid gland in true cretinism render a separation possible.

Prognosis.—The prognosis in cretinism is very good, even better than in the myxœdema of adults, provided the treatment is instituted while the patient is yet a child in years. If the patient has survived till adult years are reached, the results are not so satisfactory and extraordinary.

Treatment.—The treatment consists in the administration of thyroid gland or thyroid extract, beginning with ¼ of a grain of the extract three times a day and gradually increasing it, or using 1 or 2 grains of the dry gland once, twice, or thrice a day, according to the size and age of the child. Under the administration of these substances the most remarkable change takes place in the patient. The first noticeable alteration is a great decrease in body weight, with a similar decrease in the bulkiness of the child. The skin becomes more moist and appears better nourished, and the expression improves. There is also an improvement in the color of the skin and in the quantity of hæmoglobin in each blood corpuscle. Still later, after this primary decrease in weight, a real improvement in nutrition takes place, and the child begins to gain, so that it no longer looks stunted, but appears more like a healthy individual. The mental improvement is perhaps the slowest part of the cure, and in some instances the mind never fully reaches the development of that of a healthy child, although the nutrition of the body may be excellent. The effect of the administration of thyroid upon the growth of the teeth is equally remarkable with that upon the general nutrition. Before thyroid is given the milk teeth are usually badly formed and rapidly decay; but if thyroid is administered freely before the permanent tooth appear, they are often developed as they would be in the jaws of a healthy child.

DISEASES OF THE SUPRARENAL GLANDS.

ADDISON'S DISEASE.

Definition.—The name Addison's disease is applied to a condition in which the patient suffers from a characteristic pigmentation of the skin, pallor, and loss of strength, and in which the chief microscopic changes are alterations in the suprarenal bodies.

History.—Addison's disease gets its name from Thomas Addison, the physician who first clearly described the malady in 1849 at Guy's Hospital, London. The condition did not receive attention from the profession in general until 1854, when Addison wrote a special monograph on the subject.

Etiology and Pathology.—The cause of Addison's disease is not known in the sense that we recognize a cause which is responsible for all cases. In about 50 per cent. of the cases so far reported which have come to autopsy, tuberculosis of the suprarenal glands has been found. That this lesion is not sufficient in all cases to cause the general systemic manifestations of the disease is proved by the fact that identical changes have been found in the suprarenal bodies when none of these symptoms have been present. In certain cases hemorrhages into the suprarenal bodies as the result of injuries have caused the symptoms to develop.

As a matter of fact, the view, as to the relationship of these causes to the disease, expressed by Addison fifty years ago is probably correct, namely, that any lesion of these bodies which interferes with their function may cause the malady.

In some instances the disease seems to be primarily the result of pathological changes in the semilunar ganglia of the abdominal sympathetic nervous system. Rolleston has expressed the plausible view that in these cases the disease arises in all probability by reason of the fact that the glands are cut off in circulation and nerve supply by growths or inflammatory exudates.

Morbid Anatomy.—The common lesion in cases of Addison's disease is, as already stated, tuberculosis, and next to this in frequency is atrophy. The tuberculous change is of the fibrocaseous type, except in rare instances, and usually begins in the medulla of the gland. The stage of infiltration is followed by caseation and commonly more or less fibrosis; in other words, there is an attempt at healing. The fibrocaseous area may be restricted to the adrenal or extend beyond it. Calcification is not uncommon, and pyogenic infection may cause abscess. The tuberculous process may be primary and confined to the adrenal body, as in the two cases reported by Symes and Fisher.

In the form of the disease in which atrophic changes occur in the glands, the wasting may be so complete that only a small fibrous mass remains to indicate their former existence. In still others an overgrowth of fibrous tissue resembling the sclerosis found in the other organs of the body may take place, with secondary atrophy of the parenchyma. Peterson has collected 26 such cases. In still other cases the glands have been found to be the seat of hemorrhage (adrenal apoplexy), thrombosis of the vessels, or malignant disease. The changes found in the semilunar ganglia and in the plexuses composing the abdominal sympathetic system are, as already stated, in all probability, indirect causes of the disease, although Hale White has shown that changes take place in these tissues in ordinary individuals as the result of advancing age. It is difficult to determine, therefore, whether the reports of changes in these tissues made by some observers have been really etiological factors in the disease. Further, a very large proportion of cases in which Addison's disease has been present have failed to show alterations in the semilunar ganglia or in the abdominal sympathetic. In some cases a hyperplasia of the lymphoid structures in the alimentary canal has been noted.

Finally, it is not to be forgotten that Addison's disease may be present without noticeable lesions in the suprarenal bodies, and it is also a fact that these bodies may be almost completely destroyed by a growth or by tuberculosis without any symptoms of this malady developing.

The pigmentation of the skin is due to the deposition of pigment in the cells of the Malpighian stratum, and according to Earkshevitch, who studied the skin removed from a living subject, in the subjacent tissues. The pigmented cells are supposed to obtain their pigment from the hæmoglobin of the blood, but they contain no iron. The discoloration of the mucous membranes is due also to the deposit of pigment. The pigmentation of the mucous membranes is in patches, and Mann asserts that it is deposited only where the parts are rubbed or subjected to causes that produce hyperæmia.

That there have been, and are in existence at present, several theories as to the lesions which result in Addison's disease must have been evident from what has already been said. The only ones that have received general recognition have been the "nervous theory," that the disease was due to changes in the abdominal nervous apparatus; or the original theory of Addison, that it is due to failure of the suprarenal glands to carry out their normal function. The nervous theory has now been generally cast aside, and we have left Addison's own proposition modified by our extended knowledge of internal glandular secretion. Space does not permit a discussion of the views for and against this opinion. These can be found exhaustively, and most capably, discussed by Rolleston in Allbutt's *System of Medicine*, vol. iv. Suffice it to say, that the opinion generally held to-day is that these symptoms come on because the suprarenal secretion fails to find its way into the general economy. Changes in the sympathetic nervous system may also be a factor.

Symptoms.—The symptoms of Addison's disease are chiefly those which are represented by the term *general asthenia*. The patient gives the history of being *easily tired* and, indeed, of a *constant sense of fatigue*. Even after a night's rest he feels as weary in the morning as when he went to bed. The sensation of feebleness is associated with profound muscular weakness as the disease progresses, but there is little or no true emaciation. The term "*invincible languor*" used by Rolleston well describes the patient's state. An examination of the heart shows its muscle is greatly enfeebled, so that the *cardiac* sounds are *lacking in normal* tone. The *pulse* is *soft,* and easily extinguishable by the pressure of the finger. The *extremities are cold* and the general body *temperature* may be *subnormal. Anæmia* is well marked, but usually not excessive, the blood cells being decreased to 3,000,000 or a little lower.

No mention as yet has been made among this list of symptoms of the one characteristic manifestation of the malady which is practically pathognomonic, namely, the *pigmentation of the skin.* While its peculiarities would naturally lead one to speak of it first, mention of it has been delayed because its appearance is often delayed until the other symptoms are quite well developed. In other words, it usually follows and does not precede the constitutional manifestations of the disease. There are, however, rare exceptions to this, and cases have been recorded in which the pigmentation has been present for long periods before any other signs of Addison's disease developed. The pigmentation may be over the entire body, but as a rule it is in patches, and chiefly affects the *skin of the face,* of the *neck,* and the extensor *surfaces of the hands* and *forearms.* If the *mucous membrane* of the *mouth* is examined the lips at the point of contact are noticeably darkened and the edges of the tongue, particularly on its under surface, may show discoloration as if ink had been taken into the mouth.

Diagnosis.—Except in well-defined cases it may require weeks or months of watching to determine that a patient has this malady. Pregnancy not rarely is associated with the presence of pigmented spots on the skin, but the condition of the uterus and its contents prevent a mistake being made as to the cause. In some cases of hypertrophic cirrhosis of the liver there may be in addition to jaundice very marked pigmentation. I have under my care at the time this is written a man who has hypertrophic cirrhosis, jaundice, and such deep pigmentation of the skin that he looks as if coated with coal-dust. In such a case the state of the liver reveals the cause.

In the rare malady called *diabetes bronzé,* in which there is hypertrophic cirrhosis, jaundice, diabetes, and pigmentation of the skin, the state of the liver and urine will aid in the differentiation. In certain cases of advanced pulmonary tuberculosis the skin is pigmented a dirty brown, and in patches may be considerably discolored, but here the pulmonary state prevents confusion as to its cause.

The prolonged use of arsenic may have a similar effect, not only on the extremities, but on the skin of the chest and abdomen, which may become much darker than normal without any neuritis being present. In syphilitics the site of old eruptions may be stained, and in vagabonds who are infested with lice, and have been much exposed to the weather, areas of discoloration of the skin may be present. It is said that the discoloration of the skin called chronic argyria, due to the prolonged use of silver internally, has been confused with the state of the skin in Addison's disease. This would be scarcely possible if the observer had ever seen a case of chronic silver poisoning, for the discoloration of argyria is a peculiar lividity rather than a pigmentation, and it is uniform on exposed parts of the body.

As Addison's disease is due in a large proportion of cases to tuberculosis of the adrenal bodies, the tuberculin test may be employed to give additional information in the case; but even if this test is positive the possibility of tuberculous foci elsewhere giving the reaction, and the fact that syphilitics sometimes react, should make us hesitate before resting too heavily upon this means of diagnosis.

Prognosis.—It may be stated that given a patient with Addison's disease developed so far that a diagnosis is certain, then death from the malady is certain also. Lewin in a collection of 500 cases found that 5 were cured and 28 improved. In the 2 cases I have seen, 1 of which is now under observation, the disease had, at times, certainly been arrested in its progress under the use of full doses of suprarenal gland. Because of the gradual development of the malady, its average duration is difficult to determine, but its course is not very rapid. Wilks believes it to be about eighteen months, but in some cases it lasts for years. Very rapidly fatal cases are also on record.

Treatment.—The question of the proper plan of treatment of Addison's disease cannot be answered positively until the pathologist is able to give us a clear conception of the morbid processes which produce the chain of symptoms already described. The very fact that different pathological changes, or diseases, affect the suprarenal bodies, and so produce the symptoms of this malady, indicates that the therapy must vary with the cause. Theoretically the use of suprarenal gland of the sheep is indicated in every case, but practical experience has shown that only in a small proportion of those cases in which it has been used has it done good. It is not, therefore, to be compared to the value of thyroid gland in myxœdema or cretinism. It has been proved that suprarenal gland has little effect on blood pressure if it is taken by the stomach, and that its active principle when in concentrated form often causes abscess when it is given hypodermically. Probably the best plan is to give the patient adrenalin chloride in normal salt solution by hypodermoclysis every day or every other day, using 1 to 2 drachms of the adrenalin chloride (1:1000 solution), as put upon the market, to a half-pint or pint of saline fluid. When the desiccated gland is used by the mouth, from 2 to 10 grains may be given three times a day in capsule. It is unfortunately true that even when the adrenalin gland is freely used, the disease is, at the best, only delayed in its progress in most cases.

Adams has collected 97 cases treated with suprarenal gland. Of these 7 were made worse, 43 experienced no real benefit, 31 showed marked improvement, and 16 were said to be cured. In the successful cases the gland was given solely by the mouth. If the patient is at all feeble he should be kept in bed, not only because in this manner we conserve his flagging energies, but also because a number of cases of syncope and sudden death from this disease have occurred in patients who have made a sudden effort. An easily digested diet, the avoidance of purgatives which may induce dangerous purging, and the use of iron and arsenic as tonics form the rest of the treatment.

DISEASES OF THE SPLEEN.

Diseases of the spleen occurring independently of other diseases may be said not to exist. In myelogenous leukæmia, in splenic anæmia, in malarial fever, and in cases of hepatic cirrhosis or heart disease, the spleen is often greatly enlarged, but in no case is this condition primary. So, too, in the prolonged infectious fevers, such as typhoid fever, the spleen is usually swollen.

In some cases the surface of the spleen may be traversed by a crevice or indentation which almost divides its body into different parts, and in others there may be found an accessory spleen or accessory spleens in nearby parts of the abdominal cavity. In very old people the spleen is often greatly atrophied.

An *infarct* of the spleen is due to an embolus which usually has its origin in the heart, or which arises from some area of septic infection, or in other cases a thrombus forms in the splenic vein and produces a similar effect. The latter condition is the cause of the infarct met with in typhoid fever and in leukæmia.

Abscess of the spleen as the result of septic infection is by no means rare, and such abscesses always depend upon infected emboli.

Hydatid cyst of the spleen is rare not only because hydatid cyst is rare in this country, but also because it seldom develops in this gland even in those parts of the world in which hydatid disease is common.

Malignant growths in the spleen are among the rarest pathological lesions. Primary growths are practically unknown and secondary growths are also very rare. From statistics at St. George's Hospital, London, collected by Walker, it is found that in 161 cases of carcinoma involving all parts of the body secondary growths appeared in the spleen seven times, and in 50 cases of sarcoma the spleen was affected once. Taylor in 677 cases of carcinoma, epithelioma, and sarcoma found secondary growths in the spleen in twenty-three instances. Sometimes in cases of cancer of the gall-bladder or of the pylorus the growth extends to the spleen by direct invasion.

Movable spleen, like movable kidney, is a condition in which this organ wanders away from its normal position so that it may be found far removed from its ordinary area, and even so low as the pelvis. Its displacement is usually associated with a sense of dragging in the left hypochondrium or loin, and if the pedicle becomes twisted great pain may be suffered, with fever, collapse, and finally necrosis of the splenic tissues. Osler records a case in which this occurred and in which abdominal section resulted in recovery, although a considerable part of the spleen was lost by sloughing.

It is necessary to separate wandering spleen from floating kidney. This can be done by the discovery of the splenic notch, by the greater sense of resistance in the otherwise normal kidney, and by the presence of resonance on percussion in the splenic area where splenic dulness is usually demonstrable.

Rupture of the spleen is a rare accident, but occurs occasionally in those who, while suffering from great congestion or enlargement of this organ, meet with an accident in which the splenic area is subjected to a severe blow. Cases are also on record in which the spleen has ruptured as the result of great distention. Rupture of the spleen will be found discussed under Malaria. The symptoms are those of internal hemorrhage and demand an immediate abdominal section.

The *treatment* of wandering spleen consists in the wearing of a bandage and pad to retain the organ in its place, and, if need be, we may resort to an operation to fix it by causing adhesions to form around it. Extirpation of the spleen has been advised in cases in which the symptoms are very distressing, but this should be done only when the condition is very urgent; for while the spleen has been proved to be not necessary to life, its removal jeopardizes existence and the patient passes through a long period of convalescence before recovery occurs.

SPLENIC ANÆMIA.

Definition.—Among the types of splenomegaly the condition known as splenic anæmia is one about which great difference of opinion has existed. Its existence has been denied by some physicians and asserted by others. At present it is generally considered that a distinct morbid state to which this name may be applied really exists. It is essentially a condition of anæmia with enlargement of the spleen, and lacks all of the additional conditions which are associated with these states in other maladies, as, for example, leukæmia, lymphadenoma, or lymphatic leukæmia.

Etiology.—This is unknown, but some suppose it to be due to intestinal infection.

Pathology and Morbid Anatomy.—An examination of the spleen in this disease shows that it is not only greatly enlarged, but that it shows signs of the existence of a perisplenitis with localized areas of capsular thickening. In some portions of the organ old infarcts may be found which in turn have caused puckering of its surface or depressed scars. When the spleen is cut it is found to be more resistant to the knife than normal, and it is somewhat fibroid. If a section is placed under the microscope it is found that the connective tissue is increased and the lymphoid elements wasted. The Malpighian bodies are fibroid. There is also in some cases a marked proliferation of the endothelial cells which line the blood sinuses. These cells are very large and may be so numerous as to fill these spaces till they resemble an endotheliomatous growth.

Dock and Warthin have called particular attention to hyperplasia of the hæmolymph nodes and thrombosis of the splenic vein. The association of splenic fibrosis with enlargement and cirrhosis of the liver constitute the type studied by Banti (Banti's disease), and this is thought to be a later stage of the process characterized in its early manifestations by splenic enlargement.

Symptoms.—The symptoms of splenic anæmia are *pallor, dyspnœa* on *exertion*, and *feebleness* associated with *enlargement of the spleen*. An examination of the blood does not throw any great light on the character of the case. Indeed, the blood changes are often in no way different from those of lymphadenoma or gumma of the spleen. The red cells number about 3,500,000, and the hæmoglobin equals about 50 per cent. When the disease is advanced poikilocytes and nucleated red cells are present. The white cells are not increased as in leukæmia, but usually are below the normal number, amounting to about 4500 per c.mm.

Diagnosis.—The diagnosis of splenic anæmia is very difficult and should not be reached until a careful study of the patient's past and present condition has been carefully made and his blood repeatedly examined. A considerable number of cases of so-called splenic anæmia have proved to be other diseases when studied longer or examined postmortem. All possible causes for enlargement of the spleen should be excluded before a decision is reached.

Splenic anæmia must be separated from the "anæmia infantum" of von Jaksch, in which the spleen is enlarged, but in which the changes in the blood and in the liver and spleen in no way correspond to those seen in splenic anæmia. This condition must also not be confused with enlargement of the spleen due to syphilis with the formation of gummata; nor with sarcoma of the spleen; nor with the anæmia of chronic malarial poisoning, with secondary splenic enlargement, nor with amyloid disease. It must also be separated from the anæmia associated with enlargement of the spleen secondary to cirrhosis of the liver, and from a condition described by Gaucher of chronic inflammation of the spleen (*epitheliome primitive*). Sometimes, too, in children suffering from rickets and marasmus with gastrointestinal intoxication, there is a considerable degree of anæmia and some enlargement of the spleen. Such cases have been thought to represent an infantile form of splenic anæmia, but the subsequent history of the patient seems to contradict this view.

Rolleston gives the following as the clinical characteristics of splenic anæmia:

1. Splenic enlargement which cannot be correlated with any known cause.
2. Absence of enlargement of the lymphatic glands.
3. Anæmia of a type midway between secondary anæmia and chlorosis.
4. Leukopenia, or at most no increase in the number of white blood corpuscles.
5. An extremely prolonged course lasting years.
6. A tendency to periodic hemorrhages, especially from the gastrointestinal tract.

Prognosis.—The prognosis under medical treatment is always unfavorable.

Treatment.—The treatment consists in the administration of full doses of arsenic, but, so far as we know, no method of treatment has yet been devised which materially alters the general progress of the disease.

According to Harris and Herzog, of 19 cases subjected to splenectomy, 14 recovered. To these series Scott has added 6 cases with 4 recoveries. Queen and Duval collected 6 cases and added 1 original case in which removal of the spleen was followed by a cure. They state in addition that a beginning hepatic cirrhosis may be arrested by splenectomy, although it is difficult to understand how this operation can exercise this effect.

BANTI'S DISEASE.

Under the name of Banti's disease a condition characterized by enlargement of the spleen, anæmia, cirrhosis of the liver, jaundice, and ascites is met with. It is thought by some that Banti's disease is a terminal stage of splenic anæmia; but, on the other hand, it is certain that this is not always the case. The disease is very rare.

HODGKIN'S DISEASE.

Definition.—Hodgkin's disease is a condition in which there is marked swelling and overgrowth of the lymphatic glands, both internal and external, with a moderate degree of anæmia which is in no way peculiar to this malady. The spleen is usually enlarged. The overgrowth of the lymph nodes and lymphatic tissues generally is closely allied to malignant lymphadenoma. Another name for the disease is "pseudoleukæmia."

History.—Although a difference between this state and scrofulous enlargement of the lymph nodes had been made prior to 1830, it was not until Hodgkin in 1832 described cases seen at Guy's Hospital that it was generally recognized. In 1856 Wilkes in London and Bonfils in France still further illuminated the subject.

It was not until Virchow completed his work on the histology of the blood that Hodgkin's disease became clearly differentiated from leukæmia of the lymphatic type. The fact that changes of a peculiar character exist in the lymph nodes was not known until 1897, when several investigators, notably Fischer, described them.

Etiology.—The cause of Hodgkin's disease is unknown. Within the last few years the view that it was a peculiar condition due to infection by the *Bacillus tuberculosis* gained a number of adherents, and there are without doubt certain facts connected with the malady which tend to substantiate this view. On the other hand, it may be considered as pretty well settled that this view is erroneous, for Dorothy Reed, Longcope and Simmons have published careful investigations which seem to prove that Hodgkin's disease possesses definite pathological characteristics peculiar to itself. The disease is more frequent in males than in females, and in adults than in childhood.

Pathology and Morbid Anatomy.—The changes which are most marked are enlargement of the lymphatic glands at first in limited areas and later all over the body. The cervical and inguinal glands are usually the most prominent, and the outlines of the neck may be completely obliterated. At autopsy the retrobronchial and retroperitoneal glands may be found enormously increased in size, forming a mass as large as the arm, and pressing on adjoining tissues such as the thoracic duct and the bloodvessels. The affected nodes are discrete and regularly enlarged. Their consistency varies. Sometimes they are firm and dense, at others so soft as to fluctuate. The cut surface of these glands is translucent, gray, or more rarely yellowish, and the tissues of the glands bulge forward. An overgrowth of lymphoid tissue may take place at the apices of the lungs and lead to a diagnosis of tuberculosis. Death may be due to pressure on the thoracic bloodvessels, and perhaps to pulmonary infiltration and exudation. Osler asserts that infiltration of the lung does not occur in this disease, and that when such an infiltration does take place the disease is true lymphosarcoma. There is also enlargement of the spleen with overgrowth of the lymphoid bodies, which are grayish-white in appearance, and consist of lymph follicles held together by a reticulum of connective tissue. The marrow of the long bones may be lymphoid or purulent in appearance, as it is in some cases of myelogenous leukæmia. The liver and kidneys may also be enlarged and contain lymphoid masses.

The characteristic lesions of Hodgkin's disease are an early increase in the lymphadenoid tissues with a proliferation of endothelial cells, the formation of uninuclear and multinuclear giant cells, thickening of the reticulum, and lastly an overgrowth of the connective tissue of the lymph nodes to the development of which the increased density of these masses is due. Eosinophile cells in most cases are present in very large number in the lymph nodes and in the bone-marrow. Not only is the disease characterized by these changes in the pre-existing lymph nodes, but there is a constant formation of new nodes which soon become similarly affected.

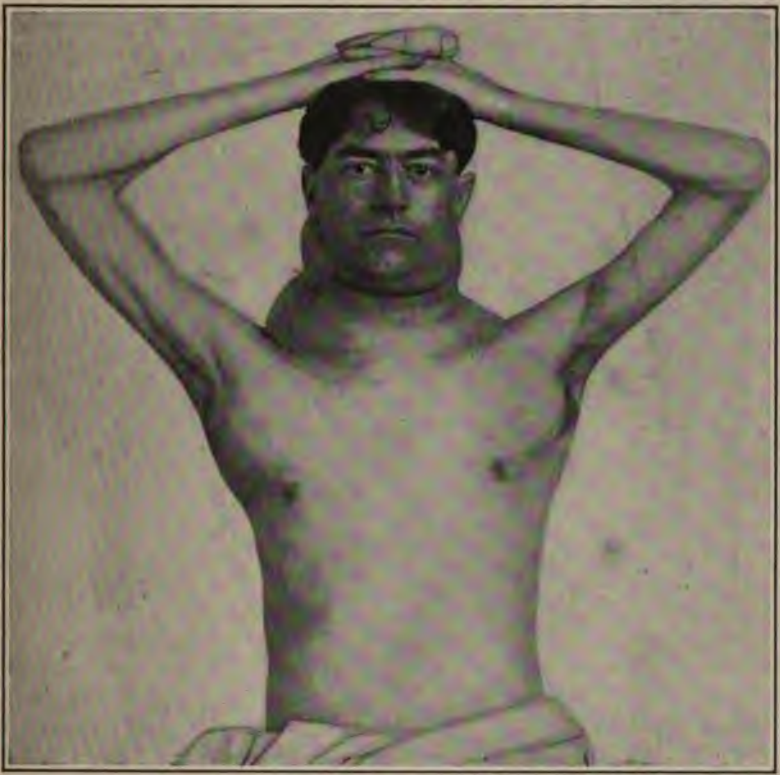
The blood changes are most variable. In some cases they are moderate, in others severe, in that the red cells may be decreased in number, but even when the patient is at death's door there may be fully 3,000,000 cells present. The red cells are not altered in a manner which is in any way characteristic. The changes consist solely in a diminution in number to a moderate degree, and in a reduction of the amount of hæmoglobin. The leukocytes are not increased as in true leukæmia, but are often actually diminished. Pinkus thought a relative increase of the lymphocytes constant, but this change was present in but 1 of Longcope's 7 cases, although a very large proportion of the white cells may be of this variety. Occasionally the increase of white cells may rise to the number seen in certain inflammatory states, as from 30,000 to 40,000. In the later stages the blood picture may assume all the characteristics of an intense secondary anæmia.

Symptoms.—The symptoms of pseudoleukæmia are those of ordinary severe secondary anæmia, with *shortness of breath* and *palpitation* of the *heart* on exertion. The enlarged masses of glands in the neck and groins may be very characteristic in appearance, associated as they are with *pallor* and *puffiness* of the *face*. When the internal glands are primarily and chiefly involved the diagnosis from tuberculosis may be difficult because the pressure may cause consolidation with patches of dulness on percussion, and because a distinct febrile movement is often present.

These masses by producing pressure may give rise to *paroxysms of cough* or of *pain* or constant *dyspnœa*. When the inguinal glands are affected *œdema* of the *lower extremities* may develop, and shooting pains may be felt in the legs. After the glandular masses become very large, *superficial sloughing* may occur, and therefore the resemblance to a suppurating tuberculous mass may be increased (Fig. 103). I have seen an actinomycosis of the neck produce similar symptoms. *Bronzing of the skin* may occur. Moderate *fever* is often present. It may be low and regular or high and regular in type. Occasionally it has an intermittent character with *sharp xacerbations,* so that it resembles intermittent or remittent malarial fever. *aundice* due to pressure on the bile-ducts may appear.

Additional symptoms met with in some cases are *murmurs* in the great essels, produced partly by the anæmia but chiefly by the pressure caused y the growths. *Deafness* due to closure of the Eustachian tubes by growths the pharynx and unequal pupils due to pressure on the cervical sym-athetic, may be present.

FIG. 103



Hodgkin's disease.

Diagnosis.—From true leukæmia pseudoleukæmia is to be separated y the absence of the large excess of leukocytes, and the lack of the leuko-ytes peculiar to that disease. From enlargement of the lymph glands due tuberculosis it is to be separated by the test with tuberculin (see Tuber-ulosis), and by the fact that tuberculosis rarely produces enlargement of e cervical glands on both sides and in both groins, and by the absence of tuberculous focus elsewhere. When doubt exists a part or all of an nlarged mass of glands may be excised and examined microscopically to etermine the character of the disease.

In many cases the clinical diagnosis is most difficult. I have seen the most eminent clinicians mistake this malady for tuberculosis. Very rarely a form of multiple lipoma distributed in the lymph-node areas closely resembles Hodgkin's disease; the picture may further be confused by the presence of glandular and calcific masses in the fatty tumors; such a case has been observed in the Jefferson Medical College Hospital; Chantemesse and Podwyssotsky figure such a case under the name adenolipomatosis.

Prognosis.—The prognosis in Hodgkin's disease is absolutely bad. Not one recovers. These patients have periods of improvement when courage runs high, but after all the inevitable progress is downward. Death comes from interference with circulation or respiration or by general asthenia.

Treatment.—The best treatment is the use of arsenic in full doses. Recently excellent results have been obtained by Senn, Finch, and others in some of these cases by the use of the *x*-rays, the parts involved being exposed to the rays, repeatedly, over a long period of time.

STATUS LYMPHATICUS.

Definition.—The term status lymphaticus or "lymphatic constitution," sometimes called "*constitutio lymphatica,*" is applied to a state which occurs chiefly in children or infants, and which is characterized by *hyper*plasia of the lymph nodes, the thymus gland, and the spleen, and by a *hypo*plasia of the heart and arteries. The lymphoid bone-marrow is also affected. The fact of particular interest is that patients with this condition sometimes suffer sudden death, which, coming on in children who, on superficial examination, appear unusually robust and plump, is all the more startling.

History.—As long ago as 1614 Plater made note of the fact that the thymus gland was found enlarged in certain cases of sudden death, and in 1830 Kopp described a form of difficult breathing which he called "thymic asthma," and which he considered was due to pressure upon the trachea by an enlarged thymus gland. This view, which was apparently disproved by Friedleben in 1858, received no further support until in 1888 it was revived by Grawitz, only to be controverted again by Paltauf in 1889.

Etiology.—The etiology of this state is not known, but it is apparently a congenital fault, and is associated with a low degree of vital resistance to infection. Hedniga has reported an instance in which out of a family of nine children, five died of this malady.

Blumer has recently put forward certain facts which seem to show that this condition is the result of the development in the body of a toxic substance, a cytotoxin. To this state the term lymphotoxismus has been applied. According to this view, the overgrowth of the lymphatic tissues in the different parts of the body is a sequence of the action of this poison and not the cause of its development.

Pathology and Morbid Anatomy.—An autopsy in a case of this character reveals an overgrowth and swelling of the lymph glands in the thorax, abdomen, and cervical and inguinal chains, and enlargement of the tonsils. The lymph nodes of the intestinal tract are very markedly swollen and sharply defined. The spleen appears larger than normal, and the lymphoid tissue of the Malpighian bodies is in a state of overgrowth. The changes in the thymus gland are, however, the most important, because it is supposed that the sudden congestion, hyperplasia, and swelling of the gland which occurs causes death by pressure upon the great vessels of the neck and upon the trachea. The thyroid gland may also be enlarged. If the shafts of the long bones are opened, yellow marrow is found to be substituted for red marrow. The heart and aorta are poorly developed.

Congestion and œdema of the lungs have been found in some cases at autopsy, and in others atelectatic patches have been discovered. In all of these cases the pressure upon the air passages was responsible for these changes.

Symptoms.—There are no symptoms in any way characteristic of this state, except that the child may, on close examination, be found to suffer from rickets, some enlargement of the lymphatics, and a rather *feeble heart*. The recollection of the possible presence of this state should make the physician particularly careful when administering an anæsthetic to a child of the lymphatic type, especially if the condition to be relieved is overgrowth of the lymph tissues, such as the postnasal adenoids and spongy tonsils, as sudden death may ensue. Hand has reported a case in which *tetany* was present. In a case recently reported by Musser and Ullom an unusually robust and well-developed, but overfat, boy of five years suffered from a little hoarseness on one day, and on the next seemed about as well as usual. That same night he went to sleep at the usual time, but at 2 A.M. was seen to be somewhat pinched and livid. A few hours later he became more cyanosed and presented a rapid pulse and labored breathing. His respirations were characterized by loud rales in the bronchial tubes and larynx. His temperature gradually rose till it reached 105°, at which time convulsions developed, and he died shortly afterward. The urine was loaded with albumin.

Several cases of so-called teething convulsions in infants have been found at autopsy to be due to this disease. Sometimes the child is found dead in bed.

Treatment.—We know so little of the cause of this state, and so few cases have been subjected to treatment, that no definite plan of treatment can be outlined, save that fresh air and sunshine and iron iodide, and arsenic are useful.

If the thymic enlargement be demonstrable, operation might be resorted to. Rehn opened the mediastinum, drew the gland forward and stitched it in position; recovery followed. Carter operated on a case for tracheal obstruction, recognized the thymic enlargement, and introduced a tube which gave temporary relief, but the child died.

DISEASES OF THE THYMUS GLAND.

The thymus gland is found well developed in infants up to the second year of life, after which time it gradually decreases in size till it reaches a degree of degeneration and atrophy in which it may be said to no longer exist. This occurs about the time of puberty. In fully developed adults it is represented by a small aggregation of lymphoid and fatty cells. Very rarely the thymus persists without any change in its tissues, even in adult life. When it remains large, or increases in size as the result of disease, as in the status lymphaticus, it may cause symptoms by pressing on the trachea and the great vessels of the neck and chest.

Diseases of the thymus gland are very rare. In some cases a state of so-called hypertrophy is present, but this is rarely a true hypertrophy, the gland being swollen and filled with lymphoid cells. Occasionally minute hemorrhages may take place into its tissues or beneath its capsule. Abscess has been recorded as having occurred, and growths, benign and malignant, have been found in its tissues. It has been found affected by tuberculosis.

Many years ago enlarged thymus was supposed to be the cause of spasmodic croup. That an enlarged gland may cause some interference with respiration is conceivable, but we now know that spasmodic croup is usually due to rickets or postnasal adenoids. Those interested in this subject should read Jacobi's essay published in 1888 in the *Transactions of the Association of American Physicians*, vol. iii. p. 297.

DISEASES OF THE BLOOD.

ANÆMIA.

Definition.—The word anæmia signifies a state of the blood in which there is lacking the normal quantity of red cells or of hæmoglobin in these cells. When the cause of this state is due to some disorder of the blood-making or blood-destroying tissues, it is called essential or primary anæmia. When due to some other cause, such as hemorrhage or one of the acute infectious diseases, it is called secondary anæmia. Fortunately, most cases of anæmia belong to this latter class, and they will, therefore, be considered first.

Secondary Anæmia.

Secondary anæmia arises from a host of causes and is characterized in most cases by a diminution in the number of the red cells, and an even greater reduction in the hæmoglobin content of each cell. In some cases it comes on as a result of breathing vitiated air, as in factory girls and stenographers. In other cases it is due to overwork and insufficient or improper food, and in still other cases to protracted digestive disturbance or to chronic constipation, which, by causing autointoxication, is the active factor in producing the condition. The late Sir Andrew Clark was an earnest exponent of this view.

Hemorrhage is, of course, a very potent cause. When it is profuse the change appears so promptly that the cause is evident, but in many cases anæmia arises as the result of repeated small losses of blood, as from hemorrhoids when the patient is at stool. Under conditions of hemorrhage, nucleated cells and a few poikilocytes may be found. After a single large hemorrhage the rapidity of recovery is extraordinary, being more rapid than when the loss of blood has been prolonged. Cases are occasionally met with in which, by reason of lack of regenerative power in the blood-making organs, a sharp hemorrhage is followed by death in a short time.

Toxic states due to renal disease or to the various infectious maladies, particularly malaria and syphilis, often cause anæmia, and prolonged lactation, frequent child-bearing, and the growth of tumors of large size also produce it. When the tumors are malignant, the anæmia is also partly toxic. Not infrequently we meet with cases in which the anæmia is due to chronic metallic poisoning, as from arsenic, lead, and mercury. In other instances the anæmia is due to intestinal parasites, such as the *Ankylostomum duodenale* or the tapeworm.

In all cases of marked and apparently causeless anæmia, the possibility of the condition being due to intestinal parasites should be borne in mind, and the stools examined, not only for the ordinary tapeworm, but for uncinaria as well. Further than this, the marked increase in the eosinophiles in the blood in nearly all cases of parasitic infection should be recalled. Walker has collated the following interesting table in this connection:

	Eosinophiles.	Polymorphonuclears.	Large mononuclears.
Normal	1 to 4 per cent.	60 to 70 per cent.	5 to 8 per cent.
Tænia	6 to 13 "		
Ankylostoma duodenale .	12.43 "		
Filaria medinensis . .	6 to 36 "		
Filaria loa	53 "	23 "	
Oxyuris vermicularis .	0.4 to 13.7 "		
Bilharzia hæmatobia .	16 to 48 "	44 to 58 "	12.5 "

He makes the interesting statement that a practical application of this has been already made. In the feces obtained from one of the closets in a large college the eggs of the *Ankylostomum duodenale* were found. The pupils who had used the receptacle on the specified day were asked to submit themselves to examination, but their feces yielded negative results. An examination of the blood of each pupil was made, eosinophilia was observed in 2 cases, and ova subsequently detected in their feces.

Symptoms.—The symptoms of *secondary anæmia* vary greatly in different individuals, some patients with *marked pallor* presenting no other noteworthy symptoms, while others whose cheeks have color, nevertheless, suffer from *palpitation* and *dyspnœa* on exertion. It is important to bear in mind that there are fat anæmics and red-cheeked anæmics, and that many persons who look pallid really have a normal number of red cells and a normal percentage of hæmoglobin. *Headache, neuralgia, loss of appetite, constipation,* and *attacks of syncope* are sometimes due to anæmia, and in women *amenorrhœa* is often due to this cause.

Diagnosis.—The diagnosis between secondary and primary anæmia is to be made by the history of the patient, and chiefly by the fact that the abnormalities, as to the shape and character of the red cells found in the primary anæmias (which see), are far more marked than in the secondary forms.

Treatment.—In the treatment of secondary anæmia three things are absolutely essential: the removal of the cause, if possible, the institution of a proper diet, and a hygienic mode of life, and the administration of iron and arsenic, and often of the bitter tonics, in order that the condition of the blood may be directly improved. If the method of life of the patient is unhealthy, it must be corrected, and, above all, plenty of fresh air and sunshine must be obtained. The sleeping-rooms should be well ventilated, and the patient must be dieted in such a way that the bowels are moved regularly and adequately every day, at least once, for constipation, as already stated, is usually present, and is often the cause. Under these circumstances aloes and cascara sagrada are probably the best laxatives to administer, particularly if iron is given. When persistent diarrhœa is the cause of the anæmia, I have found that Rockbridge Alum Spring water is a useful remedy, and in some instances it is advisable to give one of the astringent preparations of iron like the dried sulphate in the dose of $\frac{1}{4}$ to $\frac{1}{2}$ grain three times a day. If the anæmia is due to a loss of blood by hemorrhoids, this loss must be arrested by local treatment. The appetite should be stimulated by the use of drugs like nux vomica, quinine, or other bitter tonics like cardamom or gentian. A good prescription for many of these cases is a pill composed as follows:

℞—Ferri redacti	gr. v.
Acid. arsenosi	gr. 1/3.
Ext. nucis vomicæ	gr. v.—M.

Ft. in pil. No. xx.
Sig.—One t. i. d. after meals.

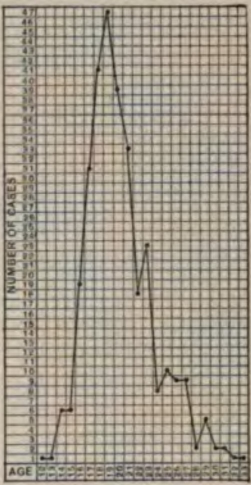
The nux vomica may, in some cases, be replaced by 2 grains of quinine. If the digestion is impaired, hydrochloric acid and pepsin, or pancreatin and bicarbonate of soda, or taka-diastase in 2 to 3 grain doses with meals is advisable.

Primary or Essential Anæmias.

Chlorosis. **Definition.**—Chlorosis is a condition of the blood usually met with in young girls, and characterized by a marked diminution in the quantity of hæmoglobin, and by a less marked decrease in the red cells. Until recently it was considered a secondary anæmia but hæmatologists now class it in the primary anæmias chiefly because it is believed to be due, at least in part, to defective hæmogenesis.

Etiology.—There are still many who believe that chlorosis is a secondary anæmia. It is certainly associated with many causes of secondary anæmia, such as constipation, inanition, and bad air. Whatever influence these causes may have, there can be no doubt that the processes which take place about the age of puberty in the female sex are closely concerned in its production, for the disease is scarcely ever met with except at a period of life near these changes. Occasionally, however, it develops in later life, and it is then called "*chlorosis tarda.*" Bramwell and others have shown that there is also a hereditary influence.

FIG. 104



Age incidence of chlorosis. (Bramwell.)

The two chief causes are a natural predisposition to anæmia and an inability to utilize iron from the food. Some believe that the intestinal mucosa is at fault, others that the spleen is functionally perverted, but, as Ewing well says, there can be no doubt that chlorosis results from a functional insufficiency of the bone-marrow, which is prone to occur at puberty.

Pathology.—The following are the chief changes in the blood: The chief alteration is the decrease of the hæmoglobin in each corpuscle, so that a low color index is one of the points necessary to a diagnosis of the disease. So low a percentage of hæmoglobin as 10 has been recorded by Bramwell, but the average low limit is from 30 to 40 per cent., and the color index about 0.5 per cent. The second change is a diminution in the number of the red cells, but usually this is not so marked. Taking the normal for a woman as approximately 4,800,000 to the cubic millimetre, the fall is usually not more than from 500,000 to 1,000,000. When a very great fall in the number of red cells is present, they may amount to only 2,000,000, but in such a case a suspicion of pernicious anæmia comes forward.

If the chlorosis is severe, the red cells vary as to size and shape, and a number of large red cells, with a full complement of hæmoglobin, may be present. As a rule, the size of the red cells is reduced. Imperfectly formed cells (poikilocytes) are found, but they are present in very small numbers. Granular degeneration of the red cells has been particularly well studied by the chief of my medical clinic, Dr. J. C. Da Costa, Jr., and by Stengel, White, and the younger Pepper.

The leukocytes in cases of chlorosis do not suffer much change, as a rule, but some patients show an increase of the lymphocytes. Dr. Da Costa has shown that most of these are large lymphocytes, and that the lymphocytes may amount to 40 per cent. of all the white cells. The specific gravity of the blood is reduced *pari passu* with the hæmoglobin. Although extravascular coagulation is retarded, in some cases there is a tendency to thrombosis.

Aside from the blood changes, a state of hypoplasia of the tissues of the heart and larger arteries is often present, but this condition is not peculiar to chlorosis, but to the lymphatic constitution. When recovery begins to take place the number of undersized red cells decreases and the cells of normal size increase their hæmoglobin content.

Symptoms.—The symptoms of chlorosis are a *peculiar pallor* of the skin, which often has a *greenish* hue, whence the name "green sickness." The patient is nearly if not always plump and possessed of a good quantity of subcutaneous fat. Occasionally the superficial vessels are well supplied with blood, so that the patient is ruddy, thereby misleading the physician who fails to study the blood. This type is called *chlorosis florida*. The subjective symptoms are *dyspnœa* on exertion, *palpitation* of the heart, *vertigo*, and perhaps attacks of *partial syncope*. *Constipation* is nearly always marked. *Headache* is quite constant, and there is usually a most persistent absence of appetite. There is also *mental depression* and *apathy*. Physical examination will reveal a diastolic hæmic murmur at the third left costal cartilage, and over the right carotid artery a bruit is often heard.

The *complications* of chlorosis which are serious are the development of thrombi in the veins of the legs and in the cerebral sinuses. From such thrombi fragments may arise, which may result in pulmonary embolism. Slight fever may occur, but the hands and feet are usually cold. Amenorrhœa is a very constant symptom.

The blood changes, as discovered by the hæmoglobinometer and hæmatocytometer have already been described.

Diagnosis.—The well-nourished state of the chlorotic patient is also present in pernicious anæmia, but the differentiation is found in the blood count (see Pernicious Anæmia), for in that disease the decrease in the white cells is usually marked. The irritability of the heart must not be taken for a sign of cardiac disease because a murmur is present, nor, in the absence of urinary changes, should the puffy face and ankles be thought to be due to renal disease, unless the urine reveals albumin and casts.

Prognosis.—The ultimate prognosis in cases of chlorosis is usually very good, but a long period often passes without much improvement. When the number of the red cells is not greatly reduced and they are normal in shape and size, recovery under proper treatment is usually rapid. This holds true even if the color-index is very low. When the red cells are as low as 3,000,000, are badly shaped, and many of them undersized, the prognosis as to rapid recovery is bad. So, too, when the lymphocytes are very numerous, a speedy cure is rarely seen. Relapses are very frequent.

Treatment.—The treatment of chlorosis does not differ materially from that which was given for the treatment of ordinary secondary anæmia, except that chlorotic patients are usually more obstinately constipated, and, therefore, particular attention must be given to the state of the bowels. Fresh air and sunshine are also very essential in these cases. More important than all, it must be remembered that chlorotic patients usually need very much larger quantities of iron than do ordinary anæmic cases. Whether this is due to an inability to absorb iron or whether there is an excess of sulphides in the bowels, which change a goodly portion of the iron into a sulphide of iron, is not known. During the winter months chlorotic patients usually do best at seaside resorts or at places like Lakewood, N. J., which are low in altitude. In the summer months they should be sent to high altitudes, varying from 3000 to 5000 feet, unless these high altitudes tend to increase palpitation of the heart and dyspnœa.

Pernicious Anæmia. **Definition.**—Pernicious anæmia is a disease of the blood arising from faulty hæmogenesis and excessive hæmolysis or blood disintegration. It is a fatal malady characterized by three chief changes, viz., an extraordinary decrease in the number and alterations in the morphology of the red cells and by certain changes in the bone-marrow.

History.—Andral in France, in 1821, reported cases of what was probably this disease, and Channing in Boston recognized them in 1832. Pepper and Tyson showed the bone-marrow changes in 1875. Sorenson, in 1874, made the first observations as to the number of the red cells, reporting cases with only 470,000 corpuscles.

Etiology.—The etiology is still unknown, but it is probable that certain of the causes of ordinary secondary anæmia may antedate pernicious anæmia. Age has no great influence. Most cases appear between twenty and forty years of age, but even young children of less than five years have been seen with the malady. In some of the reported cases the *Ankylostomum duodenale*, the *Bothriocephalus latus*, or tapeworm, and the *Oxyuris vermicularis* have been found. The first-named parasites can cause grave anæmia, but it is doubtful if any of them alone can cause pernicious anæmia. So, too, severe hemorrhage, syphilis, and pregnancy have been found associated with the development of the disease. They are not the actual cause, but rather predisposing causes. The various fevers, as malaria and typhoid fever, only exert an indirect effect, and the condition of the gastrointestinal tract, at one time thought to be responsible, has been proved to be only a predisposing or secondary condition. Hunter thinks the disease is due to bacterial infection of the alimentary canal from a foul mouth.

Morbid Anatomy.—The most noteworthy pathological changes are the alterations in the blood and in the bone-marrow. Unlike cases of chlorosis, the blood may be difficult to obtain. In marked cases it does not form a rounded drop, but flows from the puncture made by the needle or scalpel sometime after the wound is inflicted. Coagulation is usually delayed, and even at an autopsy made many hours after death the blood may still be fluid. The red cells are decreased to 2,000,000, then to 1,000,000, and sometimes to less than 500,000 to the cubic millimetre. A great proportion of the remaining cells are larger than normal (megalocytes), and some are smaller (microcytes). Many of the red cells are misshapen (poikilocytes), and the amount of hæmoglobin in most of the red cells is considerably increased, although some of them may be poor in hæmoglobin. Red cells possessing nuclei (erythroblasts) are also present in considerable number. Some of these are large (megaloblasts) and others of normal size (normoblasts). Small nucleated red cells called microblasts are also present. The presence of the megaloblast is an important aid in reaching a positive diagnosis. These nucleated cells, both large and small, contain a great amount of hæmoglobin, and some of them differ from ordinary red cells in one very important particular—namely, they possess amœboid movement—and, further, when the blood is examined, they are seen to form rouleaux as do ordinary red cells. A granular degeneration which permits basophilic staining also occurs in the red cells.

In mild cases the leukocytes may not be altered in number, although usually they are slightly reduced. Rarely they are greatly reduced in number if the case is severe. As a rule, but few myelocytes occur and the lymphocytes may be increased.

The hæmoglobin is reduced, the average being 25 to 30 per cent., but not in proportion to the red cells, hence a high color index is the rule.

When the bone-marrow is examined very marked changes are manifest. There is an excess of large nucleated red cells, many of which are gigantoblasts. The liver, spleen, lymph glands, and particularly the liver, are loaded with iron derived from the destroyed red blood cells, and even the urine contains pigment from this source; Hunter denies an excess of iron in the spleen. Fatty degeneration of the liver, kidneys, and of the heart muscle are often present, and because of similar changes in the walls of the arteries and of the altered character of the blood, hemorrhages into the retina and into other parts may occur. In some cases marked atrophy of the gastric tubules is found. A diffuse degenerative change occurs in the posterior and lateral columns of the spinal cord.

Symptoms.—A patient with pernicious anæmia usually first seeks medical advice because he is feeling *weak* and lacks initiative. Often he suffers from some *dyspnœa* on exertion and has a throbbing *headache* or attacks of *vertigo*. His tissues are well filled with fat and his appearance is plump, but he is pallid and cheesy looking. The *sclerotic* part of the eyes is peculiarly *pearly* and the *lips, gums,* and *tongue* are prone to be very *pale* and *bloodless*. There may be slight *puffiness of the face* on the dependent side if the patient lies on his side in bed. A purring *hæmic murmur* is often heard over the pulmonary artery at the third left costal cartilage, and the arteries of the neck pulsate and throb with a peculiar jerking, expansile movement. An irregular fever is very constantly present. When the disease is far advanced a state of mental torpor with muttering delirium may occur.

Diagnosis.—The fact that the patient is in middle life, or even older, serves to separate this state from chlorosis, which has its greatest frequency at the eighteenth or nineteenth year. Again, pernicious anæmia is more common in men, chlorosis in women. The skin in pernicious anæmia is prone to show a cheesy-yellow hue in distinction from the greenish-yellow of chlorosis. From the blood of the chlorotic that of pernicious anæmia differs so radically that a diagnosis is readily made in typical cases, for in the former we have cells poor in hæmoglobin and here we have cells rich in hæmoglobin. In the former the red cells are not greatly decreased in number, here they are markedly diminished. In chlorosis the size of the red cells is below the normal, in pernicious anæmia the average is above the normal. Again, in chlorosis we do not find, to the same degree, nucleated red cells or cells with mitotic nuclei, nor red cells with amœboid movement. The color-index in chlorosis is low and in pernicious anæmia high. In cases of gastric cancer there is present ordinary secondary anæmia and the presence of gastric symptoms to aid the diagnosis. Finally, Ewing states that unless at least 33 per cent. of the red cells are oversized, the diagnosis of pernicious anæmia must be made with reserve.

Prognosis and Treatment.—The prognosis of true pernicious anæmia is almost invariably fatal, although there have been a considerable number of cases in which recovery has been said to have occurred. Many cases have periods of extraordinary improvement in all the symptoms as well as in the blood, and then a disheartening relapse takes place. This may be repeated several times. Most cases die within a year after they are first seen. A great decrease in the number of red cells and a large number of megaloblasts are bad signs.

The prognosis in pernicious anæmia depends not a little upon the quantities of arsenic which the patient can take without developing disagreeable symptoms from its use, for many of these cases are markedly benefited if they can take what might be called massive doses of this drug. By a process of training with ascending doses I have known patients to take as much as 30 minims of Fowler's solution three times a day with no bad results except some exfoliation of the skin of the hands after several weeks' treatment. Arsenic administered in this manner up to the point of tolerance sometimes produces periods of remarkable improvement in this disease, the patient's symptoms becoming modified and the number of red blood cells becoming markedly increased.

Of course, all measures which tend to increase the general health of the patient are advantageous, such as plenty of sunshine, fresh air, and good food. Small quantities of iron may be given from time to time with advantage. Diarrhœa is to be controlled by the use of sulphuric acid or one of the vegetable astringents, such as fluid extract of hæmatoxylon, kino, or catechu. Constipation, if present, is to be relieved by the use of cascara sagrada and aloes. In some instances hypodermic injections of arsenic have been resorted to with asserted advantage, but this is a method of treatment which is rarely if ever justified. If the patient is very anxious to carry out a plan of treatment which may possibly be advantageous, inhalations of oxygen may be suggested.

In the treatment of pernicious anæmia it is the universal experience of clinicians that iron is by no means as beneficial as is arsenic. Indeed, the general proposition may be stated that if anæmia is associated with diminution in the number of red blood cells, arsenic is more advantageous than iron; whereas, in those anæmias which are characterized by a low color-index or a diminution in hæmoglobin, iron is more useful than arsenic.

LEUKÆMIA.

Definition.—Leukæmia is a disease in which the blood suffers an extraordinary increase of leukocytes with associated alterations in the bone-marrow, the spleen, and in the lymphatic glands. It is divided into two types, that form in which the spleen and bone-marrow are chiefly affected, and that in which the lymphatic glands are chiefly involved, the first being called *splenomedullary leukæmia* and the second *lymphatic leukæmia*. Because of the important role played by the bone-marrow in splenomedullary type it is sometimes called *myelogenous leukæmia*. Leukæmia is also sometimes called *leukocythæmia*.

Although a division of the disease into two types is to some extent justified because it renders the study of leukæmia less difficult, and because changes in certain tissues preponderate in one instance and in other tissues in other instances, it is not to be forgotten that intermediate cases occur in which both types of the malady are represented, or at least in which no definite dividing line can be drawn. Thus cases are recorded in which the blood cells presented the picture of lymphatic leukæmia, but the lymphatic glands were not involved but the bone-marrow was altered. Again, it has been thought, in times past, that acute and rapidly progressive leukæmia was usually of the lymphatic type, and that the subacute or chronic form of the disease was commonly of the splenomedullary variety. While this view still holds true, we have been forced to recognize the fact that acute cases may be of splenomedullary type and that some of the chronic cases may be lymphatic.

History.—As long ago as 1801 Bichat, in France, noted a condition of the blood which was probably identical with leukæmia as we know it to-day. Thirty years later because of the peculiar appearance of the blood it was called "suppurative hæmatitis," and in 1839 Donne described the blood in these cases as consisting largely of "white or mucous globules." Virchow, about the middle of the last century, described it as "Weisses Blut," and showed that there was no suppurative process present. Bennett made the first complete study of the disease in 1851. Since then a host of investigators have thrown light upon its characteristics.

Etiology.—The cause of leukæmia is unknown. It may occur at any time of life, but is most frequently met with about the fourth decade. It is about twice as common in males as in females. A very large number of conditions have been brought forward as causes, but none of them have been proved to exercise a determining influence. Among these may be named syphilis, maaria, and intestinal intoxication. The view has been advanced that leukæmia and tuberculosis are nearly related in the sense that the latter may act as a cause of the former. This view is incorrect. Gowers tells us that the appearances of the lungs in one or two cases have been those of tubercle, that extravasations of blood into the lungs are common, and that these organs may undergo caseation and a tuberculous process may be simulated. Cavities may result from lymphoid infiltration. This, however, is not phthisis. Susmann has been able to collect only 25 cases from literature in which tuberculosis and leukæmia were even associated. Further, when the diseases are combined there is a tendency to a decrease in the number of leukocytes. A number of observers are strongly inclined to the belief that the condition is due to an infection by a parasite, but careful observation and experimental studies have afforded no conclusive result. Löwit and others have sought to establish a protozoal origin, but their observations are inconclusive, and bacteriology, so far, has yielded no promising results.

Pathology and Morbid Anatomy.—When defining the disease leukæmia it was stated that it is a malady which appears in two forms, and that these forms may be quite dissimilar in their chief features, or overlap one another, according to the degree to which the lymphatic system and the bone-marrow are chiefly affected. It is probable that in nearly every instance the bone-marrow is involved in the disease process, and almost never are the lesions situated only in the lymph nodes and other lymphatic tissues. Further, although the lymphocytes of a person in good health are usually derived from the lymphatic system, in the patient suffering from lymphatic leukæmia they are derived, to a large extent, from the bone-marrow as well.

Having made these preliminary remarks, we can best proceed to the study of two *forms of leukæmia* by considering them separately.

Splenomedullary Leukæmia.—The color of the blood may be normal in some cases, but if the disease is well developed it is much paler in hue because of the anæmia and the excess of white cells. The coagulability of the blood is greatly decreased or lost. The red corpuscles are not very greatly diminished in number until the disease is far advanced, and sometimes not then. They rarely fall below 3,000,000 to the cubic millimetre, but they may drop to 1,000,000. Nucleated red cells, especially normoblasts, are present in varying numbers, but megaloblasts are rarely present in any great degree. There is a decrease of hæmoglobin so that the color-index is about 0.5 to 0.7.

The white cells show remarkable changes as to number, shape, size, and variety. Even in cases which may be called moderate they usually amount to 100,000 or 200,000 to the cubic millimetre, and as high as 1,000,000 have been reported, as against a normal of about 6000 to 10,000. Of the varieties of white blood cells we find a form which never appears in normal blood, and which, if present in considerable numbers, is pathognomonic of the disease, namely, large mononuclear leukocytes containing neutrophile granules. These cells are called neutrophile myelocytes and appear in two forms, viz., the smaller myelocytes, about the size of the polymorphonuclear leukocyte, possessing a central nucleus which stains quite deeply, and a larger cell, which has a pale staining nucleus placed at one side of the corpuscle. Eosinophilic and basophilic myelocytes also may be found. The relative percentage of polymorphonuclear cells is decreased, although the total number of these corpuscles far exceeds the normal. The presence of the myelocytes is at the expense of the polymorphonuclear cells.

The eosinophile cells, leukocytes the granules of which stain intensely with eosin, are generally increased in myelæmia, although they are never as numerous as are the other forms already named.

The number of lymphocytes, both large and small, varies within wide limits in most cases of splenomedullary leukæmia, and at different times in the same case. Relatively they are reduced (as low as 2 per cent.), but even in this percentage the blood may contain more mononuclears to the cubic millimetre than in health. Very large mononuclear leukocytes with faintly staining nuclei are rarely conspicuous by their number. "Mast-cells" are usually present, not infrequently reaching 5 or 10 per cent. When present in large numbers they are second to myelocytes only in diagnostic significance. These mast-cells are polynuclear cells with coarse basophile granules.

The onset of any one of the acute infections may completely change the appearance of the blood so that it is no longer characteristic and there may be a great increase in polymorphonuclear cells.

The normal red bone-marrow shows marked hyperplasia and the fatty marrow of the long bones undergoes a similar transformation. It contains nucleated red cells in unusual numbers, and is crowded with leukocytes which are ancestral to those found in the blood, including all forms, but particularly myelocytes. In some cases, however, these changes appear in patches rather than all through the bone cavity. At certain points the compact portion of the bone may atrophy before the hyperplastic marrow, and new subperiosteal nodes may develop.

The spleen is very much enlarged, sometimes to fifteen times its normal size. It is frequently attached to adjacent tissues. Its capsule is usually thickened and roughened, and the consistency of the organ increased. On section it is seen to be mottled red and gray, or it may be a homogeneous red. The trabeculæ may be thickened and hemorrhagic infarctions may be present. The venous system is engorged and purulent-looking clots may be found in the heart and vessels. The liver is often enlarged and leukæmic nodules may be found in it and in the kidneys and thymus gland.

Lymphatic Leukæmia.—The conspicuous blood changes in this disease are confined to the lymphocytes, which are greatly increased in number so that they may exceed 80 per cent. of the total number of white cells. For this reason the condition is sometimes called lymphæmia. Usually most of the cells are small lymphocytes, but the large form may predominate, especially in the acute type of the disease. The total increase in leukocytes rarely approaches that seen in the splenomedullary form. Nucleated red cells are rarely encountered.

The lymphatic glands, particularly those which are deeply situated, are enlarged, but the spleen and the medulla of the long bones are not greatly altered. Cases are on record in which chronic lymphatic leukæmia has occurred without any enlargement of the lymph nodes.

Symptoms.—The symptoms of *splenomedullary leukæmia* are at first those of *anæmia*, the patient presenting himself because of *dyspnœa on exertion*, or because of lack of energy and *poor digestion*. Sometimes the *swollen spleen* first calls his attention to his condition. In other cases *nose-bleed* or *gastric hemorrhage* or *renal hemorrhage* comes on very early in the disease. In other cases there is *purpura hæmorrhagica*. If the hand is placed over the splenic area and the abdominal wall moved over the enlarged spleen a creaking sensation may sometimes be felt. Not rarely the liver is greatly enlarged, and *ascites* may be present. There is *dizziness* and *vertigo*. Occasionally a violent *diarrhœa* develops. The urine is normal, save that it contains uric acid in excess. The heart sounds are normal, although the first sound may be feeble, and *anæmic murmurs* can occasionally be heard. Retinal hemorrhages may cause blindness. *Hemiplegia* with coma, the result of cerebral hemorrhage, may occur. A moderate but varying *febrile movement* is nearly always present. Occasionally persistent *priapism*, probably due to irritation of the spinal cells by anæmia, is present. *Sudden death* may take place.

In cases of *lymphatic leukæmia* the symptoms complained of by the patient are not different from those just described, but the spleen is not much enlarged. The *lymph* nodes are, however, *increased in size*, and so the appearance of the patient may not be unlike that of Hodgkin's disease.

It is important to recall the fact that in both of these states an irregular febrile movement may be present and give rise to the belief that some one of the acute or chronic infections characterized by fever are present. This is particularly true in certain cases of acute lymphatic leukæmia, in which the condition of the patient may be so like that of typhoid fever that an examination of the blood is required to determine the exact nature of the illness. The condition often runs its course in three to four weeks; fever of moderate degree is present, and the general state is asthenic. Even an autopsy may not reveal the real cause of the illness, because the lymph nodes in the solitary and agminated glands of the intestine are infiltrated as in typhoid fever.

Diagnosis.—The pallor, the enlarged spleen, and the state of the blood are all part of a picture which cannot be mistaken for any other disease, but in doubtful early cases repeated blood examinations may be necessary to determine the diagnosis positively.

Prognosis.—The prognosis of leukæmia, like that of pernicious anæmia, is, in the great majority of cases, fatal, but rare instances have been recorded in which recovery has taken place. Life may be preserved from a year to three years. Unfavorable prognostic signs are marked dyspnœa, an excessive number of leukocytes, a tendency to exhausting diarrhœa or to hemorrhages, and high fever. The lymphatic type usually runs a more rapidly fatal course than the splenomedullary form. When death occurs, the cause is usually pulmonary œdema, pneumonia, or exhaustion.

Treatment.—The treatment of leukæmia in both its forms is identical, but, unfortunately, it is by no means successful, for the disease in all instances proceeds by a more or less rapid course to a fatal ending. There can, however, be no doubt that the administration of arsenic in ascending doses until the point of intolerance is reached seems to exercise a favorable influence upon the malady, at least in so far that it delays its advance. Cases of leukæmia not subjected to treatment not infrequently have periods of remission, in which temporary improvement may take place. It is, therefore, difficult to determine how much credit should be given to arsenic when the remissions occur under its use. Those members of the profession who have had the most experience in the treatment of leukæmia, however, regard arsenic as being practically the only remedy of any value, and it should always be tried, preferably in the form of Fowler's solution. The beginning doses should be 3 drops three times a day, rapidly increased until the patient has some puffiness of the face or some griping of the bowels. Recently several clinicians have reported " cures " of this disease by exposing the patient to the Roentgen rays. As well shown by Warthin in his exhaustive discussion of this subject the final outcome of most if not all of the cases thus treated is relapse and death from the disease. Warthin's experimental studies indicate a possible danger from the absorption of substances liberated by the destruction of cells brought about by the Roentgen-ray exposures. Further investigations will be necessary to place this procedure upon a sound therapeutic basis, as it deals with a disease that is notoriously resistant to ordinary remedial measures. However, the question is one that merits careful study.

CHLOROMA.

Under the name chloroma is recognized a condition characterized by the formation of greenish lymphoid tumors, especially in the cranial bones and periosteum, and the occurrence of a profound anæmia which Dock and Warthin regard as, in some if not all cases, a malignant type of leukæmia. Exophthalmos and lymphoid infiltration of the cornea and of the conjunctiva may be present. Later the periosteum of the bones of the spinal column become affected. Localized paralyses, from pressure exerted by these growths upon nerve trunks, may arise. Another characteristic of chloroma is the peculiar green hue of the new-growths ; the cause of this coloration is unknown.

The latest study of this remarkable and rare disease is that made by Dock and Warthin. These investigators conclude that the disease consists in a neoplastic hyperplasia of the parent cells of the leukocytes which develops primarily in the red bone-marrow and secondarily affects the periosteum. Typical and atypical leukocytes are, therefore, developed, set free in the blood, and may appear as large lymphocytes or as neutrophiles or eosinophile myelocytes. On the other hand, it is not essential for the diagnosis of chloroma that these leukocytic changes be present, for sometimes they fail to appear. Chloroma may, therefore, be regarded as a malady lying midway between leukæmia and lymphosarcoma. The essential point of differentiation is the development of the green masses of lymphoid tissue which are distinctly neoplastic in character. The prognosis of chloroma is invariably fatal.

ANÆMIA INFANTUM.

Definition.—Under this term von Jaksch has described a form of anæmia occurring in children under four years of age and resembling leukæmia in many respects, in that there is great enlargement of the spleen, marked leukocytosis, some increase in the size of the liver and of the lymph nodes. Von Jaksch believes that this malady separates itself from a true leukæmia of infancy by the fact that the increase in the white cells is never so marked as in true leukæmia, that children often recover, and because there is never any leukæmic infiltration of the viscera. The cause is not known.

The red cells are so much decreased that they number only from 3,000,000 to 1,500,000. There are also present poikilocytes and usually a large number of nucleated red cells. The total leukocytosis rarely exceeds 50,000, the chief increase being in the mononuclear corpuscles. Myelocytes are absent, or not present in sufficient number to justify a diagnosis of leukæmia. The liver and spleen are enlarged. The condition is usually met with in rachitic or syphilitic children and in those suffering from chronic gastrointestinal catarrh.

Treatment.—The treatment is identical with that of leukæmia.

PURPURA.

Under this term is included all those cases in which, as the result of various causes, extravasations of small quantities of blood take place into the skin. These extravasations are multiple and often very widespread. It must be borne in mind that under no condition is purpura a disease in itself. It is a symptom or manifestation of some disturbance in the nutrition of the smaller bloodvessels or of the blood itself. Thus, it occurs as a manifestation of severe infections, such as profound septicæmia, scarlet fever, typhus fever, measles, and smallpox, and in infections not so well understood, in which micro-organisms, known and unknown, are manifestly the cause of the condition. Various investigators have isolated from cases of purpura such micro-organisms as the *Streptococcus pyogenes*, the *Staphylococcus pyogenes aureus*, the *pneumococcus*, and the *Bacillus aërogenes capsulatus*. The *Bacillus coli communis* has also been obtained from the blood. Again, certain poisons, as snake venom and poisons from the mineral kingdom, may cause purpura. A large number of cases of marked purpura have developed in persons who have taken iodide of potassium, and after the use of mercury, copaiba, and the chlorate of potassium. It may also develop as the result of some congenital defect in the blood, as in hæmophilia.

Diseases generally called diathetic, such as scurvy, tuberculosis, Hodgkin's disease, and chronic nephritis, may cause this symptom. Purpuric extravasations sometimes develop after severe neuralgic seizures in locomotor ataxia and along the course of certain nerves in hysterical women.

Some writers have given the name purpura hæmorrhagica to that form of purpura in which, in addition to the extravasations into the skin, there are also lesions of this character in the mucous membrane of the mouth.

Under the name "purpura rheumatica," or "peliosis rheumatica," a form of purpura develops in which there is a distribution of the spots chiefly about the large joints, particularly about the knees. Associated with this eruption there is swelling of the tissues about the joints resembling that seen in rheumatism. There is usually no fever and little pain, although the joints appear stiffened at times. It was this arthritic state that gave the name "purpura rheumatica" to the condition. In some instances the joint disorder is due to acute rheumatism but in the majority it is some other form of infection.

Under the name of "Schönlein's disease" a very much more severe type of this condition has been described. Many of the joints are affected, so that the patient is bedridden, and the extravasations of blood into the submucous tissues and into the skin are so copious that great swelling and even sloughing may result. Some years ago I saw, in consultation with Dr. Wilson, of Woodbury, New Jersey, another physician who not only had the joints of the extremities greatly affected, but the inferior maxillary joint was also involved. The buccal mucous membrane was so infiltrated that we feared the development of noma, and the whole face was much distorted by the infiltration. Notwithstanding the severity of the lesions and the intense prostration of the patient, recovery usually takes place. Care must be taken that this form of purpura is not confused with scurvy or scorbutus (which see).

"Henoch's purpura" is a condition affecting children and characterized by lesions in the skin, which may be a combination of purpura and erythema multiforma. The joints may or may not be affected, and bleeding from the gums may appear. The most distinctive symptom, which is not present in all cases, but which may be present when the others are absent, is gastrointestinal crises, in which the child is seized with pain, diarrhœa, and vomiting. All these symptoms are prone to recur at irregular intervals. Recovery usually occurs, except in those cases in which the hemorrhagic state affects the kidneys, when a fatal result may ensue.

A form of fulminating purpura sometimes develops in young girls and causes death in a few days, the patient being apparently overwhelmed by some unknown infection.

There are three *hemorrhagic affections of the newborn* that occasionally occur.

In children with inherited syphilis, hemorrhage from the mucous membranes and from the navel, with intense subcutaneous extravasations, may occur and cause death. The autopsy shows hemorrhages into the liver and kidneys, and signs of inherited syphilis in these parts as well.

Under the name of Winckel's disease a condition of jaundice develops within a week of birth, followed by dyspnœa, hæmoglobinuria, and deep cyanosis. I saw a case some years ago in which the child was so cyanotic that parts of its body were blue-black. The autopsy in such a case shows swelling of the spleen and fatty degeneration of the liver and kidneys.

A third state called "morbus maculosus neonatorum" develops in new-born infants, and consists in hemorrhages from the stomach, intestines, or from the navel. It is rarely seen in private practice, and is probably due to some infection.

Hayem and Bensaude have stated that in purpura hæmorrhagica the blood, when allowed to stand in a vessel for twenty-four hours, slowly clots, but the clot does not contract to any extent, and therefore does not squeeze out the serum as it does in normal blood.

Treatment.—The treatment of all forms of purpura is based upon the recollection of two facts, viz., first, that the condition is due in most cases to an infection or at least to a cause which has impaired the health, and therefore every means to aid the vital resistance of the body must be resorted to. The food should be easily digested and nutritious; the patient, if able to travel, should be removed to some place where he can bask all day in the sunshine. Moderate doses of tincture of the chloride of iron should be given each day to combat the infection and the anæmia.

The second point to be recalled is that certain drugs may be employed to increase the coagulability of the blood. Of these, the only ones with any real claim to power are the lactate and chloride of calcium, which may be given in the dose of 20 grains three times a day to an adult, well diluted with water. Certain cases seem unable to absorb calcium salts through the alimentary canal and these should be treated by hypodermic injection. The solution should not be stronger than 1 part in 20 of water and the lactate should be used because the chloride is too irritating. The effect of one day's dose of 60 grains lasts three or four days, and it should not be too frequently repeated, since, if this is done, coagulability is decreased. Turpentine, oil of erigeron, ergot, and sulphuric acid have all been used. Their employment is purely empirical, and there is little reason to rely on them. Several ounces of nutritious food, fresh air, and a day in the bright sunlight will do more good in this state than all the medicines can accomplish, and this, too, without damaging the stomach. The use of these remedies in this state is putting "drugs of which we know little into bodies of which we know less."

HÆMOPHILIA.

Definition.—Hæmophilia is a condition of the body in which there is an inability to arrest hemorrhage by the normal coagulation of the blood, or in which hemorrhage arises apparently without cause and persists without any attempt being made by nature to arrest it. The disease is essentially hereditary in most cases, and it is an extraordinary fact that the hemorrhagic tendency is transmitted to males only through the female parent, although the mother is herself usually not afflicted.

Etiology.—The cause of this condition is entirely unknown. Virchow believed it to be dependent upon an abnormal thinness and narrowness of the arteries, but it is probably due to some deficiency in the coagulability of the blood as well. Stengel has recorded an instance in which the hemorrhagic tendency was limited to one part of the body, namely, the head, and Osler states that the late D. Hayes Agnew described to him a similar case.

Pathology and Morbid Anatomy.—There are no changes in the blood cells that account for this condition; indeed, no peculiar state of the blood is found save a diminished coagulability. An examination of the tissues of the various organs is also practically negative, save that if the hemorrhage has been profuse the changes always met with in marked anæmia are present.

Symptoms.—An active but *oozing capillary hemorrhage* is the form in which the bleeding usually occurs, and it follows in some instances very slight injury. Thus, the mere blowing of the nose may be sufficient to rupture the fine vessels of the nasal mucous membrane and cause a dangerous loss of blood, and *epistaxis* is the most common form in which this condition manifests itself. Another common source of the blood is from the gum after tooth extraction.

In some cases bloody effusions take place into the large joints.

Prognosis.—The prognosis depends largely upon the severity of the loss of blood and upon the ability of the patient to restore the quantity lost before the next bleeding comes on. Fully 50 per cent. of bleeders die before the seventh year, but some live to old age. Although girls who reach puberty menstruate with their entrance upon adult life, and so are exposed to an excessive loss of blood, their mortality rate in this disease is not so high as that of boys.

Treatment.—The treatment consists in the building up of the general health by out-door life and exposure to sunshine, and in the use of small tonic doses of iron and arsenic if anæmia is present. If a special tendency to hemorrhage exists at any particular time, calcium lactate or chloride should be given in doses varying from 10 to 20 grains three or four times a day, well diluted, to increase the coagulability of the blood, but this must not be used for long periods without intermission, as after a certain amount is taken the coagulability of the blood is decreased and not increased. The local treatment consists in the use of tampons wet with adrenalin chloride, 1:1000, or gelatin solution of the consistency of thin mucilage may be used for the same purpose. When adrenalin cannot be obtained, peroxide of hydrogen may be applied in the same manner to the bleeding spot. When the hemorrhage is from the gum, a compress made of punk may be used, or a compress wet with a saturated solution of alum, with Monsel's solution, or with adrenalin chloride solution, 1:1000.

DISEASES OF NUTRITION.

DIABETES MELLITUS.

Definition.—Diabetes mellitus is a disease characterized by the appearance in the urine of glucose, and the development of polyuria, thirst, and excessive appetite, with impairment of nutrition, and in some cases progressive emaciation. The mere presence of glucose in the urine does not necessarily indicate that diabetes is present. The glycosuria must be associated with other morbid processes to present the symptom-complex of the disease.

History.—Diabetes mellitus has been known since the time of Christ, but it was not till the latter part of the seventeenth century that Willis, in England, noted that the sweet taste of the urine was probably due to sugar, and not until 1775 that Dobson, of England, actually obtained sugar from the urine. Since that time a host of experimental investigators and clinicians have studied the disease from every aspect and have added much to our knowledge of it, but no one has as yet been able to give us a clear conception of the causes of the malady.

Distribution and Frequency.—Diabetes occurs in all parts of the civilized world, but is much more common in Europe than in the United States. It is frequently met with in France, in Sweden, in Italy, in India, and Ceylon, but is comparatively rare in Russia, Holland, and in Brazil. Negroes rarely suffer from it, but Hebrews are so frequently affected by it that it may almost be said to be the prevalent disease of that race. Külz, of Germany, found that in 692 cases of diabetes 17.8 per cent. occurred in Jews, which is all the more remarkable when we consider that Hebrews constitute only 1.2 per cent. of the population of that country. Frerichs found 102 Jews in 400 diabetic patients, and von Noorden 252 Jews in 650 patients suffering from this disease.

It is a disease of adult life, as a rule, but very young children suffer from it occasionally, and even nurslings have from time to time been reported as presenting well-developed cases of the malady. To emphasize its rarity in children, Stern may be quoted in his assertion that only thirteen deaths have been reported from this malady as occurring in children under five years of age in the past thirteen years. It is more common in males than in females in the proportion of 3 to 2.

The statement generally made that diabetes is a disease of cities rather than of country districts is not altogether true. Some years ago Purdy showed that, as a rule, it was much more frequent in the country districts than in cities. In the United States the disease is much less frequent in the Gulf and South Atlantic States than anywhere else.

There can be no doubt that the disease is becoming very much more frequent than it was several decades ago. I showed this in a paper published some years since, based upon statistics gathered from the Jefferson Medical College Hospital and other large hospitals here and abroad. Since then additional statistics have been collected which indicate the correctness of these earlier conclusions.

According to the mortality statistics of the United States Census Reports for six decades, the proportion of deaths from diabetes mellitus in 100,000 deaths from all known causes has been as follows:

From 1840 to 1850	72	From 1870 to 1880	191
" 1850 to 1860	98	" 1880 to 1890	280
" 1860 to 1870	170	" 1890 to 1900	470

It seems scarcely credible that so great an increase could have occurred, and it is possible that greater care in examining patients and in regard to correct death certificates is responsible for part of the increase, yet the records of the Massachusetts General Hospital from 1824 to 1898 show that four times as many cases of diabetes were admitted to the hospital during the last thirteen years of that period as during the first fifteen years. On the other hand, it is only fair to state that the statistics of L'Hôtel Dieu, Lyons, for seventeen years, which were collected by Alix, for Lépine, who attempted to ascertain if diabetes was increasing in a certain district of France, do not show any increase in the disease.

Etiology.—The causes of diabetes are not known, although it is a well-recognized fact that lesions in certain portions of the nervous system are followed by glycosuria, and that certain alterations in the islands of Langerhans in the pancreas and in the circulation of the liver are also followed by the same symptom. (See Pathology.) As somewhat indirect causes we recognize severe nervous strain and errorrs in diet, but these alone are not sufficient to cause even glycosuria, much less true diabetes, in the vast majority of human beings.

There is some evidence to indicate that the disease is hereditary, for sometimes it happens that several members of the same family suffer from the malady, but this may be due to their being exposed to the same exciting causes, whatever they may be. A more pronounced factor than any so far named seems to be excessive indulgence in rich foods and sweet wines, but a very small proportion of the persons who commit these dietetic errors suffer from diabetes.

Pathology.—Of the pathology, or morbid physiology, of diabetes we are very ignorant, although an immense amount of skilled research has been devoted to this subject for years. It is a well-recognized fact that in all human beings glycogen is prepared from carbohydrate foods, and even from proteids and fats, and deposited in the liver and in the muscles, where it lies as in a storehouse as reserve food. It also circulates in the healthy blood stream in the proportion of about 1:1000, and so is carried to various parts of the body for nutritional purposes.

There are many conditions which produce loss, or leakage, of this substance in the form of glucose in the urine. Thus, glycosuria, or the mere presence of sugar in the urine, may follow the ingestion of an excess of either cane-sugar or grape-sugar or an excess of carbohydrate food. Under these circumstances it is simply an overflow of material which the system cannot utilize. This being true, it is readily conceivable that in certain states of disease the system may be unable to utilize the ordinary amount of glycogen, and therefore it escapes from the body. This view receives support from the theory advanced by Loewi and Kolisch, who believe that there is in the organism a body, or ferment, or agent, which binds the glycogen in the tissues in such a form that it does not appear in the blood in excess. If for any reason this binding body (Bindekörper) is diminished in power, an excess of glucose passes to the kidneys and so escapes from the body. This view explains a considerable number of cases of glycosuria, but by no means all of them.

It is a well-known fact that injury or disease of the so-called diabetic centre of Claude Bernard in the medulla is followed by glycosuria, and that this glycosuria is directly due to a disorder of the blood supply in the capillaries of the liver. This may interfere with the "binding" of the glycogen in that organ. Again, the administration of phloridzin will produce glycosuria, but this condition is quite different in its causation from ordinary glycosuria, in that the drug acts upon the kidney structure in such a way that it permits a leakage of the normal content of glycogen from the blood. In other words, in this state the fault does not lie in an inability of the body to use its glycogen, but in the inability of the kidneys to prevent its escape from the body.

We find, therefore, that glycosuria and diabetes mellitus are by no means identical conditions necessarily, although glycosuria is the predominant symptom in this disease. There may be a loss of sugar in the urine for many years without any impairment of health, or the glycosuria may not be constant, but recurrent and appear only when nutritional processes are for any reason jarred or disturbed. In true diabetes mellitus, on the other hand, there are associated with the glycosuria more or less profound impairment of nutrition, with wasting, emaciation, and the development in the body of certain poisons which act very deleteriously and may cause death.

These distinctions, which serve to separate in the mind of the student glycosuria from true diabetes mellitus, however, like many other distinctions, do not actually hold true in all cases, for we frequently see persons who begin with the leakage of sugar into the urine and end with true diabetes; and we meet intermediate cases in which the degree of emaciation, thirst, and polyphagia is so mild that it is difficult to tell whether the patient is a sufferer from an inability to deal with carbohydrate food or is really diabetic.

The question of the pathology of this disease, as far as we know it to-day, can perhaps be summed up in the following words: In certain individuals there exists, as a result of a congenital or acquired defect in the metabolic functions of the body, an inability to utilize for the purpose of nutrition all the carbohydrate material which is taken as food. Such persons suffer from simple glycosuria. If the defect just referred to becomes more marked they gradually lose the power to retain and utilize any noteworthy quantity of the carbohydrates ingested, and when this condition develops they speedily emaciate and lose vital resistance. Finally, by reason of some further defect in the organs whose functions govern nutrition, such persons actually convert their body fat and proteid tissues into sugar and pass it from them in the urine, in which cases death soon closes the scene.

Certain factors tend to produce the chain of disturbances just enumerated, and all these agencies may cause changes which may be mild and continue so, or, in another case, become severe and rapidly fatal. These may be mentioned as follows:

1. *Heredity.* It is conceivable that the parent may hand down to the offspring certain defects which will interfere with the proper utilization of carbohydrates.

2. *Errors in diet, both as to food and drink.* It is conceivable that errors of this character can so pervert, or overwhelm, the processes of nutrition or elimination that primary glycosuria followed by permanent diabetes may ensue. Thus, the excessive beer-drinkers of Bavaria often suffer from this disease, probably because of the excess of fluid, of alcohol, and of carbohydrate which they ingest. These factors pervert the function of the liver, of the pancreas, and of the kidney.

3. *Profound nervous worry and mental anxiety* are undoubtedly followed by diabetes in some persons probably because the nervous mechanism governing nutritional processes is perverted in function by the stress and strain.

4. *Certain injuries* to the central nervous system may so result, for severe trauma of the head or the growth of an intracranial tumor may produce diabetes.

5. *Certain infectious diseases* may produce temporary glycosuria, which disappears with the acute disease or persists and becomes true diabetes, probably because the acute infection has damaged beyond repair nervous centres or glands whose function is to control glycogenesis and the utilization of glycogen.

6. *Diathetic diseases* such as gout undoubtedly cause, or predispose to, diabetes in some cases, but whether this influence is direct or simply a sign of general perversion of metabolism we do not know.

Finally, it must be recalled that of all the organs of the body the liver and the pancreas are the viscera which show the greatest morbid changes in true diabetes. Not only is the liver the organ which is chiefly concerned with the manufacture and storing of glycogen, but the pancreas undoubtedly exercises a very powerful influence upon the glycogenic processes; for not only does it secrete digestive ferments which act in the intestine, but also a ferment or factor which enters the blood stream and is intimately concerned with the utilization of glycogen. Thus, if the pancreas is extirpated glycosuria is at once developed, and the same condition ensues if the gland is totally destroyed by disease. It would appear that the so-called islands of Langerhans are the portion of the gland which exercises this influence upon the processes connected with the utilization of glycogen, and in many cases these islands are found to be distinctly diseased. While disease of the pancreas is responsible for the development of diabetes in some cases of the malady, it is also a fact that in many cases of very severe diabetes the most careful examination of the pancreas after death fails to discover any lesion that can be considered in any way responsible for the malady. It seems evident, therefore, that many causes may so pervert nutritional processes that glycosuria or true diabetes may result, and in this sense it may be said that diabetes is not a primary disease, but rather a symptom of some primary lesion which we do not at present understand.

Morbid Anatomy.—The changes found in the islands of Langerhans are various. In some cases they manifest capillary engorgement or hemorrhagic extravasation. In others there is found a pericapillary or peri-insular sclerosis, an atrophy, a necrobiosis, or hyaline degeneration. The latter is probably always primary, and the former conditions usually secondary. Aside from changes of the islands of Langerhans in the pancreas, the most notable changes presented postmortem in any of the organs of the body are found in the liver. This organ is usually markedly hyperæmic and darker in color than in diseases which do not affect its functions. Microscopically it is found that its capillaries are congested, and that the liver cells are enlarged and show a tendency to coalesce. These changes are not, however, peculiar to the disease, being found in other states; and it is a fact worthy of consideration that when death results in cases of severe disease of the liver, glycosuria is rather an unusual symptom.

There is a form of diabetes, associated with hepatic cirrhosis and bronzing of the skin, called "bronzed diabetes," owing to the color of the skin.

The structure of the kidneys is diseased in a very large number of diabetics. These changes are not in any way a part of the disease itself, but result from the increased activity of these organs in excreting water and sugar, and by reason of the effect of toxic substances, such as acetone, diacetic acid, and oxybutyric acid, which, as they are eliminated, damage the renal tissues. The constancy of renal changes in diabetes is proved by the frequency with which these organs are found diseased at autopsy when death has occurred from this disease. Out of 121 autopsies, reported by Griesinger, Dickinson, and Seeger, renal changes in diabetes were found in 77 cases, and Elliott has collected statistics from European clinicians which show that albuminuria is present in 43.68 per cent. of all cases of diabetes. The renal lesions may be divided into two classes: In one class, which is usually met with in chronic cases, an ordinary chronic nephritis develops in which parenchymatous and interstitial changes both occur. In the second form, which is really toxic in origin, there is a hyaline degeneration of the tubular epithelium, the so-called "cellular necrosis of Ebstein." A so-called glycogen degeneration of Henle's loop and of the straight uriniferous tubules (Ehrlich's lesion) is also found, but it cannot be claimed that these changes are pathognomonic.

The changes in the nervous system are usually of little importance. Peripheral neuritis is often present. Probably this is purely secondary. Distinct changes have, however, been found in the spinal cord in cases of diabetes mellitus, notably by Williamson. Using Van Gieson's method this clinician found an increase in connective tissue in the columns of Goll in the cervical area. There was also a diminution in size of the nerve fibres in these columns, and the myelin sheaths and axis cylinders were also diminished in size, although a few of the myelin sheaths were distended. When Marchi's method was employed degenerated fibres were seen in Goll's columns in the cervical, dorsal, and lumbar regions, and a few degenerated fibres were present in Burdach's columns. So, too, degeneration was found in the intramedullary course of the posterior nerve roots, between the posterior surface of the cord and the posterior horn of gray matter, and to the median side of the posterior horn. These latter changes were most marked in the lumbar and cervical region. In a few instances degenerated fibres were seen in the posterior roots just outside the pia mater. These changes are probably secondary and due to the altered condition of the blood in diabetes.

The blood is not only unduly rich in glucose, but sometimes contains an excess of fat-globules to such an extent that it may form a cream-like layer on the clot when it stands after withdrawal. Fraser has recorded a case in which an analysis of the blood showed that it contained 16.5 per cent. of fat and the pleural fluid 20 per cent.

The blood in some cases of diabetes mellitus is of a pale salmon color which has been thought to be due to the presence of fat, and for this reason this condition has been called lipæmia. Futcher, however, has shown that this appearance is not entirely due to fat, for it is not possible to remove it from drawn blood by the use of ether, and the granules do not stain black with osmic acid. He suggests that these granules are in part albuminous. More recently Cole, in testing the blood in a case for Hale White, reaches the conclusion that true fat is not present, but that the foreign material seems to be an ester of cholesterine with one of the higher fatty acids. Heyl showed in 1880 that this state of the blood could be demonstrated in the retinal vessels, and Hale White has more recently recorded a beautiful case of the same character.

The lungs are often found to contain tuberculous foci, and may show well-developed bronchopneumonia or croupous pneumonia, but these are the result of terminal infections and not part of the primary disease. Arteriosclerosis and its train of associated lesions are of frequent occurrence in diabetes mellitus.

Symptoms.—The symptoms of diabetes vary very greatly in their severity in different cases, so greatly that it is almost impossible to detail any array of symptoms which are common to all cases. In many instances the *glycosuria* exists for a considerable period of time before the patient suffers from symptoms which lead him to think he is not well. A very well-known medical writer in London, some years ago, first discovered he was diabetic by observing that flies were unduly attracted to the vessel in which he urinated, and later by noticing that a few drops of urine which accidentally fell on his black trousers left a white stain on drying. Later on, all the diabetic manifestations developed, and he died of diabetic gangrene of the foot.

As a rule, as the disease progresses the patient notices that he *passes water* more frequently and in *larger quantity* than is normal, he develops more or less thirst, and *loses sexual desire* and *power*. Later on he begins to feel languid and inert; he is usually constipated because of his polyuria, and he may develop an inordinate appetite in the endeavor to compensate for the loss of nutriment through his urine. The *thirst*, the *polyphagia*, and the *loss of strength* and *flesh* are usually in direct proportion to the *polyuria* and the quantity of sugar excreted. When the polyuria is marked the *tongue* becomes *glazed*, dry and raw in appearance, and attacks of *stomatitis* or *thrush* may develop. The *skin* is also *dry* and *harsh*, and the hair lustreless and brittle. The *pulse* is *feeble* and the *temperature subnormal*.

Although the disease is often characterized by excessive *emaciation*, this symptom is subject to extraordinary variations in different patients. Marked loss of flesh is almost constant in all persons under twenty-five years of age, but after this time it is by no means uncommon to meet with patients who maintain their weight for years. This holds true in direct proportion to the years of age and the degree of polyuria and loss of sugar. Where the tissues are freely drained of fluid or starved beyond repair the weight of course suffers.

The *urine* in diabetes mellitus is not only abnormal in that it contains *sugar*, but it not uncommonly contains *albumin* as well. (See Complications.) Its *specific gravity* is high and ranges from 1.025 to 1.045, and one instance of 1.074 is recorded by Trousseau. Such a specific gravity, however, is exceedingly rare. Notwithstanding its high specific gravity, however, the *urine* is usually exceedingly *limpid* and *clear*, it has a *sweet odor*, and is *acid* in *reaction*.

The *quantity of sugar* present varies over wide ranges. Sometimes it is found in as small an amount as 1 to 3 per cent.; in others it is found to be present in the proportion of 10 per cent. The total quantity passed in twenty-four hours may be from one ounce, or less, to a pound and a half. Very rarely even more escapes. Dickinson reports a case that passed the incredible amount of fifty ounces of sugar a day.

The *quantity of urine* is also very great in some cases, while in others it may not be much above the normal quantity. As much as six to twelve pints are often passed in each twenty-four hours, and cases are recorded in which as much as thirty pints were passed in this time.

Complications and Sequelæ.—The complications and sequelæ of diabetes mellitus are important, and so constantly present that they may aid materially in the diagnosis of the disease. Many of them are dependent upon the fact that the constant loss of sugar lowers nutrition and so decreases vital resistance to the various infections, or they result from perverted metabolic processes closely associated with the inability of the body to properly control the functions governing glycogenesis and the proper utilization of glucose in the economy.

Sweet has demonstrated that for certain organisms the blood loses its bactericidal power in diabetes.

It not infrequently happens that the first symptom presented by a diabetic patient is repeated *crops of boils or carbuncles*. When the urine is examined sugar is found, and it becomes evident that the lowered vitality caused by diabetes mellitus has permitted infection of the skin to occur. Sometimes the carbuncle becomes malignant and speedily destroys the patient. In still other instances, which are not as common as has been thought, *diabetic gangrene* occurs, the primary lesion being some break in the skin of a finger or toe resulting from the blister made by an ill-fitting shoe, or by wounding a corn. Through this lesion infection takes place, and vital resistance is so low that the micro-organism speedily causes the local death of the part, and almost equally rapidly may involve the blood in a diabetic septicæmia. In such cases the gangrene is moist. In another class of cases, which depend upon secondary vascular changes not due to direct infection, the gangrene is dry and of the so-called senile type.

Elliott's statistics, already quoted, show that *albuminuria* is present in about 43.68 per cent. of diabetics. This albuminuria may at times possess grave significance, and is worthy of careful consideration. It may be said to arise from three causes, namely, from renal congestion, due to cardiac feebleness and impairment of the circulation; from degeneration of the kidney, due to true nephritis, and, finally, to severe irritation or inflammation of the renal tissues by the poisons of the disease. The first type can usually be relieved by careful treatment of the heart, and the second type, with casts in the urine, is to be regarded as a complicating condition of gravity superimposed upon one already exceedingly severe. The third or toxic type is, however, the form which presents the most grave and alarming aspect, for its onset is usually acute; it comes on when the patient is already profoundly ill, and it often betokens the rapid approach of diabetic coma, which in such a case may be said to be partly due to the renal lesions. Many clinicians consider that coma never comes on without this associated symptom. The kidney condition is therefore to be studied carefully in these cases, as it may give warning of approaching coma.

Dyspeptic symptoms are often very annoying. They depend upon the excessive eating and drinking, to which many diabetics are forced by their thirst and hunger, the feebleness of the digestion arising from the failure in vital power, or to perversion of the digestive functions by the toxæmic state often developed as the disease advances. Extreme constipation is often a very troublesome symptom.

Pulmonary complications, such as bronchopneumonia or tuberculosis, are very frequently met with in diabetics, owing to the lowered vital resistance which permits infection. Such complications are very often the cause of death, particularly tuberculosis.

Although albuminuria is quite a common symptom, particularly in those patients who have arteriosclerosis, general dropsy is rare, notwithstanding the enfeeblement of the heart and the impaired state of the kidneys, because the urinary flow is so profuse that the body is rapidly drained of fluid.

Callian has especially studied the influence of diabetes on the female genitals; pruritus vulvæ is very common; menstrual disturbances are common; the atrophy of uterus and ovaries, he thinks, depends on the associated sclerosis of their nutritive vessels.

Diabetes occurs in a certain percentage of cases of acromegaly and exophthalmic goitre.

The *nervous complications* of diabetes mellitus may be divided into the acute and chronic. The acute complications are very serious from a prognostic point of view, and consist chiefly of *diabetic coma*. Many theories have been advanced as to its direct cause. It is undoubtedly toxic in origin, and seems to be chiefly associated with a state of acidiosis, or the presence of one or more abnormal acids in the blood, of which one is called β-oxybutyric acid. The idea that acetone and diacetic acid are the causes has been cast aside. In some instances the onset of the coma (sometimes called "Kussmaul's coma") is sudden, but it may be gradual, although, even in the gradual cases, it is a matter of a few hours at the most, as a rule.

Diabetic coma may be said to appear in three types: The first, and most common, is often met with in young persons, and develops with suddenness in many instances; that is to say, its onset lasts but a few hours at the most. The early symptoms are those of disorders of digestion, with abdominal pain, vomiting, muscular weakness, and drowsiness, which soon ends in coma. The breathing in this coma is often slow and deep, very much as it is in the second stage of opium poisoning. At other times it is sighing. To the respiratory state in this condition Kussmaul applied the descriptive word "Lufthunger." The second form often comes on after fatigue, particularly in elderly persons, and the symptoms may be those of profound collapse. In the third form the early symptoms are those of ataxia and confusion of speech.

Sometimes the unconsciousness is preceded by great restlessness and irritability, while in other instances the onset of the comatose state is gentle.

Of the *ocular complications*, cataract, optic nerve atrophy, and diabetic retinitis are to be remembered. Sudden blindness due to optic nerve or retinal changes occasionally ensues and palsies of the ocular muscles may take place.

Diabetics are also subject to apoplexy.

Of the subacute nervous symptoms we find painful neuritis, and not infrequently a pseudotabes due to this cause, with loss of knee-jerk, Romberg's symptom, and even the Argyll-Robertson pupil. Sometimes a true tabes dorsalis seems to develop.

Diagnosis.—The diagnosis of diabetes mellitus is easily made if the physician will carefully examine the urine and will bear in mind the fact that he is not justified in deciding that the well-developed disease is present unless some symptoms which are characteristic are associated with the glycosuria. It may be said that he who has constant glycosuria is in the early stages of diabetes mellitus, and this is particularly true if this symptom be constant in a young person. On the other hand, it not infrequently happens that a person of fifty-five or sixty years develops a mild glycosuria which lasts for years, and does not materially impair the health for a long period of time. Even these cases, however, often develop into the true disease. In other words, we may say that while glycosuria is not diabetes, it is a state that indicates a tendency to this disease or the presence of its early stages. The separation of the polyuria of diabetes insipidus from that of diabetes mellitus is made by the low specific gravity of the urine in the former disease, and the fact that sugar is present in the latter malady.

It not infrequently happens that diabetes mellitus is overlooked because the patient does not complain either of thirst or of excessive urination, and the physician fails to examine the urine as a matter of routine. But patients often present symptoms which, while not distinctly urinary, should at once call the attention of the physician to the possibility of diabetes being present. Thus, any patient who suffers from marked loss of flesh and increasing weakness should always be suspected of having diabetes, even if signs of tuberculosis are present, for not infrequently the tuberculosis is secondary to the diabetes. So, too, women will sometimes complain of pruritus of the vulva or eczema of the genitals, or men will state that they are becoming impotent, in all of which cases the urine should be examined, since diabetes often produces these signs. So, too, defects of vision, due to diabetic cataract, or, more rarely, to retinal changes, may be the first symptoms manifest to the patient, and still others complain of numbness or tingling in the extremities and present the symptoms of locomotor ataxia. On the other hand, as already pointed out, physicians not infrequently are so careless as to examine the urine only once, and when they discover sugar consider that the case is one of diabetes; or they obtain a reaction with Fehling's test, because of the presence of some sugar-reducing substance, as when the patient is taking chloral; or, again, they mistake physiological glycosuria for true diabetes. It is evident that most of these mistakes in the diagnosis of diabetes depend upon lack of urinary examination or imperfect methods of testing this secretion.

As an illustration of how necessary it is to examine a number of samples of urine before determining that the patient has or has not diabetes, it may be recalled that urine passed during the night or before breakfast is often free from sugar; while that passed after breakfast and during the day may contain much of it.

The condition of diabetic coma is separated from the unconsciousness of uræmia by the cider-like odor of the breath, the presence of glycosuria and acetonuria, and the absence of the high arterial tension usually met with in renal disease, for in this state the pulse is feeble and of low tension. At the same time, it is to be recalled that uræmia may complicate diabetes. The patient lies in a condition which resembles profound alcoholic intoxication. Deeply unconscious, with half-opened eyelids, wandering eyeballs, and dilated pupils, he breathes in a panting manner, a deep inspiration being followed by a quick expiration. The respiratory rate may not be greatly different from the normal, but sometimes it is hurried, and then forms "diabetic dyspnœa," with a gradually increasing cyanosis. The temperature is usually normal or below normal. This state almost invariably ends in from one to two days in death.

BLOOD TESTS.—In 1896 Williamson, of Manchester, England, introduced a modification and improvement upon Bremer's method of distinguishing diabetic from non-diabetic blood, which is of practical clinical value in cases of temporary disappearance of sugar from the urine. This test, which is based upon the fact that glucose, even in minute quantity, decolorizes warm alkaline solutions of methylene blue, is performed as follows: 20 c.mm. of the suspected blood are added to 40 c.mm. of distilled water contained in a test-tube. Then 1 c.c. of a 1:6000 watery solution of methylene blue and 40 c.mm. of liquor potassæ are added to the mixture. The contents of the tube are then well mixed by shaking. A control experiment is made by preparing the same quantities of normal blood and reagents in another tube. Both tubes are placed in a beaker of water, which is brought to the boiling point over a spirit lamp and then allowed to boil for four minutes. By the end of this time the fluid containing the diabetic blood will have turned to a dirty pale-yellow color.

In diabetes mellitus the erythrocytes do not stain at all or stain only faintly with certain aniline dyes. This has been proposed as a means of diagnosis, but as the same phenomenon has been observed in cases of exophthalmic goitre, leukæmia, and Hodgkin's disease, it cannot be regarded as of practical value. Diabetic blood stains with biebrich-scarlet, while normal blood does not.

URINARY TESTS.—For many years the most popular tests for the determination of the presence of sugar in the urine have been those which depend upon the fact that strongly alkaline solutions of grape-sugar reduce copper oxide to lower grades of oxidation. The most frequently employed of these tests is that which is made by means of *Fehling's solution.* This is best made in the following manner:

Copper sulphate, 34.64 gm., with water enough to make 500 c.c. Mix and keep in a bottle by itself. Pure Rochelle salts, 173 gm.; solution of sodium hydrate, specific gravity 1.330, 100 c.c., and water enough to make 500 c.c. For use mix equal volumes of these two solutions, thereby forming Fehling's solution. About one drachm of this solution is placed in an ordinary test-tube and boiled. If the solution does not remain clear, it is unsuitable for use. If, on the other hand, it does remain clear on boiling the suspected urine is to be added to it a few drops at a time, and the boiling continued, when, if sugar is present, the solution becomes opaque and yellow in hue, and soon a dense, yellowish-red sediment falls to the bottom. Should the quantity of sugar present be exceedingly small, it may be necessary to add urine until the volume of urine and the volume of Fehling's solution are equal. But the volume of urine must never exceed that of the Fehling solution.

Trommer's test is performed in the following manner:

A drachm of urine is placed in an ordinary test-tube, and is treated with sufficient quantity of sulphate of copper solution to render the fluid a light-green color. An equal volume of liquor potassæ is then added. This results in a blue precipitate of hydrated copper protoxide, which dissolves upon shaking the tube, so that a clear-blue solution remains. If the test-tube be allowed to stand for some time the copper is gradually reduced, and precipitation of the yellowish-red suboxide of copper occurs. If the solution is heated, the tests act more promptly. Care must be taken that the fluid is not boiled actively, as under these circumstances precipitation may take place without sugar being present.

It is hardly necessary to add that in making these tests the greatest possible cleanliness in the test-tubes and bottles should be maintained.

Occasionally the urine in cases of diabetes mellitus contains acetone in excess and diacetic and oxybutyric acids. The appearance of acetone in amounts greater than normal (0.008 to 0.027) is always to be considered a signal of danger of diabetic coma. The test for the presence of acetone consists in distilling the urine and adding to several cubic centimetres of the distillate a few drops of liquor potassæ, to render it alkaline. Several drops of Lugol's solution are now added, when, if acetone is present, the fluid becomes turbid, and iodoform is precipitated in crystals. If this fluid is now heated, the odor of iodoform is noticeable.

Gerhardt's test consists in adding tincture of chloride of iron to the urine, when, if acetone is present in large amount, the fluid becomes a deep red.

Prognosis.—The prognosis of diabetes is largely influenced by a number of factors. In the first place, as a rule, but by no means always, the outlook is favorable for long life in direct proportion to the age of the patient. Thus, it not infrequently happens that men and women who develop the disease after fifty or sixty years of age live the full length of years usually credited to human beings of that age. Even in these cases, however, the possibility of some intercurrent infection, like pneumonia, is to be borne in mind as a constant threat against life. Conversely, the disease is rapidly fatal in proportion to the youth of the patient. In young persons it runs a rapid course and may destroy life in a few weeks. Great emaciation usually develops in these cases; whereas, older persons may maintain their weight. The mere presence of glycosuria is not justification for as grave a prognosis, even if the amount of sugar be marked, as is the presence of glycosuria with associated thirst, hunger, and loss of flesh. For the first state is a leakage, while the second shows that nutritional changes are marked, and that true complete diabetes mellitus is present.

The prognosis also depends somewhat upon the manner in which the patient responds to the regulation of his diet. Thus, if on the gradual withdrawal of carbohydrates and the use of proteid and fatty food the sugar is no longer found in the urine, and the urine gives no reaction with perchloride of iron for acetone, the condition may be considered as a mild form of the disease. If, on the other hand, the quantity of sugar diminishes, but does not disappear, and the urine gives any reaction with perchloride of iron, the case should be considered as one of moderate severity. Again, if the gradual decrease in starchy foods, until the patient is taking no carbohydrates, fails to diminish the sugar excretion, and if a perchloride of iron test gives a Burgundy or port-wine coloration, showing the presence of acetone in excess, then the disease is to be considered as severe.

The cause of death in diabetes mellitus is usually one of the acute infections, such as pneumonia, tuberculosis, or septicæmia, with or without carbuncle. Diabetic coma is another common cause of death. Frerichs found that 150 out of 250 deaths in diabetes were caused by coma. Of 43 fatal cases observed by Taylor, death resulted from coma in 26. Mackenzie found 19 deaths from coma in 87 fatal cases.

Mandel and Lusk have recently stated the following proposition as to prognosis: If a diabetic be put on a meat-fat diet (rich cream, meat, butter, and eggs), and the twenty-four-hour urine of the second day be properly collected,[1] the discovery of 3.65 grams of dextrose to 1 gram of nitrogen signifies a complete intolerance for carbohydrates, and probably a quickly fatal outcome. They have called this the fatal ratio.

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[1] "The urine should be collected so that an early morning hour (before breakfast) terminates the period for one day. This is necessary, because the sugar formed from eaten proteid is eliminated before the nitrogen belonging to the same. The long period between the evening meal and breakfast allows for the elimination of both constituents."

It may be laid down as a rule, that true diabetes mellitus never gets well, but that temporary glycosuria often does so under proper treatment. Diabetes may, however, be controlled and life prolonged very materially by resort to suitable diet and remedial agents.

Treatment.—By treatment much can be done for the control of this disease. As already stated, simple glycosuria can usually be entirely relieved by proper attention to exercise, the regulation of the diet, so that the patient does not overeat or overdrink, and particularly by the limitation of the amount of carbohydrate food which he ingests. In these cases the appearance of sugar in the urine is to be regarded as evidence of the inability of the patient to properly utilize these substances in the body. If there is reason to believe that he is too sedentary in his habits, it sometimes happens that a moderate amount of exercise causes a disappearance of the glucose. Again, if he is a thin, spare individual, who naturally worries much about business or professional duties, absolute rest from these causes of stress must be insisted upon, in order that the nervous system may recover its equipoise.

When true diabetes mellitus is present, it is even more essential that these etiological factors should be controlled. Indeed, it may be well said that to attempt treatment by a diet and drugs is useless in a case of diabetes mellitus, unless the patient can be properly controlled in regard to his manner of life, provided that manner of life is deleterious. In other words, it is futile, in the majority of instances, to regulate the diet and to give drugs if the patient is to be continually exposed to causes which are more potent for evil than the remedies are for good.

There can be no doubt that the dietetic treatment of diabetes is far more important than that by drugs, and it is essential that this fact be borne in mind, since physicians are often careless in regard to the question of dietetics, and patients are still more so, even after the importance of a proper diet has been conveyed to them. Quite frequently they follow the directions of the physician for a short time, and then, wearying of being deprived of favorite articles of food, take these articles surreptitiously, or openly declare that whether it does them harm or good they do not intend to be deprived of things of which they are fond. For these reasons the dietetic treatment of diabetes is much the more difficult part of the care of these cases.

As diabetes is a condition in which the body is unable to properly utilize carbohydrates and their educts, it is manifest at once that an excess of carbohydrates must be forbidden; but what is an excess to one individual may not be an excess to another, for an excess is that quantity which is more than the body can use. For this reason it is usually wise, when placing a patient upon an antidiabetic diet, to diminish the quantity of carbohydrates which he receives, by a very gradual process, and to watch the quantity of sugar in the urine from day to day, since by this means the quantity of carbohydrate material which he can utilize may perhaps be approximated. A second reason for carrying out this gradual diminution in the quantity of starchy food lies in the important fact that not infrequently cases of diabetes are plunged into diabetic coma by the institution of a diet practically free from carbohydrates, perhaps because the system is in such a condition that no sudden variations in the character of the food can be permitted. Not only is it a clinical fact that coma may be precipitated in this manner, but we also know that the quantity of acetone in the urine is greatly increased by severe restrictions of carbohydrates. For this reason the physician, when restricting diet, should always examine the urine, not only as to its content of sugar, but as to content of acetone as well, and if this latter ingredient is present in an amount in excess of that which may be considered normal, for a minute trace is sometimes present in non-diabetic persons, it is absolutely essential that he shall at once restore the full carbohydrate diet, since by so doing the quantity of acetone is diminished and the condition of acidiosis which produces coma is diminished. (See Treatment of Coma.) The elimination of more than one gram of acetone in twenty-four hours is to be considered an excess.

Thirdly, patients will often resent the total removal of carbohydrates from their diet list, and yet yield to their gradual removal. On the other hand, it is not to be forgotten that in some diabetics a certain amount of carbohydrate food seems to be essential, in order that they may not manufacture glucose from other articles of food, or from the proteids of their own bodies, and in order that acidiosis be not produced. That is to say, the administration of starch in moderate quantity may compensate for their loss of glucose.

Sugars should always be excluded. They are unnecessary articles of diet, and, aside from the fact that the body is unable to utilize them, they are apt to disturb digestion.

Because carbohydrate food cannot be utilized, it has come to be well recognized that the patient must subsist largely upon the different forms of meat, both salt and fresh, excepting liver, which contains glycogen, and which, therefore, ought not to be given. So, too, butter, cheese, and the various oils and fats may be used.

It has already been pointed out that diabetic patients whose supply of carbohydrate material has been cut down should be provided with an amount of fat over and above that usually ingested, provided, of course, that the individual can digest and assimilate fats. It is evident, however, from a series of investigations made by von Noorden and others, that butter, when taken in excess of five ounces a day, may cause an increase in the quantity of oxybutyric acid in the blood. Von Noorden has pointed out, however, that this deleterious effect of large amounts of butter can be diminished if the butter is first washed with cold water in a most thorough manner, since by this means we remove the lower fatty acids which are chiefly concerned in the production of acidiosis. Under these circumstances, von Noorden tells us that as much as seven ounces of butter can be taken daily without difficulty.

Most of the shell-fish are useful, but contain too much glycogen.

In the way of fresh vegetables, the patient may receive the various greens, such as lettuce, spinach, dandelion, cabbage, cauliflower, Brussels sprouts, string-beans, celery, watercress, tomatoes, onions, cucumbers, olives, and the various kinds of pickles, and practically all of the nuts which are commonly employed as foods, except chestnuts, which contain too large a proportion of starch.

Not rarely the patient does best when he is placed upon a diet which varies in carbohydrates from week to week; that is to say, he is given a very small quantity of carbohydrate one week, and a fairly large quantity of it the next. In those instances in which the acetone reaction persists in the urine, whether carbohydrates are removed or allowed, von Noorden has strongly recommended what he calls "the oatmeal cure." In this cure the patient eats nothing but oatmeal gruel for from one to two weeks, save that in addition to the eight ounces of oatmeal he is given a similar quantity of butter and some vegetable albumin. Often this mixture is administered as frequently as every two hours. Von Noorden asserts that on this diet the excretion of sugar falls to a point far below that excreted on a mixed diet from which all carbohydrate has been removed. At the end of a week or two it is always necessary to return to other foods temporarily, as otherwise the patient rebels against the pursuance of a pure oatmeal diet; but even with these frequent returns to an ordinary diet, excellent results are said to be reached.

Tea, coffee, and cocoa may be employed, provided they are not sweetened by cane-sugar, but by saccharin. Dry wines which contain little sugar may be given to those who are accustomed to alcoholic drinks, although Scotch whiskey, rye whiskey, and dry gin are better than most wines. The various simple mineral waters may also be given, and of these both the natural and artificial Vichy waters are excellent, because of the quantity of bicarbonate of sodium which they contain. The old idea that because the patient urinates in excess he should be deprived of water is no longer followed. These patients should be allowed water freely, in order that the system may be flushed of toxic materials. When constipation is present, the mild saline purgative waters may be given, varying from Apenta, Carlsbad, and Hathorn water, to the more powerful saline purges.

Theoretically, gluten provides a source of nourishment for diabetic patients, but practically it is almost impossible to obtain a satisfactory gluten bread which does not contain a very considerable quantity of starch. There are upon the market a few samples of biscuits made from gluten flour which probably contain a very small percentage of starch, and these may be freely given to these patients. The difficulty in the majority of instances is that patients get exceedingly tired of a diet from which all forms of bread are excluded, and for this reason it may be impossible to entirely exclude bread from the diet list. Most of the biscuits which are made from substitutes for wheat flour, such as that of the soya bean, are so oily that patients find it difficult to digest them. Almond meal, which also contains a very large percentage of oil and no starch, may be given. But here, again, the difficulty in digesting the fats it contains is often marked. Perhaps the most satisfactory bread is that which is known as aleuronat, and which has been highly recommended by von Noorden. Williamson gives the following formula for its preparation: Mix two ounces (62 gm.) of desiccated cocoanut powder with a little water containing a small quantity of German yeast. Make the mass into a sort of paste, and put in a warm place for half an hour or longer. The small amount of sugar contained in the cocoanut is almost entirely decomposed by the fermentation produced by the yeast, and the cocoanut paste becomes spongy. Add two ounces (62 gm.) of aleuronat, one beaten egg, and a small quantity of water, in which a little saccharin has been dissolved, and mix well until a dough is formed. Divide into cakes and bake in a moderate oven for twenty or thirty minutes. The great difficulty is to obtain cocoanut fibres sufficiently desiccated and powdered.

Among the articles which are to be carefully avoided are all the sweet fruits, such as melons, grapes, peaches, and those vegetables which contain a very large amount of starch and sugar, such as rice, sweet potatoes, beets, beans, peas, and carrots.

Although potatoes are eminently a starchy food, recent investigations indicate that it is perhaps the best form of starch which can be taken by the diabetic.

The medicinal treatment of diabetes mellitus has narrow limits. It is true that a host of drugs have been recommended by various clinicians at various times, the statement being made that they were capable of materially decreasing the quantity of sugar which was lost in the urine, but further experience has almost universally proved that they possess little power. Furthermore, very few of them have been shown to possess any influence upon the symptoms associated with the glycosuria. In other words, at the best they affect only the one symptom of loss of sugar, and in no way correct the underlying cause of the malady.

Without doubt opium is the most valuable drug in the treatment of diabetes mellitus in the majority of cases, for it exercises a more potent influence in diminishing the elimination of sugar in the urine than any other drug. Its alkaloids, morphine and codeine, are also exceedingly valuable, and may be employed when they prove capable of controlling the glycosuria and when the opium increases the constipation, but neither of these alkaloids is the equal of the crude drug.

There are several important points in regard to the employment of opium, or its derivatives, in diabetes: First, patients of all ages seem to be able to take large quantities of opium in this disease without developing the evil manifestations of the opium habit. Second, these patients usually have to take ascending doses of the drug until they reach a dose which controls the glycosuria more or less completely. Third, opiates possess the advantage that they diminish to a large extent nervous irritation and stress. Not only do they protect the nervous system from external causes of irritation, but by producing mental quiet and diminishing worry they indirectly cause good results. An endeavor should be made from time to time to diminish, at least temporarily, the quantity of the drug which is taken. Ordinary deodorized opium is the best preparation. Patients may start on ¼ grain once, twice, or thrice a day, and gradually increase it, if necessary. Or, ⅛ to ¼ grain of morphine may be given at these intervals. In other instances ½ to 1 grain of codeine may be used as a beginning dose. Some patients get so much comfort and such a diminution of glycosuria under moderate doses of these drugs that the size of the dose does not have to be increased. Thus, I have had under my care for nearly twelve years a woman who has taken but 3 grains of codeine a day during all that time. She has never had any desire to increase the dose beyond this amount, and it has kept her glycosuria within bounds, besides giving her a great deal of comfort.

In cases which do not possess marked nervous symptoms, but which are rather phlegmatic in type, and have a gouty tendency, the salicylate of sodium or salicylate of strontium may be given in full doses varying from 10 to 20 grains three or four times a day; or, in their place, we may employ some of the new coal-tar products, such as antipyrin, acetanilid, and phenacetin. These drugs, however, must be given in full doses to have any effect, and they so greatly increase the susceptibility of the patient to cold that they must be used with great caution. In cases which have a syphilitic history, or which seem to be gouty, the iodide of potassium, in the dose of 10 to 30 grains or more three or four times a day, often does good. When the patient can digest it, cod-liver oil is an exceedingly valuable alterative and nutrient.

With the idea that the alkalies aid oxidation processes in the body and so help to burn up sugar, various alkalies, as the potassium and sodium salts, have been largely employed. Thus, potassium or sodium bicarbonate may be given in 10, 15, or 20 grain doses three or four times a day.

Another remedy which is of value in some cases, particularly if anæmia is present, is Fowler's solution in doses varying from 1 to 3 minims three times a day. With some clinicians it has a great reputation in this disease.

In the treatment of the various complications of diabetes we must first consider diabetic coma. After coma is once established, we have no method of treatment which promises permanent recovery. I have several times seen a temporary return to consciousness follow the intravenous injection of one quart of normal saline solution, and Continental clinicians have employed and strongly recommended the injection of carbonate of sodium in solution. (See page 806.)

When the presence of acetone in the urine or of the early symptoms of intoxication indicate that diabetic coma is not far distant, two plans of treatment should be promptly instituted. One of these is directed to the prevention of the further formation of acidiosis, due to acetone-producing substances, and the oxidation of those already found. The other is designed to deprive these substances of their poisonous properties. As already pointed out, the addition of moderate amounts of starchy foods to the diet results in the decrease or disappearance of acetone from the urine—acetone being the symbol of intoxication. If this cannot be done by the use of remedies by the mouth, because the stomach is unfit to deal with food, then one of the monosaccharids, such as levulose or dextrose, should be dissolved in sterile salt solution and injected subcutaneously or into a vein. Ordinary sugars (disaccharids) cannot be used in this way, because they require the action of the digestive juices to be disintegrated. The quantity of fluid used should be a quart with 10 per cent. of dextrose. Not less than 50 to 100 grams of levulose should be given a day by the mouth. If neither of these can be had, glycerin may be given by the mouth. Another point of importance is to cut down the fats given to these patients, who before the onset of these symptoms have been subsisting largely upon fats and proteids, because the poisons of coma are derived from fats and fatty acids.

For the diminution of the poisonous properties of the toxic substances already formed everyone is in accord that alkalies should be freely administered. Vichy water should be taken in large quantities, and 20 or 30 grains of bicarbonate of sodium may be given every two or three hours dissolved in Vichy water, thereby fortifying it. Stadelmann has advised the intravenous injection of carbonate of sodium in order that it may combine with the acids in the blood, diminish acidiosis, and aid in their elimination. For this reason the quantity of acetone in the urine may be temporarily increased by this plan of treatment. Naunyn uses 35 to 40 grams of çarbonate of sodium (not bicarbonate) dissolved in a quart of water. This must be given very slowly by intravenous injection. Even this plan, if instituted after coma is present, rarely does more than restore consciousness temporarily. If the bowels are confined they should be opened by some saline purge, but active purgation should not be resorted to, since by this means concentration of the poison may take place if the bowel is not active in the process of eliminating poisons from the blood.

DIABETES INSIPIDUS.

Definition.—Diabetes insipidus is a condition in which a person passes excessive quantities of urine containing no abnormal constituents and of a low specific gravity. This term is sometimes applied erroneously to a fleeting attack of polyuria due to nervousness or fright and to profuse diuresis following the ingestion of excessive amounts of water. It is also to be separated from the constant polyuria sometimes seen in hysterical women.

Etiology.—Diabetes insipidus is most commonly met with in persons under thirty years of age and may occur in early childhood. It is more common in males than in females. Very rarely it is definitely hereditary, and occasionally there is in the history of the patient a statement that it developed after some severe injury, as a railroad accident or fall. In some instances this may be due to damage of the central nervous system, in others to local nervous lesions. It has also followed sunstroke and prolonged fevers of an infectious type, and it has been met with as a symptom in cases of brain tumor.

The cause is unknown, but it is probably due to some condition of relaxation of the bloodvessels supplying the Malpighian tufts, with the result that an excessive quantity of fluid passes downward from these tufts through the tubules.

Morbid Anatomy.—No distinct lesions have been found constantly in cases of this character at autopsy, and nothing has been learned of the pathology of the malady by dead-room investigation. Sometimes the kidneys are found to be swollen and congested, but no real renal lesion explaining the polyuria has as yet been described.

Symptoms.—The dominant symptom of diabetes insipidus is, of course, a *profuse urinary flow*. Next to this symptom is the *constant thirst* suffered by the patient, who no sooner provides his system with fluid by drinking than it escapes from the kidneys. The third symptom of importance is the annoyance caused by the necessity of emptying the bladder many times a day and the loss of rest at night by reason of the same condition. Closely related to these symptoms in its causation is *dryness of the mouth* and excessive *dryness of the skin.* Partly because of the fact that the condition develops usually in nervous patients, or in those whose nerves have been shattered by accident, persons suffering from diabetes insipidus are often very *irritable* and *peevish,* an irritability which is maintained by the necessity of frequent micturition. The body temperature may be normal or subnormal. Tyson states that some cases can take inordinate quantities of alcohol without intoxication, but that others are unduly susceptible to the cerebral effects of this drug.

The quantity of urine passed by some cases of diabetes insipidus quite equals that passed by well-advanced cases of diabetes mellitus with free polyuria. As much as eighty and ninety pints a day have been excreted but the usual quantity is rarely above ten or twelve pints. The specific gravity is almost as low as ordinary water, and rarely exceeds 1.003 or 1.005, owing to the fact that the normal urinary solids are dissolved in such an exceedingly large quantity of fluid; but a high specific gravity alone does not indicate glycosuria, since diabetic urine may not be over 1.012 or 1.015. At times the total urea is greatly in excess of that normally excreted. Albumin is never present except in very small amount.

Diagnosis.—Before deciding that a patient has true diabetes insipidus it must be determined that the condition is not a fleeting polyuria, but a constant state. Tests as to specific gravity of the urine and for sugar will reveal diabetes mellitus. The state of the cardiovascular system and the eye-grounds may reveal chronic contracted kidney.

Prognosis.—Prognosis so far as life is concerned is good. Recovery is by no means rare, and even if it does not take place death from the malady rarely, if ever, occurs. The celebrated case of Willis lived fifty years with this condition present. It is only when the diabetes insipidus depends upon a serious nervous lesion that the prognosis is bad, and then because of the lesion and not because of the polyuria.

Treatment.—No treatment for diabetes insipidus which has yet been instituted has proved satisfactory. It is quite true that a large number of remedies have been spoken of in terms of praise by various practitioners, but the very number of them indicates that no one of them gives results which are definitely curative. The use of vegetable astringents, such as gallic acid, with the idea that by this means a diminution in the secretion of urine may be brought about, sometimes produces favorable results. The dose must be large, from 5 to 20 grains three or four times a day; but even when such large doses are used as to disorder the stomach, it not infrequently happens that no decrease in the quantity of urine is brought about. In other instances good results are said to accrue from the employment of an active fluid extract of ergot given in the dose of 20 to 30 minims three or four times a day, alone or with the bromide of sodium in the dose of 20 grains. The ergot is supposed to act by contracting the capillaries in the Malpighian tufts. When the polyuria causes much restlessness and insomnia, the remedies already named may be aided by the simultaneous administration of codeine, which will probably not decrease the quantity of urine, but which usually acts as a nervous sedative with sufficient power to prevent the bladder from waking the patient more frequently than is absolutely necessary. The bromides may also be used for this purpose. In those cases which are associated with neurasthenia or which follow prolonged nervous strain, the "Rest Cure" or a vacation where the patient is not annoyed by business or family cares will probably give better results than will drugs.

GOUT.

Definition.—Gout is a disease which depends for its existence upon a disorder of metabolism, as a result of which deposits of biurate of sodium take place in the joints and in the fibrous tissues surrounding them. It is characterized by associated changes of a fibroid and calcareous character in other parts of the body in many instances, and in its acute exacerbations it frequently causes severe inflammation and pain in one or more joints. The joint of the big toe is very commonly the chief seat of the articular disorder. Gout is sometimes called "podagra."

Etiology.—The precise cause of gout is unknown, but certain etiological factors in its development are universally recognized as being active. The first of these is undoubtedly heredity, but while it is true that the tendency to the disease is often inherited it is also true that the descendants of gouty persons often fail to develop the disease, and that other persons who have no gout in their family history suffer from the malady. It is interesting in this connection to note that younger children of gouty persons more frequently fall victims to gout than the children of their earlier years, probably because the gouty diathesis is better developed in advanced years in the parents than in youth.

A second factor in the production of gout is mode of life as to exercise and mental labor. There is universal accord that great mental and nervous stress with little physical exercise frequently produces a gouty diathesis and often precipitates an acute attack of the malady in those already gouty. Duckworth states that political life in England is notoriously conducive to gout, and that lawyers are very prone to it. In the case of the country squire who is so often gouty, high living and drinking, with a long heritage of dietetic indiscretion, probably overcomes all the good effects of an active out-door existence. In those who live chiefly out-of-doors, as farmers, soldiers, and sailors, the disease is rare.

A third factor of some importance is age. While cases of well-developed gout are met with in young children and, very rarely, even in infancy, the malady commonly does not develop till after the thirtieth year, but rarely waits till the fifth decade of life before at least beginning its early manifestations.

A fourth factor is the abuse of alcohol, not in the sense of going on sprees, but in such a manner that the system is all the time engaged in oxidizing or destroying this drug. Ales and beers—that is, malt liquors—are more prone to cause gout than are whiskies and other distilled liquors. Sweet and sour wines are also provocative of this disorder, particularly champagne.

A fifth factor is overeating. There is an increasing number of persons in America who do not eat to live, but live to eat, and who stimulate the digestive organs to greater activity by the use of highly seasoned dishes, with the result that they ingest and absorb more food than the system can use, stifling oxidation and clogging elimination.

Finally, a very powerful factor in producing gout, in those who are exposed to the metal, is lead poisoning of the chronic type.

We find, then, that the chief causes of gout are heredity, lack of exercise, nervous stress, and the ingestion of more food or drink than the body can properly deal with.

Frequency.—True gout in its frank forms is far less common in England than it was in the early part of the last century or in the eighteenth century. In America it is certainly very rare. On the other hand, both in England and in this country "lurking," "lateral," or masked gout is certainly greatly on the increase. The disease is more common in men than in women.

Pathology.—When we come to a study of this disease from the standpoint of perverted physiology or pathology, we encounter a task over which the profession has toiled unceasingly year after year with little advance in our understanding of gout, but great advance in our knowledge of the metabolic changes in the body. When Sydenham wrote, after being a sufferer from gout for years, that it is due to "the impaired concoction of matters both in the parts and juices of the body," he expressed himself as clearly and correctly as do many modern writers on this subject.

The pages of a text-book are not suitable for a discourse in which a multitude of researches are analyzed and judged, yet it is proper to take note of several theories as to the cause of gout that have been strongly advocated by one or more investigators, with some basis for their views.

One theory is that when the blood and lymph are saturated with uric acid the urates are precipitated by a slight lowering of the temperature, such as is apt to occur in an exposed joint. This theory is not adequate to explain the disease, because it has been proved that the fluids are not saturated with uric acid or urates in cases of gout, and the disease affects parts which are not chilled.

Again, Kolisch advanced the view that the so-called xanthin bases are the cause of gout. This investigator believes that the nucleinic bodies are broken up into xanthin and hypoxanthin, and that in the healthy kidneys these are in turn changed into uric acid. In gout he thinks that the kidneys fail to perform their function properly, that the xanthins are not transformed into uric acid, and that this results in the retention of xanthins, which straightway proceed to cause gout. This view has been impaired by the fact that his methods of research were faulty and by the fact that other investigators, using more accurate methods, get different results.

His believes that the uric acid of the gouty is a product of the disease, and that it is capable of causing evil effects in the body. It is not the prime factor, in other words, but a secondary factor, just as the bacillus of diphtheria is the prime factor, and the toxins which it produces cause widespread lesions as secondary factors. In the normal body uric acid is in large part destroyed; whereas, in gout it is permitted to exist and induce secondary evil effects. Any cause which prevents the destruction of uric acid predisposes the patient to its deleterious influences, and these causes may be inherited, acquired, or due to poisons, such as lead. Here, again, we are met by the contradictory fact that in a number of diseases an excess of uric acid develops without any signs of gout appearing. Thus, in leukæmia, pneumonia, and chronic kidney disease this acid circulates in the blood in excess, but no gout is produced.

The view of Ebstein is that there is a primary nutritional disturbance in the affected joints, and in other tissues, which results in tissue death within those parts, and that in these devitalized areas urates are deposited. Finally, von Noorden believes that a special ferment acts to produce these local nutritional changes and that the deposit of urates then ensues.

The most popular theory as to the cause of the symptoms has been that there is present in the body an excess of uric acid. This is the theory of Garrod, and in more recent times has had its most enthusiastic advocate in Haig. Garrod's theory that the decrease of uric acid in the urine at the time of an attack is due to its retention in the body, and that this retention causes an outbreak, is now held to be erroneous, or at least is regarded with grave doubt, as is also his view that a decreased alkalinity of the blood causes a precipitation of the urates, for Magnus-Levy, Luff, and W. His, Jr., have all proved that the quantity of uric acid in the blood is not increased during or before an attack, nor is the alkalinity of the blood decreased. Still others have shown that the decrease in the excretion of uric acid just before an attack of gout is due, in part at least, to a decreased ingestion of food. These researches do not prove, however, that uric acid producing substances are not present in excess, and it is entirely possible that the scanty elimination of uric acid in many of these patients in the interval between attacks is due to the failure of the body to change them into uric acid, with the result that they cause an attack, at which time the percentage of uric acid excreted often temporarily rises. I confess that this view seems the more attractive.

When we consider that the injection of uric acid into the blood does not cause gout, that it is present in leukæmia, pneumonia, and nephritis without causing gout, and that no excess of uric acid is found in the blood in gout, it is hard to believe that uric acid causes gout. While Kolisch's theory may be imperfect in detail, and while the kidney may not transform xanthin into uric acid, it is entirely possible that an excess of xanthin may be present in gout. This view is supported by several facts, which indicate that uric acid is an end-product derived from nuclein breakdown, and that it is not this healthy end-product, but by-products which are morbid in effect. From nuclein we can obtain albumin and nucleinic acid; from nucleinic acid we can obtain phosphoric acid and a substance which in turn may be split up into xanthin bases and uric acid. If oxidation is complete, uric acid is the chief end-product. if it is incomplete, then xanthin is the chief product. Bain and Futcher have both shown that when there is an increase in uric acid excretion there is an increase in phosphoric acid secretion, and they believe that this throws a side light upon the relation of uric acid and xanthin to gout. Thus, in one of Futcher's cases there was a marked fall in both phosphoric acid and uric acid immediately before an attack, followed by a very great increase at the time of the attack, and this again by a fall. The phosphoric acid curve was far greater than the uric acid curve.

It would seem, therefore, that the destruction of nucleinic bodies may be perverted in gout, and this is all that Futcher's study proves, namely, that as phosphoric acid and uric acid are both derived from nuclein, and as they are greatly disturbed in amount in relation to the attack, it is fair to assume that some relation exists between nucleinic bodies, their derivatives, and gout.

At the present time it would seem probable that we may divide this question of the pathology of gout into two sub-questions, viz.:

1. Is there present in the body at the time of an attack of gout an excess of uric acid, or, rather, of material capable of producing uric acid? The answer is "Yes."

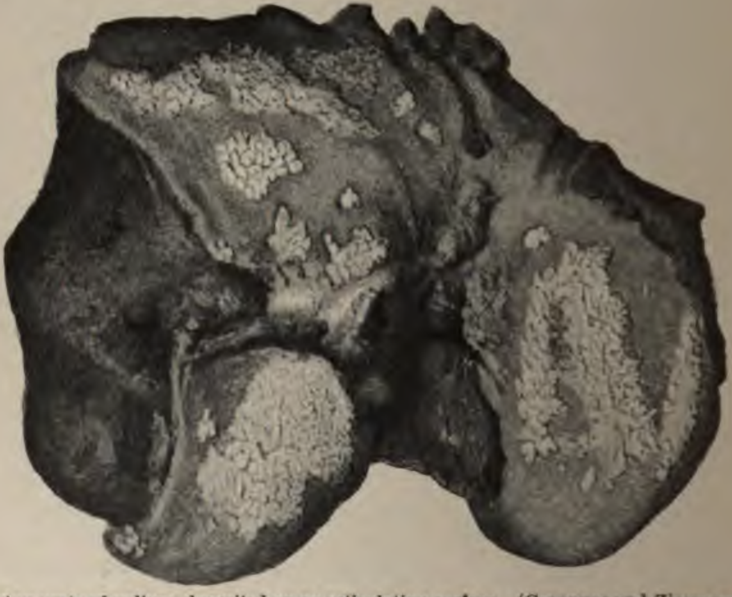
2. What is the reason that this condition develops? The answer is that we do not know, but that it is dependent upon a perversion of metabolism not yet understood, whereby in health uric acid, a primarily harmless body, is produced, and in disease a by-product is found, which causes an attack during, or before, which urate of sodium is deposited in the tissues.

Morbid Anatomy.—Gout may produce morbid changes in every tissue of the body, even to the hair and nails, but the parts which are most frequently impaired by its existence are the heart and the bloodvessels, the kidneys and the joints. From the standpoint of outward manifestations and early discomfort, the joint changes are, of course, the most important, but from the standpoint of the physician, whose duty it is to prolong life, the cardiovascular and renal changes are the factors which deserve most attention. Because of the wide distribution of gout in the body, Sydenham wrote: "Totum corpus est podagra."

The lesions in the joints are characteristic; the ligaments, tendons, and bursæ all become affected, and even the articular cartilages are involved. In all these tissues deposits of biurate of sodium take place, and they may be so copious that the parts are deformed and incapacitated by roughening of the cartilaginous surfaces, or by thickening of the sheaths of the tendons and joints. In typical cases the disease first attacks the joint of the big toe, then the finger joints, after this the metacarpal and metatarsal joints, and still later, but much more rarely, the large joints. When these are affected, the wrist, elbow, and knee are the parts usually involved. The ball-and-socket joints (hip and shoulder) are very rarely involved in gout, and the upper extremities are much less frequently affected than the lower ones. When the disease is well advanced there is so great a deposit of biurate of sodium that it lies under the skin in a white knob, or pea-shaped mass, which looks white and chalky. If the skin covering such a deposit is injured, it not rarely undergoes necrosis, and biurate of sodium exudes from the part, looking like wet chalk. Sometimes on the fingers, near the base of the finger-nails, or about the first phalangeal joint, there develop small, hard, uratic masses called "crabs' eyes." It is a remarkable fact that these deposits often take place without any other manifestation of gout being present and without any pain, so that attention may be called to them only by reason of the disfiguration produced. Gouty deposits about the base of the finger or at the second joint are, however, usually part of a general gouty outbreak.

When the articulating cartilages are affected two conditions may be presented. If no injury has occurred, and if no cause of irritation has existed other than the gout, the articulating surface is seen to be smeared or plastered, to use Duckworth's expression, with a uratic deposit looking as white and smooth as fresh white lead (Fig. 105). When irritation has been present the articular cartilage may be eroded. In this latter form there is often overgrowth of connective or fibrous tissue in the surrounding parts along with the deposit of urates, as already described, and stiffening or distortion of the joint. The changes in the bursa are often noteworthy. I have a case now under treatment in which there is a bursa swollen to the size of a bantam's egg on the heel at the insertion of the tendo Achillis. It is a very dusky red and is exquisitely sensitive, but contains nothing but fluid. It has often developed before, and under active treatment for gout has always disappeared and left no trace behind it.

FIG. 105



Showing urate of sodium deposited on an articulating surface. (Graupner and Zimmerman.)

The cardiovascular changes caused by gout do not result from the deposition of biurate of sodium in the heart-valves or in the bloodvessel walls. Indeed, it is rare for such deposits to be found, although cases are recorded in which the cardiac valves and the intima of the aorta have contained the biurate of sodium. But while it is true that biurate of sodium is not deposited in the vascular system, as it is in the joints, it is also true that gout causes first arterial spasm, then arteriocapillary fibrosis, and, finally, advanced calcareous changes in the vessels. The result of this is cardiac hypertrophy on the left side of the heart in particular. The endocardium is never the seat of acute endocarditis as a result of true gout. The only endocardial changes are those common to ordinary cases of general atheromatous degeneration, in that sclerotic changes take place in the valves, particularly those guarding the aortic and mitral orifices. The chorda tendinea are also shortened by a similar process and rendered inelastic.

The pericardium is very rarely affected.

The venous system is prone to varicosities and calcareous plates may be present in the walls of the veins. These in turn may result in thrombosis and phlebitis.

Gout, cardiovascular disease, and angina pectoris are a wicked trio that bring many a noble man to death.

Whatever the gouty poison may be which causes changes in the general vascular system, that poison also damages the renal tissues as well. The bloodvessels of the kidneys are, of course, involved in the general vascular fibrosis, and we have the small, contracted kidney often called a "gouty kidney," because it is often the result of gout. Many years ago Ord and Greenfield showed that in two-thirds of all cases examined at autopsy in which there was gout in the great toe there was chronic granular kidney, and in the remaining one-third a condition allied to it. While it is quite true that deposits of biurate of sodium are sometimes found in the kidney structure in the region of the papillæ, and extending outward along the pyramids toward the periphery of the organ in whitish streaks, such cases are very rare when we consider the number of cases of gout and the number of cases of renal disease complicating its existence.

Symptoms.—These are best studied in *three divisions:* the *acute*, the *chronic*, and the *aberrant types*.

ACUTE GOUT.—In this form the fully developed symptoms are often preceded by several hours or days of *nervous irritability*, of *insomnia*, or of *general wretchedness* not easily described. In some cases *pruritus ani* is present, or itching elsewhere may annoy the patient and keep him restless at night. The *urinary secretion* is often *scanty* and the *bladder* may be *irritable*. In other cases these symptoms are entirely absent and the patient will recall, during his hours of suffering, that he has seldom felt as well as he felt for a day or two before the attack came on.

The attack itself usually consists in the sudden onset of *sharp pain* and inflammation in the *ball* of the *great toe*. The pain is very severe and stabbing in character, often extending upward into the foot. A *swelling* develops with surprising rapidity and the *skin* over it is *red, hot*, and *burnished*. In addition, the part affected is exquisitely sensitive, so that the pressure of the bed-clothes, much less that of a shoe, is insupportable. Not rarely a *distinct febrile condition* is present, the temperature reaching 102° or more. After a few hours the agony diminishes, the swelling decreases, and the patient is more comfortable, but within the next twenty-four hours the malady may return with fresh severity. This may persist for several days, but at the end of that time the patient is not only soon on the road to recovery, but feels better than for a considerable time before the attack, although the ball of the toe may be swollen and inflamed for some days longer.

There are three noteworthy peculiarities about these seizures, namely, that they usually *develop after midnight*, waking the patient from sleep; that although the inflammatory process in and about the joint seems furious in its severity, the part *never goes on to suppuration*, and the onset and disappearance of the attack is *followed* by *little* if any *disability* in the affected part. Only when repeated attacks take place is there developed much deformity of the area involved in the gouty manifestation.

Acute gout is nearly *always recurrent*. Sometimes it attacks the patient every few weeks, in other cases every few months, and in others every few years.

This form of gout is very rare in the United States, but frequent in England, although less so than it was many years ago.

CHRONIC GOUT.—Chronic gout as a distinct condition from acute gout does not really exist; that is to say, no sharp line separates this type from the acute form. Two types of it may be recognized. In one, repeated attacks of acute gout are connected with one another by modified gouty manifestations, such as stiffness and soreness in various parts, as in the wrists and elbows, or the maintenance of a certain degree of *low-grade inflammation* in the joint of the big toe. In the other form there are no acute outbreaks such as have just been described, but a gradual process of gouty thickening of fibrous tissues and an equally gradual deposition of *biurate of sodium about the tendons*, the joints, and in the articular cartilages. Similar deposits called tophi are found in the edges of the ears, and the "*crab's eye*" *formations* in the fingers already referred to are found.

The urine is scanty, the arterial tension is usually high, and the aortic second sound accentuated. (See Morbid Anatomy.)

In some instances sudden inflammatory attacks of moderate severity, as compared to the acute attacks in the big toe, develop in one or several of the large joints, as the elbow and knee, and may give rise to the belief that acute articular rheumatism is present, particularly as a rise of two or three degrees of fever may take place. I have seen a patient with severe nodular gout of the hands develop an attack of universal articular gout after having his hands baked in a hot-air apparatus, probably because the treatment caused the distribution of a mass of gouty material in his body. The alterations in the appearance of the joints in these attacks has already been described when writing of the morbid anatomy of this disease. They may resemble very closely that malady called arthritis deformans.

After this type of gout continues for years the patient comes to his death as a result of renal or cardiovascular disease, or by some acute infection, such as pneumonia, which finds a ready victim in one whose vital organs are already impaired. In other words, death is due to a terminal infection.

Not rarely patients with this type of gout are intellectually brilliant up to the moment of their final illness. The presence of the disease seems to be a spur to mental activity.

IRREGULAR GOUT.—Without this division of gout some modern physicians would be sadly at sea in diagnosis. It affords a loophole of escape when a patient insists on a diagnosis, and Haig deserves the gratitude of many practitioners for having popularized the idea of "uric acid as a factor in disease." There can be no doubt that many patients do present symptoms of aberrant or modified gout, but they are by no means as numerous as they are thought to be. The gouty poison is capable of producing almost as many symptoms and ailments as is hysteria, but not all the symptoms which are credited to it, and it is unfortunate that "uric acid" is so often a cloak to ignorance which so pacifies the physician and patient that a search for a true cause is discontinued. Uric acid is, as already stated, in all probability not the cause even in the true gouty cases of many of the symptoms presented, but rather the result of the metabolic disorders underlying the illness.

There can be no doubt, on the other hand, that gout really is responsible in many cases for the presence of a very large number of disorders in widely different organs of the body, such as eczema and other forms of inflammatory or irritative changes in the skin, notably pruritus. Duckworth speaks of a painful induration of the ala of the nose in some gouty persons. In the circulatory system the changes caused by gout, even in those who have no outward manifestation of the disease, may be very notable, as already pointed out.

In the article on gonorrhœal infection it has already been stated that a *suppurative urethritis* may be due to gout, and irritability of the bladder and renal stone may arise from this cause. So, too, it is not infrequent for diabetes mellitus to develop in gouty persons and to be modified in its course by anti-gout remedies.

Albuminuria is also frequently present as a result of the high arterial tension or of the nephritis produced by the gouty poison.

Gouty persons often suffer from sudden and severe attacks of *acute pharyngitis* or *laryngitis*, and from *acid dyspepsia*, and ophthalmologists constantly meet with *iritis*, *conjunctivitis*, and other inflammatory processes in the eye due to this cause. So, too, it often happens that a thickening of the tympanic membrane, which causes *deafness*, has its origin in this malady.

Finally, and by no means least important, gout or gouty tendencies often cause furious *attacks of neuralgia* and of *migraine*. Not rarely after such a seizure of pain the patient feels unusually well, just as he does after a frank attack of gout.

Retrocedent gout is a condition in which the gouty process suddenly leaves the toe or other joint and attacks some one of the internal viscera, producing, it may be, violent purging and vomiting or precipitating an attack of angina pectoris. In other cases the patient suffers from an asthmatic seizure. Sometimes a sudden uræmia makes it appear that a retrocedent gout has gone to the brain. As a matter of fact retrocedent gout is not often seen. A letter of inquiry sent by me to several eminent English physicians brought replies that they had rarely seen this accident occur, although they have frequently met with cases, as we all have, in which a man subject to acute gout of the toe has had a gouty angina pectoris after prolonged freedom from trouble in the toe.

Diagnosis.—The diagnosis of gout when it affects the big toe is a simple matter. When present in the form of dermal, ocular, otic, or muscular gout, the history of the patient and the character of the attack render a diagnosis possible; but when the polyarticular form of gout with fever develops, only careful study and the absence of heart changes will enable us to separate the conditions if we can find no gouty history and no gouty signs, as in tophi in the ears. Again, there may be some hesitancy in separating gout from chalky deposits and fixation of joints of arthritis deformans in which the articular process is somewhat inflammatory and painful, and in which fibroid changes in the connective tissue about a joint are present. Arthritis deformans is, however, a more surely progressive malady; it causes greater crippling of the patient; it is not characterized by chalky crab's eyes and tophi in the ears, nor by inflammatory attacks in the eyes or the great toe, nor in the tendons and bursæ.

Many of the cases of masked gout can only be diagnosticated by the improvement which is produced by proper dietetic measures and the use of therapeutic measures known to be beneficial to gouty persons.

As stated at the beginning of the discussion of irregular gout, remarkably various symptoms can be caused by this malady, but all curious symptoms should not be credited to it. As Duckworth well says: "Without doubt many morbid states have often been flippantly or erroneously set down to irregular gout which owned no such designation, and thus a cloak for ignorance has always been at hand to throw over careless observation, ignorance, or wilful misinterpretation of symptoms. As a consequence of such errors, some have come to regard even truly gouty manifestations, when not articular, as actually non-existent, and to deny the dependence of such upon a gouty habit. The latter error is no more to be condoned than the former, and it may be fraught with mischief to the sufferer."

Prognosis.—The prognosis of gout is better when it develops after forty than if it appears after thirty years of age. In many cases of frank gout the prognosis as to life is better than in the insidious form, for the latter often attacks the circulatory and renal tissues. Much depends upon the vascular system. If it is fibroid the outlook is bad, and if it is not the mere existence of gout need not shorten life unless alcohol is abused. Gouty persons, however, are not good "risks" in life insurance, as has been proved by several sets of statistics.

Treatment.—The treatment of gout is hygienic, dietetic, and medicinal. A further subdivision of the subject may be made into that which is devoted to the treatment of an acute attack and into those measures which are taken for the relief of the more subacute or irregular manifestations. The hygienic measures which are to be employed in the treatment of a person suffering from either gout with acute exacerbations, or suppressed gout, consist in such exercise as can be taken in the open air without at any time producing more than healthy fatigue. Exercise taken to the point of exhaustion is of course always deleterious, and particularly in those who are gouty, as it is prone to produce an acute attack or reduce vital resistance to such a degree that intercurrent maladies may develop. Golf, horseback exercise, and similar out-door pursuits are therefore exceedingly advantageous, but are not to be carried to an excess. These patients should also be directed to drink water in as large a quantity as may be taken without overloading the stomach at any one time. Many persons can take half a glass of water every hour without producing gastric discomfort or interfering with digestion; whereas, if they are content to drink only at meal times, but small quantities of liquid may be ingested. Water aids in producing a profuse urinary flow, and so helps to eliminate impurities from the body. If the heart is not feeble, hot baths or hot Turkish baths may be taken two or three times a week with advantage. Often such patients are greatly benefited by going to some of the health resorts where hot springs exist.

The dietetic treatment of gout consists in the exclusion of all sweet wines and of fatty or rich foods, and in the ingestion of meals which are sufficiently varied in character to maintain the appetite and adequate to maintain nutrition. The patient should, however, be particularly warned against an excessive quantity of food. In many instances gouty persons will be found to take quantities of food which are far in excess of those which are required to provide the patient with the 2500 to 3000 calories per day which he requires for healthy existence. All wines and beers are also disadvantageous for this class of patients, but sweet and sour wines and champagnes are peculiarly so. In regard to individual articles of food, it has been held in the past that red meats were distinctly more harmful than white meats; but, as has been pointed out in discussing the dietetics of Bright's disease, this view of the relative harmfulness of red and white meat is becoming obsolete. Chemical analysis fails to reveal any material difference between them. The point of importance is that the patient shall not eat an excessive quantity of meat, or meats, which is prepared in such a way that it is difficult of digestion, as, for example, larded game birds or larded beef. Of the various beverages cocoa is perhaps the best, but rich chocolates are harmful. Coffee is usually considered much better than tea. Indeed, Dr. Haig is quite confident that tea is an abomination for the gouty.

In the way of treatment by drugs, there are only three which can be considered as exercising an approximately specific influence, namely, colchicum, iodine in its various forms, and the salicylates. Of these the iodides and salicylates are most useful for the subacute or irregular forms of gout, while the colchicum is of most value for the purpose of combating an acute paroxysm. Most patients with constant, irregular, gouty manifestations do well if they take continuously, over a long period of time, 10 to 15 grains of salicylate of strontium three or four times a day, or 5 or 10 grains of iodide of potash or iodide of sodium at similar intervals. By this means gouty sore throat, gouty iritis and conjunctivitis, gouty stiffness of the various muscles, and gouty neuralgia and migraine may be modified or entirely relieved. If there is any tendency to acidity of the urine full doses of citrate or acetate of potash, 10 or 15 grains three times a day, should be given, well diluted with water. In some instances the urine is alkaline, and when it is so the patient feels heavy and depressed. These symptoms can often be modified by the use of 10 to 20 grains of benzoate of sodium in capsule three or four times a day.

Acute paroxysms of gout are to be treated by the administration of full doses of the wine of colchicum root, repeated in from six to twelve hours, the dose varying from 20 to 40 minims according to the irritability of the stomach and the idiosyncrasies of the patient to the drug. Not infrequently much better results will be obtained if before the colchicum a moderate dose of the compound extract of colocynth, such as 10 to 20 grains with 1 or 2 grains of extract of hyoscyamus, is given to unload the bowels. Colocynth is chosen because experience has shown that it seems to exercise a more beneficial influence in gout than any other purgative, and the hyoscyamus is used because it prevents the colocynth from producing griping pain. Not infrequently some relief may be obtained if the inflamed joint is wrapped with lint laden with a 50 per cent. ichthyol ointment. Luff has strongly recommended the following application for the same purpose: The entire foot is surrounded by a warm pack consisting of cotton-wool saturated with the soothing lotion, and then lightly covered with oiled silk. The following lotion may be employed:

℞—Sodium bicarbonate	℥iv.
Linimentum belladonnæ	f ℥iv.
Tincture of opium	f ℥iss.
Water	f ℥viij

Equal portions of this lotion and hot water should be used to saturate the wool which has been rolled around the joint, and the dressing should be changed every four hours.

It is important to remember that no local depleting measures are to be instituted under any circumstances. Blisters, leeches, and other forms of bloodletting are not only valueless, but dangerous, as they afford opportunities for infection.

Many cases of acute and chronic gout are also benefited if from time to time they receive moderate doses of calomel or blue pill, such as 3 or 4 grains of blue pill or a grain of calomel in broken doses. Many physicians at the present time also prefer the active principle of colchicum, colchicine, to the wine of colchicum root. It may be given in the dose of $\frac{1}{100}$ to $\frac{1}{25}$ of a grain every two or three hours in a case of acute gout, or four or five times a day in the subacute varieties.

Although the various salts of lithium are largely employed by some practitioners for the purposes of combating the various aberrant forms of gout, it is to be remembered that the popularity of these salts depends more upon the skilful advertising of tablet manufacturers than upon the actual experience of the profession. Those who know most about these salts have found that the lithium preparations do not act as well in gout as do the salicylates of sodium or potassium, and the idea that lithium has a peculiar affinity for the uric acid is, to a large extent, blasted by the knowledge that lithium has a greater affinity for the acid sodium phosphate in the blood than it has for uric acid; beautiful test-tube experiments to the contrary notwithstanding.

ARTHRITIS DEFORMANS.

Definition.—Arthritis deformans is a chronic disease affecting the joints, and characterized by trophic disturbances in their cartilages, in the ends of the bones, and in the synovial membranes. It is to be distinctly separated from acute rheumatism and from true gout, although in some cases it appears to be a sequel of acute rheumatic infection. Sometimes arthritis deformans is called "rheumatoid arthritis," or "osteoarthritis."

Etiology.—Much discussion has arisen as to the cause of this malady, some adhering to the view that it is due to nervous lesions which result in changes in the joints, and others asserting that it is the result of an infection. Within recent years the former view has lost in popularity and the latter opinion is now in the ascendant, although as yet no one has succeeded in isolating the responsible micro-organism.

In support of the nervous theory we find that trophic changes take place not alone in the joints, but in the muscles and skin near the joints affected; that pain is often present in the nerve trunks, as in neuritis, and that diseases of the central nervous system not rarely produce lesions in the joints which resemble those found in arthritis deformans. Certain French clinicians have claimed that definite lesions are demonstrable in the spinal cord, in the columns of Goll in the cervical level, and in the posterior nerve roots as well. The difficulty in accepting the neural theory is that there is no proof that these changes are primary and not secondary, for it is well known that many joint affections are followed by similar lesions, at least in the nerves, skin, and muscles.

Those who argue in favor of the infectious nature of the malady point to the fact that the nearby lymph nodes are often enlarged, as if combating infection; that infection of the joints by various micro-organisms is the usual cause of arthritis, and that a considerable portion of the cases of arthritis deformans have had at some previous time gonorrhœa or other disease which is prone to cause secondary arthritic lesions. Ballantyne, Wohlmann, and Bloxall have isolated a bacillus, of dumb-bell shape, which stains with fuchsin or methylene blue, but not by Gram's method. Other investigators have failed to find it; Chauffard and Ramond have isolated a diplobacillus that they think is the cause. Organisms have also been found by Charrin, Le Roux, and Bouchard. Poynton and Payne have isolated a diplococcus from the synovial membranes in chronic arthritis, and by inoculation have produced the joint lesions of rheumatoid arthritis in animals.

Most clinicians divide the cases of this disease into two classes: the primary, in which the arthritic state develops without any preceding joint affection, and the secondary, in which there is a history of such an infection. The primary cases are those in which the disease develops after a prolonged nervous stress or severe physical strain, as in frequent childbearing. The secondary form follows acute articular rheumatism and occasionally develops after injury to a single joint, although in this case the relationship of cause and effect is very doubtful. Even in the cases in which acute rheumatism has preceded the malady this relationship is in doubt, and there is nothing to prove that there exists any closer tie between them than sequence by coincidence. To sum up our knowledge of the etiology of arthritis deformans, we may say that we really know but little of it.

Statistics as to the frequency of arthritis deformans in the two sexes differ greatly. In Garrod's statistics the disease affected 411 women out of 500 cases. Stewart has recorded 40 cases, of which 20 were in women; and more recently McCrae has reported 110 cases, of which 55 were women and 55 were men. My own experience at the Jefferson Medical College Hospital has been that nearly all are women.

The greatest prevalence of the disease is between twenty and fifty years of age, but Moncorvo has collected 48 cases of polyarthritis deformans in patients under fourteen years.

The disease is very rare in the negro race. In a clinic rich in negroes McCrae only met with this disease in negroes four times in 110 cases, which is all the more noteworthy because negroes are peculiarly subject to other arthritic changes.

Morbid Anatomy.—What the changes are in the early stages of this malady is not known, because patients rarely come to autopsy until after the disease has existed for a long period. What little information we have indicates that the process is primarily inflammatory, in that the synovial membranes are injected and hyperæmic. This is followed by the development of fibrous tissue, and this again by the absorption of the cartilaginous coverings of the ends of the bones. By these means cartilages become eroded and the ends of the bones become eburnated. Finally, there develops from the periosteum covering the ends of the bones, bony growths or knobs which lock the joints and because of the thickened membranes and roughened cartilages increase the tendency to immobility already present. These osteophytes are called "Haygarth's nodosities." Associated with these changes there is marked wasting of the muscles which govern the movements of the joint, a wasting not rarely met with in all serious joint affections, a glossiness of the skin covering the affected joints due to atrophy of the glands in its deeper layers and to changes in the epiderm, and not infrequently trophic changes in the nails which may be greatly thinned, ridged, or very brittle.

The changes in the vertebræ consist in overgrowth of the articular cartilages, followed by ossification. The ligaments also become thickened or atrophied. Bony formations may appear on the edges of the vertebræ, particularly on their anterior surfaces, and finally in this way the whole vertebral column becomes an inflexible pillar composed of vertebral bodies welded together.

Symptoms.—The symptoms vary considerably in the manner of their onset and in the course of the disease in different cases. The mode of *onset* may take *two forms:* the *slow progressive* or gradual type and that *type* in which a series *of attacks of articular distress* occur which leave behind them more and more chronic change in the joints. In some patients the first complaint consists in a *sensation of roughness* in the knee-joint; the part feels stiff after it has been in one position for any length of time, and "cracks" when it is moved. Not rarely this is first noticed in going up or down stairs or in rising from a chair. If the joint is flexed by the physician who holds the limb, the cracking can be felt by him. The sensation is one of distress more than of pain.

The tissues about the joint are often swollen, but rarely if ever reddened. Usually after one joint is affected another becomes involved, and, as the process gradually develops, the patient becomes more and more incapacitated owing to the advancing changes in the joints and the new areas affected.

The atrophy of the muscles also increases the inability to move about with ease.

Finally, the disease involves all the articulations, large and small, even the vertebral joints being affected, and the patient is as completely paralyzed from fixation as if suffering from a widespread multiple neuritis. Such a result, however, does not occur in most instances, the malady being less widely distributed.

It is a noteworthy fact, for which patients with this disease have cause to be devoutly thankful, that in the cases with greatest fixation of the large joints the small joints of the hand often escape, and when the hands are severely affected it often happens that the large joints are not involved. In still other cases the disease after incapacitating one part ceases to be progressive. The frequency with which pain occurs in these cases is very variable. In some there is none, in others it appears only when the parts are used, as in piano-playing, and in others the pain is severe and like that of neuritis.

Free sweating of the palms of the hands and finger-tips often is present.

Because of the fact that the manifestations of the disease are often limited to one part of the body or to one kind of joints, it has been customary to divide the cases into those with only one joint affected, the monoarticular form; those in which several joints are chiefly affected, the polyarticular type; and those in which the spinal column is the chief seat of the disease, the so-called spondylitis type. When the joints of the fingers are chiefly affected, so that they are locked by the growth of small, bony knobs on the sides, these growths are called "Heberden's nodes." An important difference between these nodes and those due to the deposit of urates, as in cases of gout, is that they are true bony growths, and not due to the deposition of urate of sodium in the fibrous tissues. It is easy to see that this classification is purely one of convenience, and has no real pathological reason for its existence.

When the large joints are involved, with associated fixation of spine, the patient lies in bed helpless, with the knees flexed and the back so stiff that it has been called "poker back."

Under the name of *spondylitis deformans* von Bechterew has described a state of spinal fixation in which there is pain, compression of the spinal nerve roots, and muscular atrophy. Ascending spinal-cord degeneration is also present. It is questionable if this can be considered a part of true arthritis deformans. So, too, under the name *spondylitis rhizomelique*, otherwise known as the Strümpell-Marie type of spinal fixation, the muscles of the shoulder and hip joints are involved, but severe nervous lesions such as those just detailed do not develop. Osler believes that both of these affections are forms of arthritis deformans. This may be a correct view, but, if so, they do not present any symptoms common to arthritis deformans as it is generally met with.

A form of general arthritis without exostosis, but with considerable swelling of the joints, is sometimes met with in children. The lymphatic glands are distinctly enlarged, the mobility of the joints greatly impaired, and the atrophy of the muscles well marked. Not only the lymph nodes nearest the joints, but all the lymphatics may be swollen. Anæmia is marked. To this condition the name "Still's disease" has been applied, because it has been carefully studied by Dr. Still. Children may also have the polyarticular form of arthritis deformans as it occurs in adults.

Attention must be called to that type of arthritis deformans in which the vertebræ suffer alone. In such instances the spine is fixed and immovable, the ankylosis producing so much rigidity that the patient in stooping can only bend at the hips. When in addition to fixation deformity takes place, the spinal column may be deflected laterally or posteriorly, and the arching of the back may be so great that the head is bowed till the chin nearly touches the chest and the patient can only see a short distance ahead by rotating the eyes upward. Lateral rotation of the head in such a case is often impossible. Associated with this form of vertebral arthritis there is often a good deal of pain, which may be felt not only in the back, but in the hips and even down the course of the sciatic nerves. In those cases in which the disease is limited to a small portion of the spinal column the case may be considered as one of tuberculous disease, and the only means by which a positive diagnosis can be reached is by the use of tuberculin, if no changes in other joints point to the malady now under consideration.

Diagnosis.—When a well-developed, bedridden case of arthritis deformans is met with, the patient being "fixed," and the bony knobs locking the joints, the diagnosis is not difficult. The difficulty lies in the cases which are in the early stages of the malady. This is particularly true of the cases which are of the subacute type, and which are ushered in by a somewhat abrupt onset, with soreness, stiffness, and pain in the affected parts. The differentiation of such cases in the first attack from a subacute form of true articular rheumatism may be practically impossible. If an endocarditis develops, the disease is probably rheumatism, for the endocardium usually escapes in rheumatoid arthritis. Another point of differentiation lies in the fact that acute articular rheumatism passes from joint to joint, often diminishing in the first joints as the others are involved. This is also true of arthritis deformans, save that the joints first affected do not get well. The pain of rheumatism is greater and the parts affected are more tender to touch. Finally, if the articular difficulty persists week after week, in the face of the salicylates freely given, if the temperature creeps along at about 99°, and if there is crackling in the joints on flexion, the diagnosis of arthritis deformans is probably correct.

Rheumatoid arthritis also must be differentiated from the arthritic changes due to gonorrhœal infection and those of locomotor ataxia.

Prognosis.—A well-developed case of arthritis deformans is absolutely incurable, and the outlook is gloomy as to freedom of movement. The fact that the disease is often characterized by very slow progress and by periods of arrest is the most encouraging feature of these cases.

Treatment.—From the stage of onset the treatment consists in the use of gentle massage of the joints with 50 per cent. ichthyol ointment, this ointment being also thickly smeared over the part between the times at which the rubbing occurs. If pain is present acetanilid or antipyrin may be used. If much stiffness is present the joint should be baked in a hot-air apparatus every day or two.

It is a vital mistake to give these joints rest as a part of treatment. Barring use to the point of fatigue the patient should be told to move the affected parts as frequently as possible.

Fresh air and sunshine are essential factors in controlling the anæmia often present. A valuable remedy, probably the best we have, is syrup of the iodide of iron in 30 or 60 drop doses three or four times a day well diluted with water and taken two hours after meals.

When deformity which interferes with movement is present it may be wise to cut contracted tendons, but this does not increase mobility of the joint, for obvious reasons.

Many patients resort to hot springs and hydrotherapeutic institutes for relief from this disease. Such a plan of treatment is good for the general health and may seem to arrest the process, but it cannot cause a cure, and if the patient's means are limited it is useless to let him waste them in a futile search for health.

CHRONIC RHEUMATISM.

Definition.—Chronic rheumatism is a condition characterized by a chronic low-grade inflammatory process in the synovial membranes and fibrous tissues about the larger joints which results in stiffness, soreness, and disability. It may or may not be a sequence of acute articular rheumatism.

Etiology.—The causes of this state are unknown. In many cases it seems to be part of the general fibrous change which occurs in other parts of the body due to old age. Exposure to cold and wet, as in the case of soldiers, firemen, and policemen, seems to be productive of the affection. The malady is very common among old negroes. It often causes great crippling.

Morbid Anatomy.—The chief change consists in thickening of the fibrous tissues about the joints, but there is little effusion, as a rule, and no deformity aside from inmobility or fixation in an awkward position until secondary muscular atrophy ensues, as it usually does in all chronic joint affections. Occasionally the articular cartilages undergo changes and some crepitus can be felt on motion.

Symptoms.—*Stiffness* and *inability* to move readily are the chief symptoms complained of. These are worse when there is a threatened change of weather. If sudden movement is attempted pain is caused. Rest increases the stiffness and exercise greatly decreases it, but, on the other hand, sufficient exercise to limber the joints is often very painful, either at once or because of the exaggerated degree of stiffness that may develop. Occasionally these patients, particularly if they be women, become exceedingly obese.

Treatment.—Unlike arthritis deformans, this malady gives us the best therapeutic results, in many cases, if the patient can be subjected to the salutary influence of the Hot Springs of Arkansas, Virginia, South Dakota, Banff, in Canada, or at Bath, in England. The various sulphur springs are useful in many cases. When these springs cannot be resorted to the patient should have the affected joints baked in a hot-air apparatus, or should take a Turkish bath several times weekly. A well-constructed Turkish bath cabinet, such as is widely sold at present, may be used at home. A 50 per cent. ichthyol ointment should be well rubbed into the joints twice a day. This may be alternated with chloroform liniment containing 4 drachms of tincture of belladonna and 2 drachms of tincture of aconite in 8 ounces. In other instances iodine ointment diluted one-half with lanolin may be used.

The use of drugs internally is not very satisfactory as to results. Pain may be decreased by acetanilid or antipyrin. The salicylates are rarely useful, but the iodides often cause great improvement. There is little use in dieting these patients. If they eat and drink sensible things the ordinary diet of health is the best. Alcohol should of course be used but little, if at all.

Warm flannel should be kept next the skin, and cold winds avoided if possible by residence in a balmy climate in the winter months.

MUSCULAR RHEUMATISM.

Under the name of muscular rheumatism there exists a condition in which a patient experiences pain and stiffness when attempting to move certain muscles. This condition is most frequently met with in patients who become chilled after severe exercise. It sometimes develops when certain portions of the body have been exposed to cold and wet, as, for example, the stiff neck which follows sitting in a draught of cool air or the lumbago which follows exposure to a storm.

The exact cause of muscular rheumatism is not well understood. It has been claimed by some persons that the liquids of the body contain so large a quantity of toxic materials, produced by perverted metabolism, that when a part is exposed to cold precipitation of these toxic materials takes place, either in crystalline or amorphous form, and this causes stiffness and pain.

Treatment.—The treatment of muscular rheumatism depends somewhat upon the condition which has produced it. If it has followed exposure to cold, a very hot bath, a Turkish bath, or a hot pack will often serve to dissipate the pain and stiffness, particularly if after the bath active massage of the part affected is practised. In still other instances, particularly if the onset of the condition has been very acute, relief is obtained by acupuncture. This consists in introducing into the substance of the affected muscle a long needle, which does good, it may be, by relieving tension in the sheath of the muscle, and allowing some of the serum to escape. In many cases relief is obtained by rubbing the affected part with chloroform liniment, to which may be added tincture of aconite, tincture of belladonna, and tincture of opium. Equal parts of chloral and camphor, which deliquesce when they are mixed, may be rubbed over the painful area. Before giving medicines internally, the urine should be examined. If it is distinctly acid, the patient should receive from 10 to 20 grains of bicarbonate of potassium in water three or four times a day. In other instances similar doses of bicarbonate of sodium will prove equally efficacious. In still others it may be necessary to administer one of the salicylates. Thus, much benefit will often follow the use of 10 or 15 grains of strontium salicylate given three times a day. Often it is advantageous to combine with this 4 grains of acetanilid or the same quantity of phenacetin or antipyrin. In obstinate cases, or in those in which the symptoms frequently occur, the use of iodide of potassium or iodide of strontium in the dose of 5 or 10 grains three times a day is advantageous, and sometimes the patient requires the additional use of $\frac{1}{100}$ of a grain of colchicine three or four times daily. It is not to be forgotten that some of these patients, particularly those that suffer from lumbago, owe their condition to autointoxication from the alimentary canal, which is due to constipation, and in such patients permanent relief will not be obtained until the bowels are thoroughly moved each day. In some cases the use of one of the saline purgatives is all that is necessary.

RICKETS.

Definition.—Rickets, or rachitis, is a nutritional disease of childhood, consisting in a general perversion of the processes by which normal growth takes place. It is chiefly characterized by imperfect osteogenesis. The ends of the bones are often larger than normal, and there is faulty growth in the cartilages, muscles, and tendons.

Etiology.—Two factors are chiefly concerned in the production of rickets, namely, dietetic faults and bad hygienic surroundings. Of these the most important are errors in diet. On the other hand, it would seem that in some cases at least the diet may be correct, yet the faults in nutrition exist because the system cannot utilize the materials which are ordinarily employed in the production of normal tissues The majority of children suffering from rickets improve as soon as their food is properly adjusted.

In the article on Scurvy attention has been called to the fact that while rickets affects both rich and poor, it is chiefly seen in the latter class, whereas scurvy in infancy is frequently a disease of the well-to-do. In many cases, however, scurvy and rickets exist simultaneously.

The most common fault with the diet consists in the use of proprietary foods for infants, in the use of condensed milk, and in the carrying on of lactation until the period is past when the child can obtain sufficient nutriment from a breast which is secreting a poor quality of milk.

It is interesting to note in this connection that valuable observations have been made upon young animals as to the effect of depriving them of ordinary diet and providing them with one not suited to their needs. Thus, Bland Sutton fed lion cubs with raw meat and rickets developed. When a diet of milk, cod-liver oil, and powdered bones was given them they speedily got well. Experiments made upon other animals have given like results.

Edlessen holds that the disease is of infective origin. Mendel thinks it depends on some change (atrophy) in the thymus and has formulated a system of treatment based on the thymic origin of the malady. Spillman rejects the chemical theory and holds that the deficiency in lime-salts is a result and not the cause of rickets. There has been much unprofitable discussion as to the congenital origin of the disease. Fede and Finizio found, among 975 newborn, 4 that might have been called cases of fetal rickets;

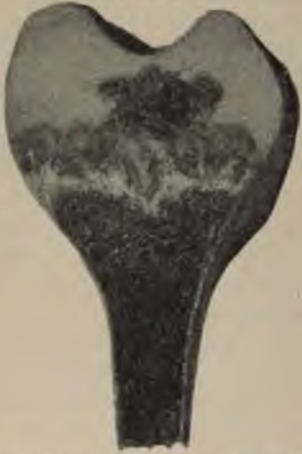
but most of the cases of so-called fetal rickets are instances of achondroplasia.

Age and nationality are factors of some importance in the causation of the disease. Holt states that it is very common in the children of the poorer class of Italians found in all our large cities, and it is certainly very common in negro children. In both of these classes the race has not so much influence as poor food and poor surroundings.

The disease is rarely met with in children over three years of age, but the signs of its existence often persist in the shape of bony deformities all through life. Cases of rickets have been reported in children at the time of birth and as old as twelve years. Both sexes suffer equally severely from it.

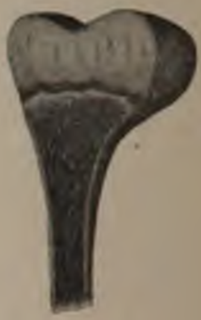
Diseases which impair nutrition may predispose the child to the development of rickets, but they do not produce it. Thus, congenital syphilis may act as a cause, but is not directly responsible.

FIG. 106



Rachitic epiphysitis, showing proliferated cells in the epiphysis. (Graupner and Zimmerman.)

FIG. 107



Syphilitic epiphysitis.

Morbid Anatomy.—While it is true that we know little of the underlying cause of rickets, we have very clear and definite knowledge of its morbid anatomy. In healthy children the bones grow longitudinally by the development of bony materials at the space between the diaphysis and the epiphysis, and in breadth by the deposition of bony materials by the inner layers of the periosteum. The medullary canal is increased in diameter by the gradual absorption of the adjacent bone. In rickets the process is abnormal in that although the development of cells at the points of growth may be carried on to excess, the deposition of mineral matter to form bone is inadequate. The resulting structure is softer in texture and more vascular than normal, that is the osteoid tissue is perverted (Fig. 106). It happens, therefore, that while the length and breadth of the bone is being abnormally developed, the lemallæ adjacent to the medullary cavity are being absorbed, thus leading to structural weakness. Under these conditions it is not difficult to understand why it is that the bones are unable to bear the normal stress and rapidly become deformed if any strain is placed upon them.

Again, the process of development may be so abnormal that areas of bone are found in the midst of cartilage and far in advance of the edge of the normal bone growth, or the reverse holds true, and in the midst of bone there may be a cartilaginous mass with little mineral matter in it.

Histologically there is excessive proliferation of the cartilages preliminary to ossification with irregular distribution of the columns of cells, excessive vascularity, and imperfect calcification. These enlargements seem particularly marked at the ends of the long bones and at the costal ends of the ribs and consist of this bone-like (osteoid) tissue, which later solidifies and perpetuates deformities of which it was originally the cause.

In addition to the deformity of the long bones produced in this manner we find similar changes occurring in the bones of the head and pelvis. Not only do the fontanelles remain open for a longer time than is normal, but in addition the surfaces of the bones are found to be soft and porous and unduly vascular. In some places the occipital bone may be irregularly developed and spaces exist which are closed by membrane alone. This condition is called " craniotabes." The frontal bones are often large and bulging, forming the so-called "bossy frontals" described by the English clinicians.

The actual deformities which result are bending of the long bones, particularly in the legs; the development of pigeon-breast, with an increase in the anteroposterior depth of the chest, and the production of spinal curvature. A very constant state is the presence of swellings or enlargements of the ends of the ribs where they join the cartilages, producing the knobs or the so-called " rachitic" rosary. The spleen and liver are increased in size and Mendel thinks this is compensatory for the loss of thymic function. Mettenheimer and Friedleben describe atrophy of the thymus in rickets.

Symptoms.—The symptoms of rickets, when the condition is well developed, are very characteristic and can scarcely be overlooked even by the most careless observer. In the earlier stages, however, the manifestations of the disease are not so evident, and yet they are important in that they should place the physician in a position in which he can prevent further advance of the malady. These early symptoms consist in *sweating of the head,* so that the child's pillow is wet with the perspiration, and *restless sleep,* with *grinding of the teeth.* Partly because of the wet pillow and partly because of lowered vitality, rachitic children are very *prone to colds* and often suffer from constant catarrh of various mucous surfaces. *Constipation* is usually a marked symptom.

In addition to these symptoms a physical examination of the patient will reveal in some cases in the early stages *beading of the ribs,* that is, *enlargement of the junctures* of the *costal cartilages* and *ribs,* the so-called "*rachitic rosary.*" The forehead may be full and large from the bulging of the frontal bones, the so-called "bossy frontals," already referred to, and the *belly is bulging* and *tumid.*

If the condition is far advanced all these signs are more marked, and in addition there may be found wide-open fontanelles and soft spots in the skull due to craniotabes. This state of craniotabes is much more marked in those cases which suffer from syphilis, and some observers have asserted that it only occurs in children who have inherited this malady. Auscultation over the open fontanelles may reveal a *humming murmur* synchronous with cardiac systole. Anæmia may or may not be a marked symptom.

The *nervous symptoms* consist in *laryngeal spasm* (spasmodic croup) and great irritability. *Epileptoid fits* or *tetany* may develop.

The shape of the thorax is also modified so that the lateral diameter is decreased and the anteroposterior diameter increased. This gives it a somewhat bulging or "chicken-breast" appearance. In the neighborhood of the ensiform cartilage there is seen, in some cases, a shallow depression of the costal cartilages and ribs, which extends outward and upward toward the axilla on both sides. This depression covers the space of from one to three ribs and is called "*Harrison's groove.*" It is chiefly due to protrusion of the lower ribs, which are pushed outward by the bulging of the abdominal wall. This deformity is most marked in children who suffer from spasmodic croup and obstruction of the upper respiratory passages. Palpation of the chest wall may reveal the fact that it is abnormally pliable or yielding.

If the child is old enough to walk the long bones become deformed because they bend under the weight of the body. For this reason the *bones* of the legs may be *bent* and badly curved anteriorly or laterally. There may be *posterior curvature* of the spine.

Dentition in rachitic children is usually considerably delayed, and is accompanied by gastrointestinal disorders, chiefly because a tendency to catarrh of these parts is always present. The teeth when cut are usually fairly well developed and do not readily decay. This is in contrast to the history of syphilitic infants, who often cut their teeth abnormally early and then promptly suffer dental decay.

Several of the symptoms described, while often found when rickets is present, are by no means characteristic of this disease, and occur frequently in other conditions. These are the craniotabes of syphilis, the laryngeal spasms, the systolic cranial murmur, restless sleep, and grinding of the teeth.

Diagnosis.—Rickets should be separated from scurvy, with which disease it is very nearly related, and which may be present simultaneously. In scurvy the nutrition of the mucous membranes and of the bloodvessels seems to be chiefly involved, hæmatoma and purpuric rashes often develop, and bleeding gums are seen. These lesions are practically never seen in rickets. There is great muscular soreness on moving the child, and paraplegia is more commonly met with in scurvy, although in both states this palsy may be present. Again, the scorbutic child rapidly improves when given fresh orange-juice and beef-juice. Palsy may be due to acute anterior poliomyelitis, but if a careful study of the case is made it will be found in rickets that the muscular weakness is universal, whereas in poliomyelitis it is limited to certain groups of muscles and the associated rachitic symptoms are absent.

Prognosis.—The prognosis as to life is good so far as the rickets itself is concerned. That is to say, death is never due to rickets. On the other hand, a child with rickets is a fair mark for every infection to which it may be exposed, and so the mortality of these cases from intercurrent maladies is high. If marked deformities exist they of course persist through life, except they be in the extremities, when they can be corrected by surgical procedures.

Treatment.—From what has been said it is evident that the first thing to be done in a case of rachitis is to regulate the diet, and to see to it that the patient receives foodstuffs which contain all the ingredients which are necessary for the maintenance of normal nutrition. It not infrequently happens that the milk which is given to the child is lacking in mineral ingredients or contains such an excess of casein that the child's digestion is disordered and assimilation is disturbed. The mere fact that the child does not subsist upon market milk, but upon milk which is obtained by keeping a cow for the special purpose of providing sustenance for the infant, is more indicative of a dietetic cause for the rickets than if the child is obtaining milk which is given by a number of cows, for it not infrequently happens that the milk of a single cow disagrees with the child or does not contain all the materials which are necessary for its proper growth. If the child has been largely fed upon proprietary foods, these should be eliminated from the diet list and fresh milk and cereals used in their place. In some instances, as already stated, the rickets does not depend upon a lack of normal ingredients in the food, but upon the inability of the child to utilize these ingredients. This may depend upon digestive disorder, or upon a disturbance of trophic function. Under these circumstances it is not only necessary to investigate the diet, but to administer tonics which will improve digestion, such as small doses of quinine, $\frac{1}{4}$ to $\frac{1}{2}$ grain, twice or thrice a day; minute doses of nux vomica, or small quantities of the hypophosphites or the more modern elixir of the glycerophosphates. Of the latter 10 to 25 minims may be given twice or thrice a day. In other instances $\frac{1}{200}$ grain of phosphorus given in a sugar-coated pill may be used twice or thrice a day; or in its place we may administer drachm doses of cod-liver oil, or cod-liver oil in the form of a well-made emulsion.

If anæmia is present 5 drops of the syrup of the iodide of iron may be given to a child of two or three years of age two or three times a day. As constipation is often a troublesome symptom in rickets, careful attention must be paid to the state of the bowels. They may be moved either by the use of a little calcined magnesia and followed by a few teaspoonfuls of orange-juice, or by one of the non-bitter preparations of cascara sagrada, such as aromatic cascara or cascara cordial.

It is important that a child suffering from rickets should be allowed to have exercise without bearing weight upon its long bones. Such a child should not be encouraged to walk, but should be placed upon a rug on the floor, where it can crawl and roll about. An endeavor on the part of the parents to teach such a child to walk when its bones are unable to bear its weight results in deformities which may be so severe as to require operative measures for their relief.

It is hardly necessary to add that fresh air and sunshine are essential in the care of such children. A few weeks at the seashore will often cause a remarkable change in nutrition; whereas the most skilful dietetic and medicinal treatment, if carried out with unfavorable hygienic surroundings, may produce no results whatever.

In families in which rickets is prone to occur it is often wise to administer to the mother during the later months of pregnancy the hypophosphites or the glycerophosphates, and other nerve and bone tonics, as by this means the antenatal nutrition of the child can be materially influenced. In a well-known case in Philadelphia the first three pregnancies resulted in the destruction of the mother's teeth and in the birth of children which speedily showed rickets; whereas, the last three pregnancies, during which a diet rich in mineral ingredients was provided, resulted in the birth of children which remained perfectly healthy.

SCURVY.

Definition.—Scurvy, or scorbutus, is a disease characterized by more or less profound nutritional changes in the body which are largely dependent upon the use of certain forms of unsuitable food. There may be extravasations of blood into the subcutaneous tissues, under the mucous membranes, and about the joints, and there is often great spinal tenderness when the disease occurs in infants.

Etiology.—There can be no doubt that scurvy is due entirely to the use of food which fails to provide all the substances needed for the perfect nutrition of the body. Not only does it follow the continued use of food which is bad in the sense that it is unwholesome, but it arises in those who are subjected to a very limited diet of certain kind for long periods of time. Prior to the introduction of steamships it was a prevalent disease upon vessels in both the navy and in the mercantile marine, often disabling the crews and rendering impossible commercial and exploring expeditions. The use of steam now causes short voyages, and the better method of preserving foodstuffs provides a change of diet almost daily, if it be needed.

In certain parts of Russia scurvy still occurs in epidemics and is thought to be infectious. If this be true it is probably only because lowered vital resistance permits an infection to take place.

Some investigators have believed that the disease arises from a lack of vegetable acids found in fruits and vegetables, others that the condition is due to an excess of sodium chloride in the blood, and still others that it is due to the presence of certain toxic substances developed in the food. Albertoni has recently shown that in scurvy there is a complete absence of free hydrochloric acid in the gastric juice and that intestinal putrefaction is marked, and that the absorption of fats and carbohydrates is impaired.

Pathology and Morbid Anatomy.—Anatomically there is nothing characteristic. The condition of the blood in scurvy resembles that of the blood in ordinary secondary anæmia with a decrease in the color index which is quite marked. If the case is studied at autopsy hemorrhages into the internal organs and upon the serous surfaces of the abdominal and thoracic viscera are found and ulceration of the small and large bowel may be present. Swelling of the gums, loosening of the teeth, enlargement of the spleen, and degenerative changes in the heart, liver, and kidneys are found. Subperiosteal hemorrhages may detach the periosteum over the shafts of the long bones and hemorrhage into the joints may also occur.

Symptoms.—The symptoms of scurvy in adults begin with a sense of *general wretchedness, pallor*, and *feebleness*. These are followed by *swelling* and *sponginess* of the gums, which may bleed if pressed upon and which may partly cover the teeth by a process of granulation. The *teeth* become *loosened*, the *mouth* becomes *foul*, the *salivary glands swell*, and *petechiæ* appear over the surface of the body. The skin is dry and badly nourished, and *extravasations of blood* may take place into the sheaths of the muscles and joints or beneath the periosteum of the long bones. These lesions at times cause patches of hardness or induration in the muscles of the thighs or calves. I saw this well developed in a case which I had in the wards of St. Agnes' Hospital some years ago.

Infantile *scurvy*, sometimes called "Barlow's disease," is an interesting result of modern life. As seamen have escaped the disease by a better diet, babies have fallen victims to it as a result of feeding them with artificial foods, and these babies are not, as a rule, the children of the poor, but the offspring of the rich.

It is only within the last decade that the possibility of scurvy occurring in young children has been generally recognized by the profession. During this period, however, evidence has been presented which shows very clearly that scurvy is by no means a very rare affection in early life, and that it often manifests itself in such a way as to lead the physician to make a very erroneous diagnosis when the patient is first brought to him.

Perhaps the most frequent error in diagnosis under these circumstances is that the child is suffering from muscular or articular rheumatism; this decision being reached by reason of the fact that the child seems to suffer great pain upon movement, and sometimes has a moderate degree of fever.

The characteristic symptoms of scurvy in a young child, when they are well developed, are so pathognomonic that it is difficult to see how an error in diagnosis can be made if the physician is acquainted with the possibility of the occurrence of this disease. Like all diseases, however, instances are met with in which many of the characteristic symptoms are entirely absent, and it is not uncommon for the painful manifestations spoken of to be the only evidence of the malady. In still others, we find a peculiar *spongy* state of the *gums*, which tend to bleed when lightly touched, and which are frequently so swollen that teeth which have recently broken through the gum are speedily covered in by the overgrowth of the mucous membrane, the edges of which, about the teeth, frequently look as if they were composed of *tiny blebs of blood* of a dark color. Another symptom of scurvy, which is by no means as constant, and yet which is equally characteristic, when it occurs, is the development of petechiæ in different portions of the body, very frequently about the ankles and feet. In still other cases *subperiosteal hæmatoma* develops with surprising rapidity, and as pain on movement and the development of great swelling are frequently first noticed after a fall or a blow, it not rarely occurs that the physician is led into the belief that traumatism is the cause of the illness, without recognizing the fact that it has played but a small part in causing the sudden development of a state which really indicates grave systemic conditions. It is true that these subperiosteal hæmatomata have been chiefly reported by French clinicians, and have been rarely seen in this country; whereas, on the other hand, considerable extravasations of bloody serum have been met with in the loose tissues after exposure to injuries which in the healthy infant would produce no symptoms whatever. Paraplegia may also be present.

The peculiar characteristics of scurvy in infants are the very grave appearance of the child when suffering from the disease in its severe forms, the rapidity with which it improves under proper treatment, and the rarity with which death occurs as the direct result of the malady, since a fatality is usually produced by some intercurrent disease. Reinert has, however, recorded a fatal case with red cells at 976,000 and hæmoglobin at 17 per cent.

Scorbutus in infancy is distinctly a disease of the children of the well-to-do in distinction from rickets which, on the other hand, seems to be a disease of the poor. Clinical experience, I think, indicates that scorbutic cases are rarely brought to hospital dispensaries while rachitic cases are constantly seen. On the other hand, scorbutic cases are not uncommonly met with in private practice. This clinical fact seems to carry out certain theories which have been advanced in regard to rickets in a way which is interesting. It is not many years since everyone believed that rickets was due to a deficient quantity of bone-forming material in the food of a child, but since that time other clinicians have stated their belief that more commonly it depends upon inability of the child to assimilate and utilize the ingredients in its food which it needs for proper bone growth. Or, in other words, the fault lies not with the food, but with the child. Among the poor this inability is probably due to unhealthy surroundings and a general lack of sanitation which interferes with development. On the other hand, such influences are not at work among the children of the well-to-do, but these children often receive, for long periods of time, the various artificial foods which, in many instances, they are incapable of digesting; and not only this, but no change is made in their diet for months, either in the quantity of the various ingredients which it contains, or in their quality. These children, therefore, suffer from the nutritive changes which come on as a result of a limited and fixed diet with no variation; whereas, the children of the poor, who often have too great a variation in their diet, rarely present scorbutic symptoms and often do manifest distinct rachitic symptoms.

Physicians, in the presence of obscure illness occurring in early childhood, should remember the possibility of either one of these affections being the underlying cause for the manifestations of disease, and thoroughly investigate the question of diet before administering remedies, such as the salicylates for rheumatism, iron for the blood, or bromides for nervous irritation.

Treatment.—The treatment of scurvy in adults consists in providing good and varied food, with plenty of oranges or lemons and green vegetables. Sunshine and fresh air are also essentials. Arsenic and iron may be given as hæmatics. If digestion is impaired it should be aided by hydrochloric acid and pepsin or lime-juice and pepsin. For the lesions in the mouth a chlorate of potash and myrrh mouth-wash (see Stomatitis) may be used.

If the disease occurs in a child it is to be treated by changing the food and in using raw milk instead of sterilized milk. Beef-juice squeezed from a half-cooked steak and orange-juice are also very useful.

OBESITY.

Definition.—The term obesity, or adiposity, is used to describe a state in which an individual suffers from an excessive deposit of fat in those parts of the body where a moderate amount of fat is found in health. In its advanced forms fat is also deposited in parts where it is never found in the normal state.

Etiology.—It must be clearly understood that the mere presence of an unusual amount of fat does not in any way indicate ill-health or that the functions of the body are perverted. In many instances a considerable degree of obesity exists, because it is the natural state of the individual. In others, however, the deposition of fat in excess is a manifestation of disease or at least of perverted function.

In the first class, of what may be called normally obese persons, the condition arises from inherited tendency. It is in this class that we find individuals who have never been heavy eaters and who for years have deprived themselves of foods of which they are fond, but still gain weight. In another class it develops from overeating and lack of exercise, and in the third class it is due to disorders of metabolism, whereby foodstuffs are not properly dealt with by the economy after they are ingested.

These three types are worth recalling, because when a patient seeks relief from obesity much depends upon the type to which he or she belongs, as to advice, prognosis, and treatment.

Symptoms.—It is not necessary to describe all the symptoms of obesity, for the manifest increase in the size of the patient determines the diagnosis of an excess of fat. There are, however, certain other symptoms which are of importance, not only because they are part of the symptom-complex of obesity, but also because their presence determines the degree to which the excess of fat is really annoying or harmful. A symptom usually complained of by the patient is *dyspnœa* on exertion, which arises from the fact that the heart and lungs are put under stress because great muscular activity is needed to move the heavy body. This dyspnœa is also due to the fact that the vascular network is greater in the obese than in those who are lean, and therefore the heart has to drive the blood through a greater number of bloodvessels. Thirdly, the heavy deposits of fat on the chest walls, in the omentum, in the mesentery, about the diaphragm, and around the heart interfere, mechanically, with the free action of the respiratory and cardiac muscles. In many cases of severe obesity the layers of fat are projected between the cardiac muscular fibres, and thus seriously impede its movements, forming the so-called "fatty heart of the obese," which is, of course, a very different state from the true fatty heart of myocardial degeneration. The *pulse* is often *small* and *rapid*. The arterial tension is also lower than normal, as a rule.

While many of these patients are mentally and physically slow and somewhat somnolent, others are active and restless and even unduly wakeful.

Next to the dyspnœa the chief complaint is of *constipation* or *indigestion* and *excessive sweating*. Some cases of obesity, however, have a digestive system all too good.

In some cases, too, there is an excess of urates in the urine, and these cause *vesical irritability*, chiefly because the larger surface of the body and the free perspiration cause a great loss of fluid, and this in time causes a scarcity of urinary flow with consequent concentration of the urinary solids.

Treatment.—The first point in treatment, as von Noorden has said, is to determine whether we shall diminish the fat already present or content ourselves with the prevention, if possible, of an increase in the obesity. As a rule, the patient is not content with a plan of treatment which does not actually diminish the fat, partly because he has delayed consulting a physician until the condition is far beyond what she desires. This is particularly the case with women who have arrived at middle life and who begin accumulating weight at the time of late childbearing, or in other cases immediately after marriage. These patients are often normally fat—that is to say, their condition is physiological—and they should be content in the majority of cases to try to prevent further obesity, rather than to remove fat already in existence. Such patients are often not unduly fat, and in their desire to maintain a "girlish figure" are willing to resort to almost any measure to become thin. Indeed, women in the fashionable walks of life, with little to think about, often make their lives miserable and destroy good health of mind and body by endeavoring, on the one hand, to get thin, or, on the other hand, to get stout.

In such cases the physician should advise against severe measures, point out that the plumpness is natural, and, if need be, assert that it is better to be in good health, and a little more weighty than the average woman, than to be in bad health and slender. I have seen several splendid specimens of healthy womanhood made physical wrecks by ill-advised efforts to get thin.

The great difficulty with all plans of treatment for the reduction of fat, in those women who desire to be slim for the sake of vanity, is that no plan can be so nicely adjusted as not to remove fat from where its presence is needful to good looks. With the decrease in the bulkiness of the hips and waist a hideous leanness of the chest and mammary glands ensues and leaves these parts covered with a skin thrown in wrinkles by disappearing fat, so that a well-preserved woman of middle age is soon converted into a hag. Further than this, pads of fat keep organs in place, and those who wilfully remove these pads may subsequently suffer from floating kidney, gastroptosis, uterine disorders, and constipation.

There is a sad lack of knowledge on our part as to the metabolism of obesity and nutrition in general, and the patient and physician must be careful how they attempt to disarrange processes so intricate and important.

On the other hand, it is often necessary to arrest a process which is manifestly excessive and in need of control.

In young persons whose nutritive processes are still in a formative stage and who are obese, we should not reduce weight already present, but simply try to prevent an abnormal increase. This holds true of those persons in later life who give a history of having always been fat. It is very unwise to ignore this rule if advanced years are already upon the patient, for under these conditions the effects of age and the efforts at reduction may produce disastrous nutritional changes.

The most favorable period of life for reduction in weight is from twenty-five to forty years of age.

Before ordering a diet and mode of life the patient should be subjected to a very careful physical examination; the urine should be repeatedly examined, and the state of the heart and vascular system carefully noted. If the urine contains albumin and sugar a reduction treatment is contraindicated, and if the heart is weak from myocardial degeneration and if the vessels are fibroid it is dangerous to institute a plan of this sort.

When it is determined that the patient is in normal health as to his vital organs the treatment for the prevention of increase is to be instituted, the patient being informed that a good result cannot be reached by a sudden and rapid process, and that patience and persistence are necessary for really valuable results.

The first factor is exercise taken to the degree of moderate fatigue. Many patients take it to excess, and then not only eat and drink heavily, but lie down and rest while the nervous system lazily permits vital oxidizing processes to go on too slowly, and so more weight is gained than lost.

In many cases no material reduction in fat can be attained unless the patient can be treated in a sanatorium, or at least in some place where an absolute diet can be maintained for a long period of time. It is not sufficient to order a reduced diet and but little to drink and much exercise. Such aids to reduction in weight will not in the ordinary case produce much improvement, because the patient is not willing to persist in the annoyance of such a strict diet for a sufficient length of time to establish a new nutritional abscissa. Unless the state of reduced weight is maintained for a long period the patient often gains more flesh on returning to the ordinary diet than if no attempt at reduction had been made.

The order for actual reduction of weight consists in cutting from the diet list all sugars and sweet articles, all fats and richly prepared foods, and in the prescribing of lean meat, and of vegetables which are bulky but contain little starch. Celery, lettuce, string-beans, spinach, cabbage, cauliflower, and limited amounts of tomatoes may be permitted; whereas potatoes, bread, peas, and beans are to be forbidden. All alcoholic drinks are to be avoided, because the alcohol has to be oxidized in the body and so prevents an active oxidation of the foodstuffs and tissues. Alcohol is also contraindicated, because it stimulates the digestive organs and so increases the desire for food.

If the avoidance of the fattening foods named above does not prevent an increase in weight, then a more rigid diet must be arranged. The patient is not to be given all the food he desires, but must suffer from privation and hunger. Instead of ordering an amount of food which will give the 2500 to 3000 calories necessary for comfortable existence, an amount calculated to allow about 2000 calories or less must suffice.

For breakfast the patient is allowed 3 ounces of lean meat, 1 ounce of bread with no butter, and a cup of tea or coffee with no milk and no sugar, the sweetening being done by the use of saccharin. At an early luncheon a single soft-boiled egg may be given with an ounce of bread. At dinner a cup of clear soup, such as consommé or Julienne (but no thickened soup or purée), may be allowed, followed by 2 ounces of fresh or salt fish, and this by 2 or 3 ounces of lean meat. At this meal small quantities of the various green vegetables already named may be taken. The dessert should consist of some fresh fruit, such as an apple, an orange, a grape fruit, or a pear. In the middle of the afternoon a glass of milk or a cup of tea with a thick water cracker may be given, and at supper-time 3 ounces of lean meat, some lettuce with oil and vinegar, celery, 2 ounces of toasted bread or Zweibach or crackers may be given. At bedtime a biscuit and a glass of milk may be allowed. While excessive drinking of water is unwise, the patient should not be deprived of water to such extent that he suffers from thirst or has not sufficient liquid in his body to carry out to the full every physiological function. Although overfilling the tissues with water will make the patient appear fat, a certain amount of fluid is necessary to healthy life.

In the way of drugs there is but one remedy which exercises a great influence in reducing flesh, and that is the thyroid gland. This substance does not reduce the weight of all cases of obesity, and often fails unless it is given in doses which are so large as to cause distinct cardiac feebleness. The doses usually given vary from 2 to 6 grains of the extract of the gland once, twice, or thrice a day. It is important to give a large amount of nitrogenous food at the same time, for the thyroid causes a loss of nitrogenous tissue in the body as well as a loss of fat. Small doses of strychnine and of digitalis are also useful to protect the heart from depression. The patient should be warned against severe exercise while using this drug, and often will do best if confined to bed and given massage and electricity.

ADIPOSIS DOLOROSA.

Under the name of *adiposis dolorosa* my colleague, F. X. Dercum, first described in 1889 a condition in which masses of fat are deposited in different parts of the body, chiefly on the chest, arms, buttocks, and legs. These formations are usually symmetrical, and, as the name implies, are accompanied by neuralgic pains which vary from slight dartings to severe suffering. The disease is of unknown origin, and affects women in or past middle life, as a rule. The deposits on the extremities are usually firm and even brawny, but they may be so soft as to be pultaceous in character. So far the best results in its treatment have been obtained by the use of thyroid gland to the point of tolerance. The condition is quite uncommon, but a considerable number of cases have been reported.

ACROMEGALY.

Definition.—Acromegaly is a slowly developing chronic disease of nutrition characterized by an overgrowth of the extremities and head, and, to a less degree, of the trunk, with associated curvature of the dorsal and cervical spine. It is sometimes called "Marie's disease," because Marie first described it in 1886.

Etiology.—Many theories have been advanced by various clinicians with the object of determining the causation of this extraordinary malady, but none of them are adequate. The one which seems most probable from our present knowledge is that the condition arises from disease of the pituitary body and that the enlargement of the thyroid gland, which is often present, is an effort of this gland to supplant its function. Acromegaly rarely appears before the thirtieth year.

Symptoms.—The appearance of a patient suffering from this disease is so peculiar and striking that there is no difficulty in diagnosis, if the malady is well developed. The *massive* and *gigantic* appearance of the *head,* of the *features* of the *face,* and, on closer inspection, the *enlargement* of the *hands,* and the increase in the length of the long bones, combined with the *kyphosis of the spine,* make the clinical picture complete. The upper part of the forehead appears low because of the abnormally prominent superciliary ridges, and this effect is exaggerated by the *projection of the lower jaw.* As a consequence, the shape of the face is elliptical. The skin of the face is thick and sallow and greasy in appearance, and lies upon the forehead in heavy transverse creases. The cheeks appear sunken, chiefly because of the great overgrowth of the malar bones. The nose is not only greatly enlarged, but often increases in size more rapidly than the other features, so that it seems out of proportion with the rest of the head. The nostrils are heavy, thick, and immovable. Not rarely the superior maxillary bone fails to develop as rapidly as nearby tissues, and as a consequence the upper jaw may seem sunken, an effect increased by the enormous nose above and the overgrown lower jaw below it. This effect is also increased by the great enlargement and *thickening* of the *lower lip.* An examination of the mouth will reveal the fact that the *tongue* and *uvula* are *broader* and *thicker* than normal. The *thorax* on inspection will be seen to be *greatly increased* in its *anteroposterior diameter,* which is in excess, as compared to its lateral diameter. The ribs are enlarged and the *clavicles thickened,* but the abdomen often appears sunken because of the projection forward of the lower part of the thorax. The muscles may, in the early stages, seem increased in size and in power, but the dominant tendency is to *muscular atrophy.* No changes of importance take place in the internal viscera. *Blindness,* partial or complete, may develop, due to optic neuritis. Rarely *nystagmus* and *squint* have appeared. (See Figs. 108 and 109.)

The subjective symptoms—that is, those complained of by the patient—consist in *headache, dimness of vision,* and *pains* in the *joints.* There is usually *slowness of thought* and perhaps *actual drowsiness.*

FIG. 108

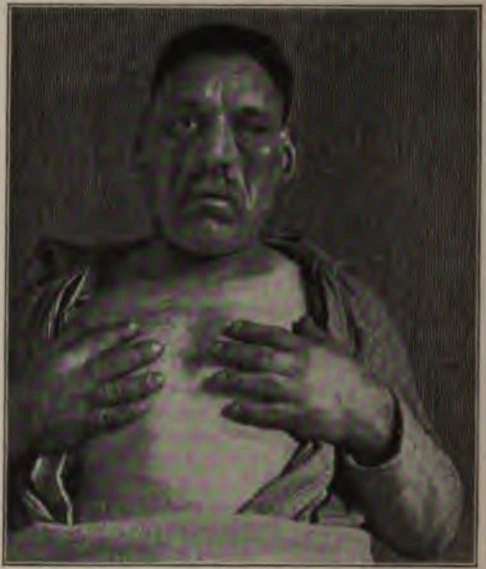
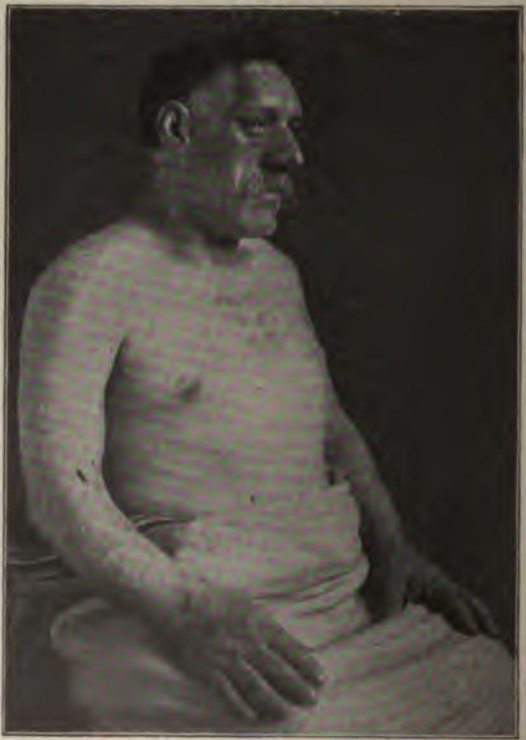


FIG. 109



Acromegaly, showing the large hands, nose, and superciliary ridges.

Diagnosis.—Acromegaly must be differentiated from gigantism, leontiasis ossea, myxœdema, arthritis deformans, osteitis deformans, and pulmonary hypertrophic osteoarthropathy. Perhaps the most frequent error in diagnosis is that of confusing myxœdema with acromegaly, but in myxœdema there is never any actual increase in the size of the bones. The face in myxœdema is round and full instead of elliptical, and the ends of the fingers are swollen and thickened instead of the whole hand being manifestly enlarged, as in the disease under consideration. Again, in myxœdema the skin is pale, puffy, and waxen in appearance, devoid of hair and also of wrinkles, whereas in acromegaly the skin upon the face is wrinkled and there is no marked falling of the hair.

Gigantism is separated from acromegaly by the fact that there is a symmetrical overgrowth all over the body, whereas, as has already been pointed out, the enlargements in acromegaly affects chiefly the extremities and the tissues of the face. Further than this, in gigantism the ends of the bones are not enlarged to such an extent as to be out of proportion to the shaft, and in acromegaly this disproportion is quite constant.

Leontiasis ossea is characterized by the development of bony tumors or osteophytes on the face and cranium which produce great deformity, but there is no marked enlargement of any one feature nor of the extremities.

Osteitis deformans is differentiated from acromegaly by the facts that the long bones are chiefly affected, are apt to be curved, and so produce great deformity. But there is no marked enlargement and the deformity is very apt to be asymmetrical. In osteitis deformans the facial bones are rarely affected, but the cranial bones are involved in the pathological process; whereas, in acromegaly it is the facial bones which are affected, the other cranial bones being but slightly diseased. Finally, and perhaps most important, the face of a case of osteitis deformans is broadened in its upper portion and narrowed in its lower portion, giving it a triangular appearance; whereas, in acromegaly the lower part of the face is broad, and therefore the general effect is elliptical.

In pulmonary hypertrophic osteoarthropathy there is enlargement of the hands and feet, but no enlargement of the face, and there is always found marked chronic pulmonary lesions, such as bronchiectasis, empyema, or other serious thoracic disease. A close examination of the hands and feet will show that the enlargement is confined chiefly to the joints, and that the whole hand is not thickened and increased in size as in acromegaly.

Treatment.—No plan of treatment is of any value.

OSTEITIS DEFORMANS.

Osteitis deformans is sometimes called "Paget's disease." It is characterized by enlargement and softening of the shafts of the long bones, by pain and deformity. The bones of the face are not affected, but the bones of the rest of the head are often involved. A careful examination of the long bones in a case of this kind reveals the presence of a rarefying osteitis associated with the development of new but imperfect lamellæ in the bones. The face has a curious triangular appearance because of the broadening of the upper portion and the narrowness of the chin. By the yielding of the bones under pressure, not only the tibiæ but the femurs also undergo great deformity, so that an extreme degree of bow-legs develops. Occasionally, the bending of these bones is forward or backward. There is also some spinal curvature. Osteitis deformans rarely develops before the fiftieth year. No treatment which has yet been discovered is of any value.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY.

This is a condition first recognized by Bamberger, but the name was given by Marie. It does not affect the bones of the head or of the face, nor the long bones of the arms or leg. In every instance it develops in association with chronic pulmonary disease, such as chronic bronchitis, bronchiectasis, fibroid lung, and chronic empyema.

The symptoms consist in the enlargement of the hands and feet, particularly about the small joints. The growth of the nails is often influenced so that they are thickened and incurvated.

LEONTIASIS OSSEA.

Leontiasis ossea is a disease in which there is a development of multiple osteophytes or symmetrical enlargement in the bones of the cranium, and sometimes in those of the face. It is a very rare affection.

SCLERODERMA.

Scleroderma is a chronic disease characterized by localized or general stiffening or rigidity of the skin, which is usually pigmented, and which seems to be bound over the tissues beneath it in much the same manner that a leather binding is sometimes placed over a wooden or metal object. In some instances the sclerodermatous process is sharply circumscribed. In other cases it shades off into the surrounding tissues, and may have a slightly reddened edge.

The first symptom usually noticed by the patient is stiffness of the part affected, which gradually increases until movement may become almost impossible. The skin undergoes atrophic changes, and becomes silvery and shiny in appearance, with a certain amount of yellowish or light-brown discoloration. When the disease affects the skin of the extremities, it may cause much interference with the movement of the large joints, and be followed by atrophy of the muscles underlying the area involved. The lesions most commonly take place in the skin of the neck, in the neighborhood of the shoulders, and over the back and chest. It not infrequently attacks the skin of the face. The general health is not seriously impaired. There may be some local discomfort, with a sense of formication, or tingling. The skin is exceedingly dry, and rarely sweats.

Treatment.—In the way of treatment thyroid extract has been highly recommended by certain clinicians. Locally the parts should be treated by massage and the local application of oils, which should be of a sedative character. Occasionally, however, if the process is exceedingly chronic, it may be advisable to apply turpentine diluted with six times its amount of sweet oil. Even with the best of treatment the prognosis is anything but favorable as to cure, although the spread of the malady may be delayed.

AINHUM.

Ainhum is a peculiar trophoneurotic disease, commonly affecting the feet of negroes and other dark-skinned races. It is widely distributed in Africa, particularly along the west coast, and it occurs in India, and Brazil. It is a rare affection among negroes in the United States. The disease usually begins in the little toe of one foot or both feet, as a narrow fissure or groove, on the plantar surface at the junction of the toe and foot. The groove gradually surrounds the toe and slowly deepens until eventually it is amputated. Microscopic examination shows that the constricting band consists of dense fibrous tissue. As it tightens, the toe becomes very much enlarged, and disorganized before it finally separates. As a rule, the disease is confined to the one toe, although other toes may be successively attacked and the disease may even appear in the leg.

The cause of this condition has not been determined. It has been ascribed to traumatism, such as frequent cuts from blades of grass. By some writers it has even been regarded as an expression of a very much attenuated form of leprosy. It is evidently a trophic disturbance.

Treatment.—The treatment is surgical, and consists in free division of the constricting bands in recent cases and amputation in advanced cases.

INTOXICATIONS.

ALCOHOLISM.

Definition.—By alcoholism is meant a condition in which the patient suffers from the effects of alcohol when taken in sufficient quantities to act as a poison.

Etiology.—An idea exists among the laity that chronic alcoholism is a manifestation of an inherited tendency in many instances, and this is sometimes offered as an excuse by a patient for his unlimited libations. There is no such disease as alcoholism, nor does an alcoholic have any justification in this excuse. The tendency to consume alcohol is not an inheritance. The inheritance is a lack of self-control, a cowardly inability to meet the hard sides of life, and a willingness to escape, if only for a time, by drowning sensation in the stupor of a narcotic. In many cases, therefore, we may not only have to combat a habit, but a state of degeneracy which permits a habit to exist.

Symptoms.—Alcoholism may be divided for readiness of consideration into the acute and chronic form.

Acute Alcoholism.—The symptoms of the acute form are familiar to everyone who sees life in the town or city, and consist in *disorderly conduct* due to removal of the inhibitory functions of the brain, so that every *silly thought* or *foolish idea* is carried out in action. Later, as the drug affects the muscle sense, and consequently *disorders co-ordination*, the individual *staggers* and perhaps falls, and finally, if the quantity of the drug is adequate, passes into a *deep sleep*, or *coma*, from which he wakes more or less confused, with depression of the nervous system and a disordered digestive tract. In cases where the dose has been very large, death may be caused by depression of all the vital functions, of which the one most involved is probably that of bodily heat, the death being in part due to cold. In the majority of cases, however, in which death follows acute alcoholism, it is due not directly to the depressing effects of the alcohol, but to the fact that the lowering of temperature and the disorder of vital function in the various organs permits infection by the pneumococcus to take place so that pneumonia causes death, or some complication such as acute hephritis is developed.

The symptoms of profound acute alcoholism are *pallor of the face, dulness* of the *eyes, widely dilated pupils, profound unconsciousness, stertorous breathing,* and a *temperature* several degrees below normal. Occasionally *convulsive* attacks may develop.

Although the symptoms of acute alcoholism are so familiar, there is no state so often confused with conditions of disease or with the results of injury. This is due to the fact that the symptoms of acute alcoholism are much like those of cerebral congestion, apoplexy, uræmia, or hemorrhage from a meningeal artery, or fracture of the skull. It is also due to the fact that alcohol often causes all these states directly or indirectly, and as there is a history of alcoholism or an odor of alcohol on the breath, it is natural to make a diagnosis of alcoholism without recognizing that another condition is present. Again, it not rarely happens that an alcoholic takes a poisonous dose of opium, and so suffers and dies from the effect of this drug. The raised temperature in apoplexy is in contrast to the lowered temperature of alcoholism, as is also the full-bounding pulse as compared to the rapid-running pulse of alcoholic poisoning. Again, apoplexy is characterized by hemiplegia and facial palsy. Opium poisoning is characterized by pinpoint pupils, slow breathing, and a warm skin, as opposed to the normal or relaxed pupils, the cool, moist skin, and the normal or rapid breathing of alcoholism.

Treatment.—The treatment of acute alcoholism consists in the administration of an emetic to empty the stomach of any alcohol still unabsorbed. Apomorphine is probably the best drug for this purpose, as it can be given hypodermically, acts promptly, is sedative in its influence, and is safe in a moderate emetic dose of $\frac{1}{8}$ grain. In other cases, or after the emetic has acted, an active cathartic, such as 30 grains of compound jalap powder or 15 grains of compound extract of colocynth, may be used to unload the bowels and the portal system, and decrease cerebral congestion. If circulatory feebleness is present, the aromatic spirit of ammonia in drachm doses, diluted with water, may be given. In other cases the physician must give full doses ($\frac{1}{20}$ grain) of strychnine by the mouth, or by the hypodermic needle if depression is marked, and hot bottles must be applied to maintain body heat. Strong black coffee by the mouth or by the rectum may be given if active stimulation seems needful. The effects manifested on the next day are to be removed by the use of calomel, followed by a saline purge and the administration of elixir of celery and guarana, or, if the patient is very nervous, by the use of guarana and bromide of sodium, 5 grains of the extract of the former and 30 grains of the latter at a dose.

Subacute and Chronic Alcoholism.—Chronic alcoholism is divisible into three classes. In one the patient suffers from a prolonged alcoholic debauch lasting over days, or even weeks, during which time he is never completely sober. In the other type he is never drunk, but always under the influence of the drug to an extent which eventually produces a train of symptoms even more grave than those which follow a debauch. Those who have made a special study of alcoholism also recognize that there is a certain class of persons, of the first division just named, who take no alcohol for a comparatively long period, varying from weeks to months, and then go on a terrific debauch, the so-called "periodical drunkard." It is the individual who takes alcohol up to the stage of intoxication for several days consecutively, and who has often used alcohol in large quantities for weeks before the acute exacerbation, who most commonly develops delirium tremens;

while the constant, moderate "soaker" is more prone to hepatic cirrhosis and affections of the peripheral nerves. Delirium tremens is very prone to develop in persons who are the subjects of subacute or chronic alcoholism if, perchance, they suffer from a severe injury, surgical operation, or great shock. Not rarely the onset of an acute illness may precipitate an attack.

Morbid Anatomy.—The morbid changes produced by the continuous use of alcohol in excess are chiefly found in the organs by which the drug gains access to and egress from the body; that is, the stomach, the liver, and the kidneys. Next to the effect of the drug upon these organs it expends its deleterious influences upon the circulatory system. The passage of alcohol directly to the liver from the stomach through the portal vessels causes congestion, irritation, and finally atrophic cirrhosis of this organ. (See Hepatic Cirrhosis.) By reason of the direct effect of the drug upon the stomach, and the indirect effect produced by the impairment of its blood supply, which arises from the hepatic changes, chronic gastric catarrh develops. The changes found in the kidneys in very chronic alcoholism consist in a condition practically identical with that found in contracted kidney, and with this state an arteriocapillary fibrosis develops, just as it does in cases of cardiovascular change arising from other causes. The most common change in the kidneys, however, consists in a hypostatic congestion, or stasis, which causes them to be swollen, cyanotic, and to be functionally inactive. Not rarely these patients develop acute tuberculosis, because of their lowered vital resistance. An alcoholic multiple neuritis may develop, and atrophy of the optic nerve may occur.

Symptoms.—The symptoms of delirium tremens are great nervous restlessness and *apprehension* with *anxiety*, and finally *delusions of persecution* and *terror*. The delusions are largely those connected with vision, and all sorts of *hideous objects* are described as crawling about the patient. Because of these delusions the *patient* is often *violent* and difficult to control, but is rarely offensive unless he believes that the attendant is in league with the "objects of evil" about him. The *pulse* is usually rapid and feeble, the *skin relaxed*, and the *tongue* exceedingly *foul*. The *bowels* are *constipated* and the *urine scanty*. *Hypostatic congestion* of the *lungs* and *congestion of the kidneys* are very commonly developed, and these states often contribute to the death of the patient.

It is not to be forgotten that acute croupous pneumonia at the apex not rarely is associated with an acute delirium not unlike that of delirium tremens.

In that form of chronic alcoholism in which the patient is never drunk, but always has alcohol in his body, the chief symptoms are irritability of temper, gradual mental deterioration, localized sensory and motor palsies, and finally dementia.

Treatment.—The treatment of this state consists in the use of an active cathartic, as already advised for acute alcoholism, and the use of full doses of morphine hypodermically, if the kidneys are not diseased, to produce nervous rest if the patient is becoming exhausted by his lack of sleep or struggling. Care must be taken that more than a few doses are not given, for such a patient may become a morphine habitué very quickly. Strychnine and atropine are to be used hypodermically if any signs of

pulmonary congestion arise, and they must be given
should be applied to the back of the chest, and Hoffm
useful as a rapidly acting diffusible stimulant. Ever
taken to disperse and prevent the congestion, which if
degree, means the death of the patient.

The question as to the medicinal use of alcohol in these
In those who are not accustomed to its constant use, and w
intoxicated for but a few days, it is not necessary to giv
the patient has been in the habit of taking alcohol in exc
prior to his acute alcoholic outbreak, whiskey must be us
cases if signs of great nervousness develop. Aside fro
renal complications the most frequent one, and a most
of nervous tension in which the symptoms are those of m
with stiffness of the limbs and neck. This stage of toxi
is preceded by muttering delirium, with periods of wak
hallucinations may make the patient difficult of control.
contracted and the pulse rapid and feeble. The temp
high as 103° or even 104°, and not rarely hypostatic conge
can be found if the bases are examined. Marked hy
skin usually exists. Patients with these manifestations
they may recover after a prolonged illness lasting se
experience is in accord with that of Dana, that if there
cervical muscles the patient usually dies. This conditio
true meningitis, but to a toxæmia with serous effusion i
Dana and others have given the name "wet brain" to th

The diet, if food can be given to the patient, should
stimulating liquid nourishment, such as highly seasoned
tonized milk, to which capsicum and salt have been adde
digestion to activity. The various highly seasoned broths

The *treatment* of the alcoholic who continually takes th
day out presents grave difficulties. Those who have been
drug every day for many years are rarely willing to put up
fort which follows abstinence, and after a few days almost
to the use of alcohol. The only treatment which is of
cases is to send the patient to some isolated region w
removed from the grog-shop to be able to obtain alcoh
for it arises, and to take care that he does not provide hi
with alcohol to be used during the trip. Usually, if the
several weeks or months of hunting in isolated regions, an
of someone who does not drink alcohol and has considera
will be the best means of cure.

For those who take alcohol more or less constantly
intoxication, either this measure can be employed or th
placed in a private room in a hospital, where he is unde
of the nurses and physicians attached to the institution
can then be immediately stricken off the list of permissi
his condition is one of feebleness, it can be gradually dimi
the end of a week he is getting none of it. In the great maj

it is utterly futile to attempt home treatment of these cases. Even if the family can prevent the man from getting alcohol, home life lacks the discipline which is necessary for the control of the patient, not only in the sense of preventing him from procuring alcohol, but in the sense of making a powerful mental impression.

In those cases in which removal of alcohol causes nervous excitation and evidences of threatened delirium tremens, it is occasionally permissible to administer full doses of chloral and the bromides to produce nervous quiet at night. Small doses of these drugs practically have no influence whatever, and if the heart is at all feeble full doses of chloral are dangerous. Morphine possesses the disadvantage that the alcoholic is very prone to develop the morphine habit in addition to his alcoholism. This drug is, however, exceedingly valuable if used on those occasions when the violence of the patient's nervous symptoms demand sedation. It should not be given day after day, but only occasionally, when insomnia is so pressing that the consequent exhaustion demands relief.

Within the last few years very strong claims have been made for the use of hyoscine for the purpose of relieving the nervous irritation and craving for alcohol. The drug must be given in sufficiently large doses, hypodermically, to place the patient deeply under its influence. If necessary as much as $\frac{1}{100}$ of a grain every two hours may be given hypodermically until the patient sleeps or is resting quietly. These doses, of course, produce the full physiological action of hyoscine and often cause great dryness of the mouth and talkative delirium. They may be continued for several days and then gradually remitted until the patient is no longer taking either alcohol or hyoscine.

If the circulation fails in these cases strychnine and digitalis may be administered. For the purpose of combating signs of acute collapse Hoffmann's anodyne and strychnine are valuable, as is also the aromatic spirit of ammonia. Another drug which has been highly praised in this condition is apomorphine given in the dose of $\frac{1}{30}$ of a grain hypodermically as a nervous sedative, the emetic effect of the drug not being desired. In some instances larger doses have to be given and may be used without producing emesis.

It must be constantly borne in mind that the most important portion of the treatment consists in the isolation of the patient, and in a complete control of his methods of life for the period covering several weeks. Drugs are of little value except to support him through the period when his system lacks his customary doses of alcohol. If the patient is unwilling or unable to resort to this form of treatment, the employment of drugs is usually worse than useless.

Careful attention to the digestive system is needful in all these cases. The liver should be unloaded by blue mass, followed by a saline purge, every few days, and bitter tonics, such as gentian with bicarbonate of sodium, are advantageous.

MORPHINISM.

Chronic morphinism, or the morphine habit, is usual
result of the employment of this drug for the purpose of
Sometimes the insomnia is due to neurasthenia, but more fre
is one who primarily suffers from sleeplessness due to pain
producing the pain is continued over any considerable
patient finally becomes so dependent upon the use of mor
sedative, that he cannot sleep without it, and so even alt
longer continues he resorts to the drug for the nervous
obtained only under its influence. Not infrequently thes
the use of the morphine long after the physician intends
stopped, and so develop the habit of taking the drug with
of the physician who has first prescribed it. Because o
this occurrence he should only prescribe that quantity
is absolutely essential for the relief of pain upon a p
and if he writes a formal prescription ordering the drug f
this prescription should contain the words "Do not re
patient will not be able to continue taking the drug a
believes that he has stopped it.

In a certain number of cases of morphine habit, the empl
rests upon the fact that the patient is a degenerate wit
mental and nervous vigor to meet the vicissitudes of life
he is one who, in the presence of any condition which pr
turbation or distress, at once resorts to some sedative t
system, instead of dominating it by his will power and con
sion with a knowledge that any yielding either to that d
desire for a drug is certain in the end to wreck his m
condition. This is an important factor to be taken into c
ever a morphine habitué is to be treated. This condition
or it may be acquired. Not rarely, when it is acquire
physical condition of the patient has been impaired
business worry, illness, or other cause. When the morp
for any length of time this very fact tends to increase
stamina on the part of the patient.

Symptoms.—One of the most noteworthy symptoms
great *irritability* of the *nervous system* so that slight ca
outbreaks of rage, or, on the other hand, a *lacrymose state*
some instances, where the drug has been used for a long
only a loss in mental power, but the patient develops *m*
sions closely resembling those seen in an ordinary case o
the mental degradation is so complete that intellectual pr
impaired, the patient develops a *slyness* quite different
frank methods of life when in health. In association wit
is always developed a skill at *prevarication* or *lying* whic
able. Persons who previously have been regarded as emi
the most skilful falsehoods, and in such a way that the

of their truth. As a rule, these falsehoods are never so clever as when they are intended to result in the obtaining of the drug which is desired, the patient resorting to every possible means, honest and dishonest, in order that he may obtain the nervous quiet which his system craves.

Treatment.—It may be asserted with truth that it is useless to attempt to treat a patient who is suffering from the morphine habit, with the idea of curing him of his taste for the drug, unless he or she is willing to enter a private room at a hospital and be placed under the constant supervision of a night and day nurse. This isolation is necessary not only because it is a form of discipline which is advantageous for the mental condition of the patient, but it is the only way in which the physician can be assured that the patient is not surreptitiously continuing his daily dose of the narcotic. It must be remembered that the most pious individuals, when they become addicted to this drug, develop an extraordinary ability to tell lies which are so like the truth that they can deceive the most cautious. In one breath, the patient, with tears running down his face, will beseech the physician to cure him of the habit which is destroying his happiness, and, at the next moment, he will use every form of deceit and cleverness to obtain the drug which he craves. Even when the patient is under the observation of trained nurses night and day, any sudden improvement in his condition after withdrawal of the morphine, or failure to develop symptoms produced by its withdrawal, should make the physician believe that in some unknown way the drug has been obtained. In some instances the patient enters the hospital with the morphine carefully sewed in the hem of the night-dress; in others, a friend or servant is bribed to bring the drug each day in some article of food. Nothing but ceaseless watchfulness can possibly prevent these patients from obtaining morphine. This being so, it can readily be understood that home treatment can rarely succeed.

Having obtained special control of the patient, the method of treating him should consist in one of two plans which have found general acceptance. The first of these is the gradual diminution of the morphine so that at the end of three or four days, or a week, none of the drug is permitted. If this method is carried out the patient usually develops after a few days, or sooner, great restlessness and irritability, not infrequently active purging, and profound mental and physical depression. Cramps in the extremities also add to the suffering. Under these circumstances it is necessary to support the patient by the use of hot, stimulating foods, such as broths highly seasoned with pepper and salt, the use of digitalis and strychnine if the circulation fails, and the employment of hyoscyamus or hyoscine to diminish irritability of the nervous system. The employment of alcohol, coca wine, or similar stimulants for the purpose of aiding the patient at this time is unwise, because he is prone to develop the alcohol or coca habit. If the diarrhœa is so violent as to require control, aromatic sulphuric acid with a vegetable astringent, like the fluid extract of hæmatoxylon, may be used. Hot compresses may be applied about the painful limbs. Great mental excitement may be relieved by chloral, but the danger of producing the chloral habit is not to be forgotten. In place of chloral, sulphonal or trional may be used. Occasionally nerve quiet can be produced by wrapping the patient

in a hot, wet blanket, care being taken that the hot pack
so long as to produce cardiac depression.

A second method of treatment is one which has been
in the last few years, and for which we are chiefly ind
physician, Dr. Lott. This consists in putting the patie
have him under absolute control, and in the administrati
hyoscine hypodermically, giving him as much as $\frac{1}{100}$ of
hours, if need be, until a condition of nervous quiet is
writer's experience these large doses fail to produce sleep,
a condition in which the patient lies awake but stupefied
bles continuously. Curiously enough the mouth does n
as one would expect from the administration of such a
these large doses. Should the circulation seem at all feebl
also be given. The idea in employing hyoscine is to
which is necessary to keep the patient under control, and
ing. These doses may be continued for a number of d
which time they are gradually diminished and the patie
return to his normal condition as the effects of the hy
By this means the acute mental and physical suffering cau
withdrawal of morphine is avoided, and in some inst
actually seems to be cured of his malady, although, of cou
danger in every case of his speedily returning to its use,
nervousness or mental stress is experienced. So common i
to go back to the employment of this drug habit that ma
ence have gone so far as to assert that no case of the mor
permanently cured. This view is, however, undoubtedl
writer has seen a number of cases in which permanent c
place.

ARSENICAL POISONING.

Arsenical poisoning occurs in two forms, the acute and
after the first stage of acute poisoning, if the patient surviv
a second subacute stage due to the effects of the retaine
poisoning usually follows the ingestion of "Rough on R
or Scheele's green. The symptoms are those of seve
with vomiting and purging, followed by death in collaps
is the hydrated sesquioxide of iron with magnesia. Whe
vives the acute stage he suffers from secondary lesions in
intestines, kidneys and liver. Widespread fatty degene
and peripheral neuritis may be present.[1]

The causes of chronic arsenical poisoning are almost
are those of lead poisoning. It may find its way into
the lungs from the air of a room the walls of which are c
heavily laden with arsenical dyes; it may enter in beer m
prepared by the use of iron pyrites contaminated with

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[1] For this train of symptoms see the author's Text-book of Therapeuti

recent celebrated epidemic in England; or it may be given in moderate poisonous dose for a long time with homicidal intent, as in a case recently tried in the Philadelphia courts.

Not very rarely a mild form of chronic arsenical poisoning is met with in cases to which a physician has found it necessary to give large doses of arsenic for long periods, as in chorea, in leukæmia, and Hodgkin's disease.

Symptoms.—Chronic arsenical poisoning manifests itself chiefly in the form of a widespread *peripheral neuritis*, with the development of a *secondary degeneration* of the epithelium of the *kidneys*. The chief symptoms are *tingling* and *pains* in the limbs, followed, after a time, by *paralysis* which affects the distal portions of the body much more than it does the nerves and muscles of the thighs or arms. *Atrophy* of the *muscles* supplied by the paralyzed nerves soon takes place. Other trophic changes also develop, such as *herpetic eruptions* resembling those of *herpes zoster* or *pemphigus*, and *glossiness* of the skin sometimes supervenes. At times curious *deposits* of *pigment* take place in the skin. As in lead poisoning, the nerve supply to the *extensor muscles suffers chiefly*, but in addition the small flexor muscles are also affected much more commonly than they are in neuritis due to alcohol or lead. The lower limbs are affected as much as the upper limbs, whereas in lead poisoning it is the upper extremities which suffer most. Again, arsenical neuritis affects the sensory and motor fibres, and for this reason pain as well as anæsthesia is often met with. The pulse is quickened and the mind confused in some cases. Because of the involvement of the sensory and motor fibres of the peripheral nerves the patient may present symptoms of tabes dorsalis (*arsenical pseudotabes*). The Argyll-Robertson pupil is a useful differential factor, for if it is present the cause of the disordered gait is probably due to true locomotor ataxia.

Prognosis.—The prognosis in such cases depends upon the severity of paralysis and the state of the kidneys. If the latter organs are affected the outlook is more grave than if they are intact. Even when the symptoms of neuritis are severe, remarkable recovery may ensue if the patient is removed from the exposure to the drug.

Treatment.—The treatment consists in the removal from exposure, the use of the iodide of potassium to aid in the elimination of the poison, the administration of strychnine in full doses if the nerves are not irritable, and in the application of massage and electricity to improve the nutrition of the affected parts. Iron may be used to combat the anæmia.

LEAD POISONING OR PLUMBISM.

Acute lead poisoning is not of frequent occurrence. Its consideration is distinctly toxicological in character, and for this reason it is not discussed in these pages.

Chronic lead poisoning, on the other hand, is of exceedingly common occurrence, not only in those who are exposed to the poison by reason of their occupation, but in persons who have suffered no such exposure but have absorbed the lead from sources in which its presence would not be suspected. Further than this, lead poisoning in its chronic form may produce the most varied symptoms, which are oftentimes so unusual that no thought of lead as a cause is entertained. In speaking of the nervous manifestations which are often present, a well-known teacher was wont to say: "When you cannot explain a curious train of nervous symptoms, always suspect syphilis, hysteria, or lead as the cause."

Etiology.—It is the insoluble rather than the soluble salts of lead which usually cause chronic lead poisoning. The most frequent sufferers are workers in manufactories where paint is made, and house painters who are continually engaged in the handling of lead paint. In rarer instances the patient is poisoned by the use of water which in passing through new lead pipes dissolves some of the lead; or lead is present in a hair-dye or cosmetics and is absorbed by the skin; or, again, it has occurred that a miller has filled holes in his grindstones with lead, which has then been ground with the flour and eaten in bread. In one instance, in Pennsylvania, a peddler sold a large number of crocks to farmers' wives. In these was placed apple butter, and as the acid in the fruit eroded the lead glazing which lined the jars, a widespread epidemic of chronic lead poisoning developed. Perhaps the most notorious illustration of how lead may cause poisoning in unsuspecting persons is the celebrated "chrome-yellow cases" in Philadelphia, in which a wholesale druggist sold chrome yellow to a number of confectioners, who saved the cost of eggs by coloring their cakes with this substance. As a result a large number of men, women, and children died, and a much larger number suffered from chronic poisoning for months before the source of the trouble was discovered. An extraordinary cause, however, is the habit of chewing silk thread weighted with lead. At least two cases of chronic poisoning from this cause have been met with in seamstresses.

The fact, therefore, that no history of exposure to lead is to be found in a given case does not negative the diagnosis of lead poisoning.

Prevention.—Chronic lead poisoning is to be prevented in workers in lead by the exercise of the greatest possible cleanliness as to their hands, which should be thoroughly washed before food is touched, as otherwise lead may be taken in small amounts and finally cause poisoning. If the workman is employed in grinding lead, he must wear a mask to prevent the poison from being inhaled in dust. The use of vessels, the glazing of which contains lead, for holding food should be avoided. Small amounts of dilute sulphuric acid to form insoluble sulphates in the mouth and stomach may be resorted to, and purgation every few days by sulphate of magnesium is advantageous.

Pathology and Morbid Anatomy.—There is no other poison from the mineral kingdom which taken into the body produces such widespread changes in different organs as does lead in the chronic form of poisoning. Even alcohol, that most ubiquitous poison, does not cause such a multitude of changes. The nervous system is the portion of the body which bears the brunt of the attack, and it is the peripheral nerves that suffer most. In them the lead produces a toxic neuritis. In advanced cases there is segmentation of the myelin and breaking up of the axis cylinder, with a proliferation of the nuclei in the sheath of Schwann. The early changes in the nerves affect chiefly the medullary sheath, which is affected in patches at irregular intervals—the so-called periaxial neuritis of Gombault. Although the lesions are more severe as the distal end of the nerves is approached, Déjerine has found them even in the anterior roots. Conspicuous changes in the spinal cord are almost never seen, but Oppenheim states that he has found alterations in the anterior cornua, and Gowers asserts that in some cases the cells in these cornua are degenerated. No constant lesions are found in the brain, even in those cases in which severe cerebral symptoms are present, except those dependent upon vascular lesions which are part of the general vascular disease produced by the poison.

It is to be especially noted that the sensory fibres of the nerves are not affected, and that the musculospiral is the nerve chiefly involved in those cases which have peripheral neuritis. As a result of the neuritis, produced by the lead, atrophy of a severe character may develop in the muscles supplied by the affected nerves. Fatty degeneration of the muscles does not ensue. The second portion of the body to feel the effect of the lead is the kidneys, which are not rarely the seat of chronic interstitial nephritis, and with this renal lesion a process of arteriofibrosis develops, which associated conditions often cause the death of the patient.

Sailer has recently shown that, in some cases at least, there is an absence of hydrochloric acid in the gastric juice.

Symptoms.—From what has been said of the changes in various organs caused by lead, it is evident that the symptoms may be very varied. *Paralysis* of the *extensor muscles* of the *forearm*, causing *wrist-drop*, is the most constant symptom. This paralysis is nearly always bilateral, but occasionally but one arm is affected. The supinator longus muscle and the short extensor of the thumb, however, usually escape, which is curious in view of the fact that the supinator longus muscle receives its nerve supply from the musculospiral nerve. In atypical cases Oppenheim states that the supinator longus, the biceps, and even the deltoid are involved.

In the legs palsy is far less frequent than in the forearms, and the muscles involved are the peroneal group, but the tibialis anticus is not affected.

Although motor paralysis is present sensory disturbances are rare.

Palsy of the ocular muscles producing *squint* may be due to lead, as may also *optic neuritis*. In very rare cases of severe plumbism cerebral symptoms develop, consisting in *epileptiform convulsions*, or *coma*. This state is called "encephalopathia saturnina."

Tremor of the forearms is sometimes present in lead poisoning.

Chronic lead poisoning greatly aids in producing *gouty lesions*, probably by forming a urate of lead in the tissues about the joints. Others believe that the lead decreases the alkalinity of the blood and so permits the precipitation of urates to occur.

There still remain to be considered several symptoms of chronic lead poisoning which are so constant in their appearance and so characteristic that they are most valuable aids in diagnosis. The first of these is the *blue line* in the edges of the gums next the teeth, formed by the deposit of sulphide of lead in the capillaries of the part. This sign is often absent in those who are cleanly in the care of the mouth. The second is the characteristic *pain in the belly,* which is exceedingly severe in the region of the umbilicus, and is described as a pain due to twisting the bowels around a stick. This is called "painters' colic," or "colica pictonum." The latter term is given to this state because it was frequently met with in Picton at one time. A third symptom of chronic lead poisoning is *anæmia,* which is in part due to the direct effect of the lead and in part to the renal changes induced by this agent. Microscopic examination of the blood will often reveal a *granular degeneration of the erythrocytes.*

Diagnosis.—In a case in which the blue line on the gum is present the diagnosis is easy. When wrist-drop is present it must be separated from that due to pressure, as by resting the head on the arm when sleeping or by the pressure of a crutch. As a rule, pressure palsy is unilateral and lead palsy bilateral, but this is not so invariably, and the history of the patient may be necessary to decide the diagnosis. When the palsy is distributed in various parts, particularly if it affects the legs, it must be separated from acute poliomyelitis Lead poisoning is rare in children and acute poliomyelitis is common. In adults chronic lead poisoning is more frequent than is acute poliomyelitis. Poliomyelitis in its acute form has a history of sudden onset with fever, and the onset of lead palsy is rarely so abrupt and is usually not febrile. The history of exposure to lead will aid in deciding the diagnosis. In chronic poliomyelitis the only way to determine the question is by the history and the frequent examination of the patient's urine for lead. Often lead will not be found in the urine unless iodide of potassium is given to set it free from the tissues where it has been deposited.

Saturnine epilepsy must be separated from true epilepsy by the history of the patient and by the association of other signs of plumbism. It must also be separated from uræmic convulsions, if possible, by the urinary examinations, but this may be impossible because the lead may at once cause encephalopathia saturnina and uræmia through its effects on the cerebral vessels and the kidneys.

Prognosis.—The prognosis as to the duration of life in chronic lead poisoning is good unless cerebral symptoms are present, or renal changes are well marked. The prognosis as to the paralysis depends largely upon the general nutrition of the patient and the stage to which the neuritis has advanced. If the muscles involved have lost all reaction to electrical stimulation, the prognosis must be bad as compared with that in a case in which the palsy has lasted for but a short time. Even when the reactions of degeneration are present the outlook is not hopeless, because if the patient is no longer exposed to the poison recovery sometimes ensues, particularly in young persons.

Treatment.—The treatment of chronic lead poisoning consists in removing the patient from continued exposure to the poison. If he is an artisan he must cease working in lead. If he has been poisoned by the metal through some accident, the source must be discovered and he must no longer be exposed to it.

The second duty of the physician is to eliminate the lead already in the body as rapidly as possiple. For this purpose the iodide of potassium should be given in full doses, with the object of forming double soluble iodides with the lead. Not only have we every reason to believe, from a chemical standpoint, that this medicinal treatment is advantageous, but it is a well-known clinical fact that chemical examination of the urine in a case of lead poisoning will fail repeatedly to show lead, and will at once indicate its presence after iodide of potassium is administered, proving that by this means lead is carried to the kidneys and speedily passed out of the body. It must also be remembered that the liver eliminates lead freely in the bile.

The third indication is to improve the patient's general health not only by the use of such tonics as iron and strychnine, but also by ordering an out-door existence, with as much sunshine as it is possible for the patient to find in the twenty-four hours.

The paralysis of chronic lead poisoning is to be treated by the administration of full doses of strychnine and the simultaneous use of large doses of iodide of potassium. The paralysis of the extensor muscles of the arms and legs is to be treated not only by the use of strychnine, but by the employment of the slowly and rapidly interrupted faradic current. In those cases in which cerebral symptoms develop, the patient should receive full doses of iodide of potassium, with the object of getting rid of the lead as rapidly as possible. If the symptoms are acute, and if a convulsion is already present, the patient should receive a hot pack in order that the sedative effect of this therapeutic measure may be exercised upon the nervous system, in order that the blood may be drawn away from the congested brain, and with the hope that by increasing the action of the skin the convulsions may be relieved of some of the work which they would otherwise be forced to perform. In other respects the convulsions should be treated, as are all other convulsions, by the use of nitrite of amyl inhalations, and by the employment of full doses of chloral and the bromides to quiet the brain and spinal cord.

Painters' colic with its attendant constipation is not to be treated by the use of purgatives, but by the use of morphine given hypodermically. This drug, which so often causes constipation in the ordinary patient, often produces active purgation in these cases, by quieting the intestinal irritation and spasm and simultaneously relieving the pain.

FOOD POISONING.

Bromatotoxismus.—Symptoms of poisoning produced by the ingestion of food which is impure by reason of faulty preparation, or the changes due to decomposition, are occasionally met with. It is rather remarkable, considering the long period of time during which many foods are kept after they are prepared for the table, that more cases of poisoning do not ensue. Much of the information given in this article is obtained from the excellent summary of this subject which can be found in Vaughan and Novy's *Cellular Toxins.*

Poisoning may be produced by the use of grains which have become infected by poisonous fungi. Animals may eat substances which may render their milk or flesh or both poisonous. The flesh of certain animals also becomes poisonous at certain stages of their life history. Foods may also become infected by the discharges of human beings; the flesh of animals may suffer from some specific disease which may be transmitted to man, and milk may carry the disease of an animal to man or may be infected by the discharges of man, and so convey specific germs to other individuals. Food may also contain micro-organisms which in their process of development produce poisonous symptoms in man.

The term *sitotoxismus* is applied to poisoning by vegetable foods which are infected by moulds or bacteria.

The most familiar form of poisoning by grains, or vegetable food, is *Ergotism* due to the eating of rye flour made from rye which has been infected by the fungus *Claviceps purpurea.* This ergot is, of course, largely employed in medicine. Several poisons are found in ergot, such as ergotinic acid, sphacelinic acid, and cornutin. The first of these, however, seems to be poisonous only when it is injected hypodermically. Sphacelinic acid, on the other hand, is supposed to be responsible for the gangrene and cachexia which sometimes develop in persons who have eaten infected rye. On the other hand, cornutin seems to be the poison which affects the nervous system and produces spasms and convulsions.

Mytilotoxismus.—Under the name of mytilotoxismus is described the symptoms of poisoning which are produced by eating poisonous mussels. These symptoms consist in some cases in violent gastrointestinal irritation with purging, but in the majority of instances the manifestations of the poisoning are nervous in character. A rash resembling urticaria and finally becoming vesicular may develop over the body, and the eyelids may be so swollen as to prevent vision by extravasation of the serum into their tissues. There is often difficulty in breathing, apparently due to intense hyperæmia of the bronchial mucous membrane. Convulsions and coma may develop and death may be due to this cause.

Treatment.—The treatment of mytilotoxismus consists in the use of an active saline cathartic to sweep the poisonous material from the alimentary canal, and in the use of ether as a diffusible stimulant.

Ichthyotoxismus.—When fish produces poisonous symptoms the term ichthyotoxismus is used to describe the condition. As is well known, certain fish are unfit to eat, and other fish become poisonous during the season at which they are spawning. In still other instances fish suffer from bacterial infections which render their flesh unsuitable as food. The ingestion of poisonous fish so seldom occurs, at least in this country, that the symptoms produced need not be described.

Poisoning from the flesh of fish which has undergone decomposition is often very violent in its manifestations. The most common symptoms are dilatation of the pupils, nausea, vomiting, and severe abdominal pain, followed by the development of a scarlatinal rash all over the body. In such cases a purge to sweep out the offending materials and also stimulants are needed.

Kreotoxismus.—The word *kreotoxismus* is applied to poisoning resulting from the ingestion of meat unfit for food, because of the presence of bacterial or animal poisons. Perhaps the most frequent instance of this is in so-called sausage poisoning. Sausages are often made from what may be called the refuse following the butchering of animals ordinarily employed as food, and the treatment of this material is such that early decomposition changes may readily set in. In most instances the process of cooking destroys the poisons, but when cooking is not resorted to the symptoms which are induced are exceedingly severe, and death may ensue. There is, in many cases of sausage poisoning, difficulty in breathing and swallowing, violent vomiting, severe abdominal pain, hoarseness, dimness of vision, and delirium. In other cases the mind remains clear. From some of these forms of food ptomaines have been isolated. In other instances certain bacteria have been found which have been considered responsible, either directly or indirectly, for the symptoms. Meat-pie poisoning and poisoning by mince-meat are essentially similar to sausage poisoning.

Tyrotoxismus and Galactotoxismus.—Under the name *galactotoxismus* is described the poisoning which results from the ingestion of impure milk. When poisoning follows the use of bad cheese it is called *tyrotoxismus*. This term is also applied to the poisoning produced by impure ice-cream. The symptoms are sometimes exceedingly severe, and consist in evidences of gastroenteritis followed by collapse. To a substance which Vaughan states he is able to isolate from cheese and ice-cream he gave the name of "tyrotoxicon."

PELLAGRA (MÄIDISMUS).

Definition.—Pellagra is a chronic intoxication due to eating fermented maize. It is characterized by gastric and intestinal symptoms, by marked motor, sensory, and psychical disturbances, and by a constant skin eruption.

History.—Pellagra has been known for about two hundred years. From that time until now it has been seen in various countries of Europe, particularly in Spain, Portugal, and Central Italy. In the Province of Venice 343 cases occurred in the year 1901, and in the marshes of Umbria and Tuscany the cases are very numerous. In 1881 the number of cases in Italy was officially estimated at 104,607, or 0.36 per cent. of the entire population. The disease occurs in Hungary, in certain departments of Southern France, in Egypt and in Algiers. On this continent it is found in Mexico (Yucatan and Campeachy).

Etiology.—Pellagra is primarily due to eating unsound maize or its products. Thus, whiskey prepared from such maize may give rise to pellagra just as corn-mush, corn-pap (polenta), or corn-bread may convey it. The cause of the fermentation of the maize is harvesting in unfavorable years, before the grain has thoroughly ripened, and storing in damp cellars or granaries. With respect to the specific changes in the grain, there is some conflict of opinion as to their exact nature. Lombroso isolated a watery extract, an alcoholic extract, and an oil with which he could produce the symptoms of pellagra in man and animals. Ceni and Besta have recently extracted, with ether, from the spores of *Aspergillus fumigatus* and *Aspergillus flavescens* a body which produced all the symptoms of pellagra. They have further shown that these organisms are constantly present in smutted maize.

Both sexes are equally attacked. All ages are subject to the disease although the majority of cases occur in later adult life. Bad food, privation, and malaria predispose to the development of pellagra. Alcoholism is a very prominent factor in the etiology of the disease. Some writers, indeed, consider it to be the principal cause.

Pathology.—The post-mortem changes observed in pellagra are extreme emaciation, with loss of the subcutaneous fat and extensive wasting of the muscles. The heart, liver, spleen, and kidneys are atrophied. Marked pigmentation is seen in the liver, spleen, heart, and spinal ganglia. In the central nervous system various changes are met with, principally chronic inflammation of the meninges with exudation, and occasionally hemorrhages. The spinal cord shows marked sclerosis of the posterior and posterolateral columns. The peripheral nerves are degenerated.

Symptoms.—In pellagra there is first of all an ill-defined prodromal stage. The patient feels *out-of-sorts* and shows marked disinclination to work. He has *dyspeptic symptoms* and complains of *constant pain* in the *head* and *spine.* At this early stage *insomnia* is common and there may even be some degree of *mental disturbance.* These symptoms continue throughout the winter, until, with the advent of spring, the first marked symptoms of pellagra present themselves in an *erythematous rash* and marked *gastrointestinal disturbances.* The rash appears on the exposed surfaces of the body, particularly on the backs of the hands, the extensor surfaces of the forearms, and on the face and neck. It persists several weeks, when desquamation occurs, leaving a thickened patch of scurfy skin. The gastrointestinal symptoms consist in *severe dyspepsia;* there are *flatulence, eructations,* and *anorexia. Diarrhœa* is commonly present, sometimes approaching dysentery in severity. These symptoms persist throughout the spring. With the advent of summer the patient becomes very much better and continues in a fair state of health until, with the next succeeding spring, a recrudescence takes place. Thus, from year to year a cachectic state is developed, with marked nervous phenomena.

The motor symptoms are *spastic paralysis of the legs, wasting,* and *contracture.* The *superficial reflexes* are *normal,* the *deep reflexes increase.* The sensory symptoms are *severe pain* in the head and spine, with *intolerable burning and itching,* referred to various skin areas. *Girdle pains* are common. *Disorders of vision* and *taste* occur. Psychical symptoms are extremely common, and a large percentage of cases end in *permanent insanity.* In all Italy, out of every 100 insane patients, 10 are sufferers from pellagra. In Venice the proportion is 35 out of every 100. The common type of insanity is *melancholia,* with delusions of persecution and impulses to self-destruction, particularly by drowning. *Mania* occurs in a small proportion of cases and dementia is a common ending of all of them.

In long-standing cases pronounced *cachexia* develops, with *profound anæmia* and *marked emaciation.* Alpago-Novello calls attention to the evidences of premature senility in these patients. They become prematurely bald, prematurely gray, a marked arcus senilis may be present, and early atheroma is frequently observed. The disease is very chronic and may last from ten to fifteen years. Cases that have had more than three or four attacks, in succeeding springs, are regarded as hopeless.

Treatment.—Treatment consists, first of all, in the interdiction of unsound maize as an article of food, and the prohibition of alcohol as a beverage. With respect to medicinal treatment, Lombroso recommends large doses of arsenic. Wurzel confirms the value of arsenic and also advises the use of quinine and strychnine in large doses. For the nervous symptoms massage, electricity, and salt-water baths are of service. The patches of erythema should be kept moist and supple with oily ointments to which cocaine, carbolic acid, salicylic acid, or chloral may be added to control the itching.

The *prophylaxis* of the disease includes education of the people in the infected area of the danger of fermented maize as an article of food. Maize should only be harvested when ripe and should only be stored in dry, well-ventilated granaries. When maize is a staple article of import it should be rigorously inspected at the first port of entry and all spoiled grain rejected.

LATHYRISM (CHICKPEA DISEASE; LUPINOSIS).

Definition.—Lathyrism is a chronic intoxication characterized by spastic paralysis of the lower limbs without disturbance of sensation. It is caused by the use of various species of lathyrus as food. That the vetches, or chickpeas, were capable of producing a disease of this type was known in the time of Hippocrates.

History.—The disease was exceedingly common in Italy and France during the seventeenth and eighteenth centuries. It was first accurately studied by Irvin in 1859. The present geographical distribution is France, Italy, Algiers, and British India.

Etiology.—Lathyrism is due to the use of different varieties of lathyrus as foods; chiefly *Lathyrus sativus*, *Lathyrus cicera*, and *Lathyrus clymenum* (the common vetch, or chickpea). In time of famine and want the seeds of these legumes are ground and mixed with flour and other meal and used in bread-making. Observation has shown that they must be eaten for some length of time, several weeks or a month, before symptoms begin. The poison lies in the peas themselves, and mustiness or fermentation is not a factor as in pellagra. It is stated that "Teora" (*Lathyrus sativus*) in India only causes the disease when baked; when boiled it is said to be innocuous. As immediate exciting causes for the outbreak of the disease, exposure to cold, rain, and damp weather are recognized. Consequently men, being much more exposed to climatic changes in their daily avocations, are more frequently attacked than women. Cattle eating the peas also develop the disease.

Symptoms.—There may be prodromal symptoms, such as *fever, dyspepsia,* and *diarrhœa.* Usually the disease begins suddenly, after exposure or a wetting. There is *pain* in the loins, legs, and knees, with weakness and tremors. From this point on there is a gradual and continuous development of a *spastic paralysis* of both legs with marked spasms, particuarly of the adductor muscles of the thighs. The *reflexes* are *exaggerated.* Sensation is not impaired. The bladder and rectum are not involved, as a rule, excepting in severe cases, when the paralysis may include the sphincters. The upper limbs are not involved. The course of the disease is exceedingly chronic and the paralysis is usually permanent.

Death from lathyrism itself is very rare. In one patient who died of an intercurrent malarial infection Grandjean found a softened area in the cord extending 6 cm. above the lumbar enlargement. The clinical symptoms point to a degeneration of the lateral columns.

Treatment.—Treatment consists in the withdrawal of lathyrus from the patient's food, and the education of the masses to the danger that lies in the use of these products. Scheube reports good results from sharp counter-irritation over the lumbar vertebra.

ATRIPLICISM.

Atriplicism ("intoxication par l'arroche") is an intoxication due to eating the coast orach, and is characterized by marked local disturbances of sensibility, by local œdema and trophic disorders. This disease was first reported by Matignon in 1898, from North China. He ascribes it to eating *Atriplex augustissima* and *Atriplex serrata* of the order *Chenopodiaceæ*, both common weeds in China. They are eaten baked or fried in dough, or raw as a salad.

Within ten to twenty hours after eating the orach there is *tingling and cold* in the *thumbs, fingers,* and *back of the hand,* followed by *itching* and *œdema,* which spreads over the back of the hand and forearm and ends sharply at the elbow. Later, *swelling* and *itching of the face* occur. The parts are cold, the nose and finger-tips cyanotic. The œdema persists from two to ten days. After it subsides the *skin is exfoliated.* In severe cases *large blebs* may form which subsequently ulcerate. Matignon reports one case in which gangrene of the fingers occurred. The disease superficially resembles erythromelalgia.

Treatment.—Treatment consists in free purgation at the beginning of the disease and the application of sedative lotions locally.

LACQUER POISONING.

This is a troublesome poisoning of the skin observed in China and Japan among workers in lacquer. Lacquer is derived from the balsamic exudate from the lacquer tree (*Rhus vernicifĕra*). The poisoning is limited to the skin and is entirely analogous to that produced by the varieties of rhus, so common in America, excepting that it is much more severe. The infection takes place not only through contact with the raw lacquer, but Scheube states that it may be conveyed from the vapors arising during the evaporation of lacquer, and that susceptible individuals may even acquire it by visiting lacquer warehouses where there are newly lacquered articles. The attack begins a few hours after exposure, with intense itching of the skin of the face and limbs. The skin becomes œdematous; numerous papules appear, which later become vesicular and are filled with a yellow seropus. The vesicles may coalesce and form large blebs. The disease is limited to the arms, legs, face, and genitals. When the face is severely affected, the adjacent mucous membrane shares in the disturbance, and there may be a sharp febrile reaction.

Treatment.—Treatment consists in soothing local applications. Lead-water with or without opium and lime-water are principally used.

The gingko tree (*Gingko biloba*) causes a dermatitis exactly like lacquer poisoning excepting that it is not so severe. The question of *gingko poisoning* is of some little interest in the United States at present. The tree is a native of China, but has recently been introduced in America as an ornamental foliage plant.

DISEASES DUE TO ANIMAL PARASITES.

MALARIAL INFECTION.

Definition.—By malarial infection we refer to a condition produced by the entrance into, and development in, the blood of specific micro-organisms known as the *Plasmodium malariæ*, the hæmatozoon of malarial fever, or, more correctly speaking, the *Hæmamœba malariæ*.

The infection is manifested by four different types: First, the so-called intermittent type, in which the patient has recurring attacks which are characterized by a chill, a fever, and a sweat. These recurrences commonly take place daily, on alternate days, or on every third day, and are called quotidian, tertian, or quartan. Second, a type in which there is present continued fever with remissions in its course, the so-called remittent malarial fever. Third, a type in which the infection is of a malignant or pernicious form with profound toxæmia. Fourth, that form in which more or less subacute, or chronic, and profound cachexia is present, associated with marked anæmia and enlargement of the spleen, and often of the liver. The first is due to the tertian or quartan parasite, the second and third to the æstivo-autumnal parasite, while the fourth form may be due to any one of the three parasites.

History.—Malarial fever was recognized as a distinct disease as long ago as five hundred years before Christ, and Hippocrates divided it into the quotidian, tertian, and quartan types we recognize to-day. Empedocles (500 B.C.) recognized the relationship of the disease to stagnant water and stopped an epidemic by draining stagnant pools; but it was not until 1880 that Laveran, a French army surgeon, first recognized the specific organism, and in 1886 Marchiafava and Celli described it more fully. In the same year Golgi showed that the malarial attack occurred simultaneously with the sporulation of the parasite, but not until 1898 did Manson and Ross, of England, and Grassi and Bignami, of Italy, prove that the infection is spread from man to man by a certain species of mosquito known as the *Anopheles*. In the United States excellent work has been done by a number of investigators, of whom the most noteworthy are Osler, Thayer, and Hewetson in Baltimore, James in New York, Craig of the U. S. Army, and Dock in Texas.

Distribution.—Malarial infection is more widely diffused throughout the tropical and temperate zones than any other disease. It is, as a rule, prevalent and severe in direct proportion to the proximity to the equator and is rare in far northern latitudes. Certain parts of the world, which at one time suffered severely from the disease, are now free from it. Forty or fifty years ago, for example, the valleys of the Delaware and Schuylkill Rivers near Philadelphia suffered greatly, whereas at present cases of the disease are rarely met with in these localities. On the other hand, it is very prevalent on the shores of the Chesapeake Bay, which is not more than one hundred miles away. At present the disease appears in its mild forms in France, Germany, and England, and in the Middle Atlantic and Central United States, and in its severe forms in the Southern States, particularly in certain lower portions of the Mississippi Valley. The virulent forms are chiefly met with in Africa, in certain parts of India, and in the tropics, as in the West Indies and in the Philippines, and in the tropical parts of South America. On the Pacific coast of the United States the disease is rare. So far as season is concerned, it may be said that the greater proportion of cases occur in July, August, September, October, and November in semi-tropical or temperate regions.

The frequency of malarial fever varies greatly and depends entirely upon the prevalence of the *Anopheles*, the sources for its infection, and the climate which permits of the growth of the mosquito. In the United States the greatest prevalence of this disease is in the States bordering the Gulf of Mexico and that tier lying immediately north of them. The death rate from malaria in this area is about 30 per 1000 deaths from known causes.

Etiology. THE MOSQUITO.—There is but one direct etiological factor in the dissemination of malarial fever in man, namely, that form of mosquito known as *Anopheles*. Many species of the genus *Anopheles* have been described, but only three have so far been found to be present as malaria-bearing hosts in the United States, namely, the *Anopheles quadrimaculatus*, which is the most common, the *Anopheles punctipennis*, and the *Anopheles crucians*. In Europe the infection is always borne by the *Anopheles claviger*, sometimes called *Anopheles maculipennis*. Fortunately, the anopheles is not universally distributed, the culex being the genus which is most commonly met with, at least in the temperate zones, and this mosquito seems to be incapable of carrying the infection. The anopheles can be readily differentiated from the culex by the fact that when it rests upon a plane surface its body is held at right angles, or at an angle of forty-five degrees, whereas the body of the culex lies parallel to the plane. Again, the wings of the anopheles show very distinct mottling, as its names punctipennis or quadrimaculatus indicate. Most of the culex species lay their eggs in rafts or bottle-shaped masses, which remain intact until the larvæ are discharged. The eggs of the anopheles are laid in groups that are readily broken up and scattered. Any stagnant or semi-stagnant accumulation of water is a suitable breeding-ground.

The indirect factors in the causation of malarial infection are, therefore, stagnant or semi-stagnant water in which this mosquito can breed, and the presence of a source from which it can obtain the parasite so that it can transmit it to a healthy individual; for even if the *Anopheles* be present, it cannot inoculate a human being with malarial fever unless it has first bitten a person whose blood contains the hæmamœba. No more interesting experiments proving these facts can be adduced than those made by Patrick Manson on his son, who had never had malarial infection. Bignami and Bastianelli sent Manson in England relays of mosquitoes which in Italy had been fed upon the blood of patients suffering from pure benign tertian malaria. These mosquitoes were allowed to bite the younger Manson, and as a result he developed the same form of malarial fever as that suffered by the Italian patients, and the same parasite was found in his blood.

THE HÆMAMŒBA MALARIÆ IN MAN.—The parasite itself passes through two cycles of existence, namely, one which is carried out in the body of man, and another in the body of the mosquito. It appears in three distinct forms, namely, as the tertian parasite, the quartan parasite, and the æstivo-autumnal parasite. Each of these live in the red cells of the blood and to some extent exist in the plasma as well.

Marchiafava, Celli, Bignami, and Grassi, of Italy, have endeavored to show that several species of the æstivo-autumnal parasite exist, but their views have not been generally accepted, and recently they have admitted that there are not sufficient grounds for advocating this proposition.

In certain cases the patient suffers from a mixed infection in which the tertian and æstivo-autumnal parasite are both present at the same time.

The young *tertian parasite* is a small colorless and hyaline body which occupies a small space in the corpuscle. When in a state of quiescence it is round, but if the specimen under the microscope is fresh and the temperature suitable it manifests active amœboid movements. As the parasite grows, reddish-brown granules develop in its interior. These pigment granules move rapidly, and are often seen in the amœboid projections of the parasite, so that it may appear that several parasites are in one corpuscle. As the growth continues the infected red cell becomes more and more pallid and swells up or expands, the amœboid movements of the contained parasite diminish in activity, and the pigment granules arrange themselves about the periphery of the parasite. At this time the corpuscle is nothing more than a shell of its former self. Later the pigment granules accumulate near the centre of the body, and as they do so the process of segmentation begins; radial lines divide the parasite into twelve to twenty segments arranged around the central mass of pigment. Each segment has a nucleus, and as soon as the process of segmentation is completed these segments break out of the corpuscular shell and float freely in the blood plasma, where they speedily attack and enter fresh red cells. The evolution and segmentation require about forty-eight hours, and the chill and other acute manifestations of illness in the patient develop at the time of segmentation. In some instances the parasite becomes unusually large, the pigment bodies become stationary without aggregation in the centre, vacuoles develop in it, and the parasite seems to die. (See Plate V.)

The tertian parasite is the one which produces that form of intermittent fever in which the paroxysm occurs every second day. If the attack occurs daily, it is due to a double infection, in which one set of parasites sporulates on one day and the other set on the next day.

The following distinctions serve to separate the tertian from the æstivo-autumnal parasite: The nuclear body and chromatin mass of the young tertian parasite are achromatic to methylene blue, whereas the nucleus of the æstivo-autumnal parasite is densely stained by this agent (Ewing). The tertian ring is coarse and granular, whereas the æstivo-autumnal ring is a perfect circle and more delicate. The tertian ring is usually pigmented before the chromatin becomes subdivided, while in the æstivo-autumnal parasite the chromatin is subdivided before pigmentation appears. There are, however, exceptions to this rule. Lastly, the infected red blood corpuscle is usually distended or swollen as soon as it is attacked by the tertian parasite, whereas it is shrunken in appearance when the æstivo-autumnal parasite enters it.

The *quartan parasite*—that is, the organism that causes an attack every third day—resembles the tertian organism just described, but differs from it in the following respects: In the early stages it occurs as a hyaline body which is smaller than the tertian parasite. It speedily develops a sharper outline, it is more refractive, and the amœboid movements are slower. The pigment granules are larger and darker, less active, and lie near the edge of the parasite. Again, it is noteworthy that the red cell does not swell as do those containing the tertian parasite, but grows smaller, darker, more refractive and metallic looking. The quartan parasite reaches its growth in from sixty-four to seventy-two hours, and then appears as occupying nearly the entire red blood cell, or it seems to float free in the blood serum. As the time for the paroxysm approaches the pigment granules at the periphery flow toward the centre in radial lines, so that it becomes arranged in stellate form and the protoplasm divides into from six to twelve pear-shaped segments, each of which has a refractive centre. These segments escape and infect new cells. Some of the parasites do not, however, go on to this development, but fail to sporulate and become sexual bodies or gametocytes.

The third form of parasite, the *æstivo-autumnal form,* is smaller than the tertian and quartan organism, and presents a ringed appearance. It contains much less pigment and, moreover, it soon causes the corpuscle into which it enters to become shrivelled and brassy looking. After a time, possibly a week, the parasite increases in size, becomes refractive, crescentic, or round, or ovoid in form, and, in the centre, masses of dark pigment accumulate. It is these latter bodies which are indicative of infection by the æstivo-autumnal type, and it is to be remembered that, as a rule, they are not constantly present in the peripheral circulation but only in the blood of such internal organs as the liver, spleen, and in the bone-marrow. Because of their small size, slow development, and the difficulty of obtaining blood from deeply situated organs, they are less readily discovered than the other two types. It appears established that the crescentic and ovoid bodies do not undergo segmentation or sporulation, but correspond to the sexual bodies described above, the gametocytes. These crescentic and ovoid bodies do not continue their development in the human being, sporulation being the human cycle. The fertilization of the female by the male body occurs in the extracorporeal or intermediate cycle. (See Plate VI.)

The three forms of malarial parasite as they appear in man have now been described. The mosquito cycle of its existence is as follows:

DESCRIPTION OF PLATES V. AND VI.[1]

The drawings were made with great care and skill by Mr. Max Broedel, with the assistance of the camera lucida, from specimens of fresh blood. A Winkel microscope, objective, 1-14 (oil-immersion), ocular, 4, was used.

Figs. 4, 13, 23, 24, and 42 of Plate V. were drawn from fresh blood, without the camera lucida.

PLATE V.

THE PARASITE OF TERTIAN FEVER.

1.—Normal red corpuscle.

2, 3, 4.—Young hyaline forms. In 4 a corpuscle contains three distinct parasites.

5, 21.—Beginning of pigmentation. The parasite was observed to form a true ring by the confluence of two pseudopodia. During observation the body burst from the corpuscle, which became decolorized and disappeared from view. The parasite became, almost immediately, deformed and motionless, as shown in Fig. 21.

6, 7, 8.—Partly developed pigmented forms.

9.—Full-grown body.

10–14.—Segmenting bodies.

15.—Degenerative form simulating a segmenting body.

16, 17.—Precocious segmentation.

18, 19, 20.—Large swollen and fragmenting extracellular bodies.

22.—Flagellate body.

23, 24.—Degenerative forms showing vacuolation.

THE PARASITE OF QUARTAN FEVER.[2]

25.—Normal red corpuscle.

26.—Young hyaline form.

27–34.—Gradual development of the intracorpuscular bodies.

35.—Full-grown body. The substance of the red corpuscle is not visible in the fresh specimen.

36–39.—Segmenting bodies.

40.—Large swollen extracellular form.

41.—Flagellate body.

42.—Degenerative form showing vacuolation.

PLATE VI.

THE PARASITE OF ÆSTIVO-AUTUMNAL FEVER (*Hæmatozoön falciparum*).

1, 2.—Small refractive ring-like bodies.

3–6.—Larger disk-like and amœboid forms.

7.—Ring-like body with a few pigment granules in a brassy, shrunken corpuscle.

8, 9, 10, 12.—Similar pigmented bodies.

11.—Amœboid body with pigment.

13.—Body with a central clump of pigment in a corpuscle showing a retraction of the hæmoglobin-containing substance about the parasite.

14–20.—Bodies with central pigment clumps or blocks. Presegmenting forms.

21–24.—Larger bodies with central pigment blocks. Presegmenting bodies. Seen in the peripheral circulation during a severe paroxysm.

25–28.—Segmenting bodies from the spleen. Figs. 25–27 represent one body where the entire process of segmentation was observed. The segments, eighteen in number, were accurately counted before separation, as in Fig. 27. The sudden separation of the segments, occurring as though some retaining membrane were ruptured, was observed.

29–37.—Crescents and ovoid bodies. Figs. 34 and 35 represent one body which was seen to extrude slowly, and later to withdraw, two rounded protrusions.

38, 39.—Round bodies.

40.—Pseudogemmation, fragmentation.

41.—Vacuolation of a crescent.

42–44.—Flagellation. The figures represent one organism. The blood was taken from the ear at 4.15 P.M.; at 4.17 the body was as represented in Fig. 42. At 4.27 the flagella appeared; at 4.33 two of the flagella had already broken away from the mother body.

45–49.—Phagocytosis. Traced with the camera lucida.

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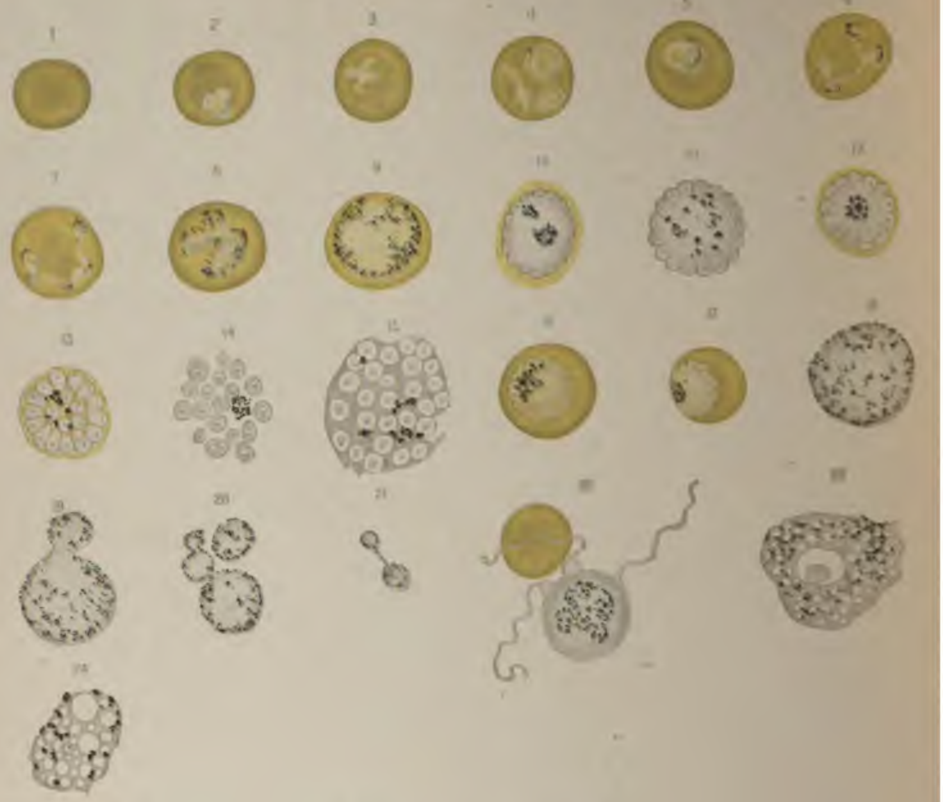
[1] These plates are taken by permission from Thayer and Hewetson's classical report in the *Johns Hopkins Hospital Reports*, vol. v., 1895. Four figures—viz., Figs. 21, 22, 23, and 24—have been added to Plate VI., and are also from the drawings of Mr. Max Broedel.

[2] The color of the pigment in these figures of the quartan parasite has too much of a reddish tint.

PLATE V

The Parasite of Tertian Fever



The Parasite of Quartan Fever

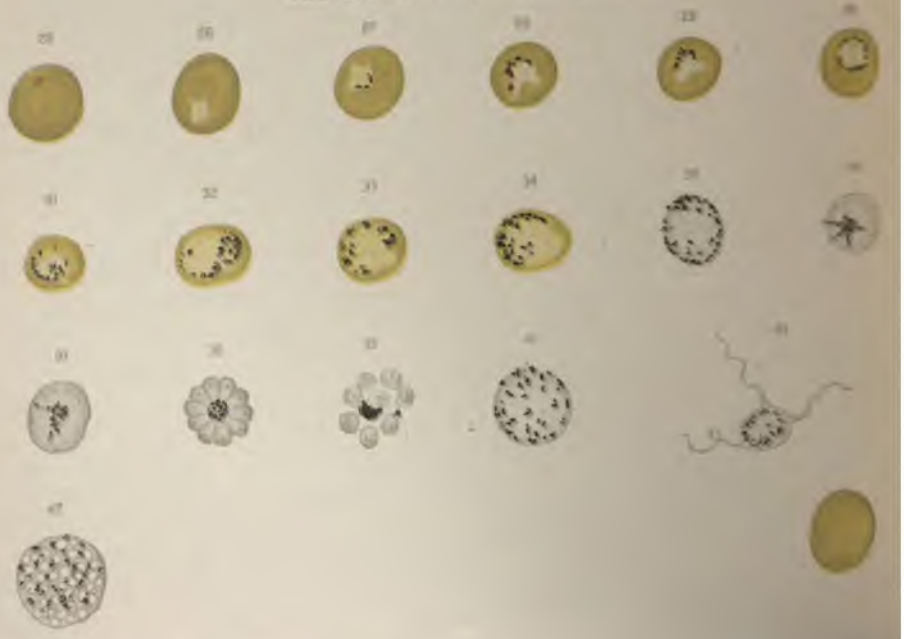
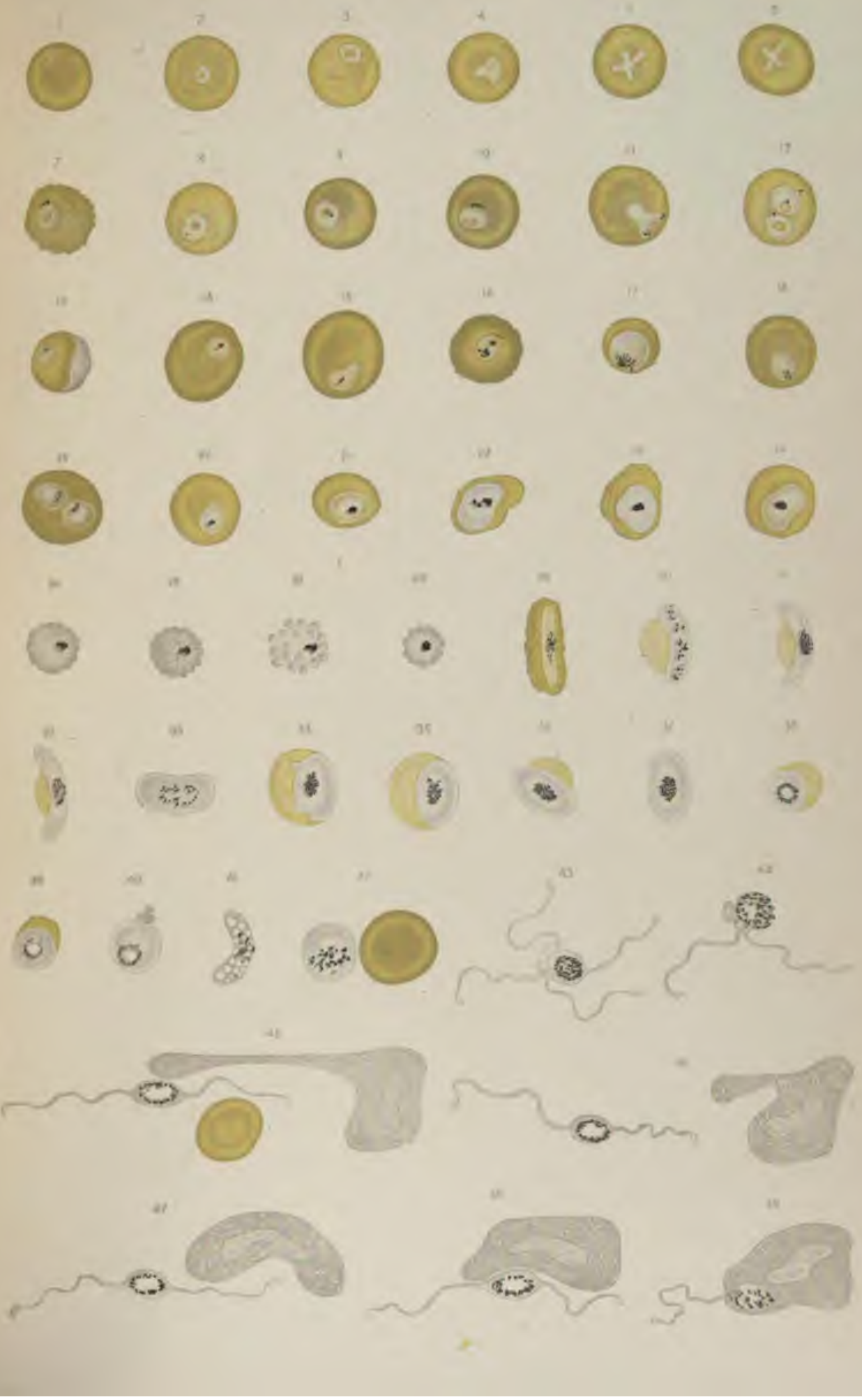


PLATE VI.

The Parasite of Aestivo-Autumnal Fever.



THE HÆMAMŒBA MALARIÆ IN THE MOSQUITO.—A mosquito of the genus *Anopheles,* when it sucks blood from an individual in whom the parasite has developed sexual forms, receives into its stomach bodies ready for the sexual process; in other words, gametocytes. The male bodies, or microgametocytes, develop long, actively moving flagella, called microgametes, which break loose from the organism and penetrate and fertilize the larger female bodies or macrogametes, these bodies being simply macrogametocytes which have extruded from their nuclear substances. The impregnated female now penetrates the wall of the mosquito's stomach, within which further development occurs. Within forty-eight hours there may be seen encapsulated in the muscular wall of the mosquito's stomach small, round, refractive, and granular bodies which have in them pigment granules much like those present in the parasite existing in the red blood corpuscles. At the end of a week the parasite has grown considerably, and it is found to be marked by radial striations forming sporoblasts. When this stage is completed the mother body, sometimes called the oöcyst, bursts, and so sets free in the cœlomic cavity of the mosquito a multitude of sporozoids. These sporozoids gain access to the veneno-salivary glands of the mosquito, and thence to the veneno-salivary ducts, from which they are ejected into the human being bitten by that mosquito. No sooner are the sporozoids deposited in the blood of man than they speedily become parasites which attack blood cells. Blood cells are therefore attacked in two ways: by parasites formed during the asexual or human cycle, and by parasites produced in the sexual or mosquito cycle.

Prevention.—The prevention of malarial fever consists in (*a*) protection from the bites of the anopheles by the use of mosquito bars, particularly at night; (*b*) in the removal of all marshes by filling them in or draining them so that the breeding-place of the mosquito is destroyed; (*c*) in the destruction of the larvæ of the mosquito by diffusing coal-oil over the surface of pools or ponds, and (*d*) by not permitting a patient who has the parasite in his blood to mingle with his fellows when the anopheles are present, for from him they derive their supply of infection. The latter measure can often only be carried out in private houses and barracks. Such patients should always sleep under a mosquito-proof canopy. Finally, it is a well-recognized fact that by the use of small doses of quinine taken daily (5 grains) it is often possible to prevent infection.

Pathology and Morbid Anatomy.—The changes produced in the body by the presence of the malarial parasite are much less pronounced if the tertian and quartan parasite is present than if the æstivo-autumnal is the offending body. Indeed, in many cases the morbid changes are so slight that they are speedily overcome by the natural processes of repair, and hence rarely cause death. For this reason our knowledge of the acute changes produced in internal organs is very limited. These changes may, however, for the sake of study, be divided into two forms, the acute and chronic. In the acute type the parts of the body which suffer chiefly are the blood, the liver, the spleen, the kidneys, and the alimentary canal.

The changes in the blood consist in a distinct decrease in the number of

red cells which are destroyed primarily by the growth of t
possibly secondarily by poisons produced by them. That
is active seems to be proved by the granular degeneratio
which is present in severe cases, and in the polychroma
met with in cells into which the parasite has not entered
diminution in the color-index, that is, of the individual ric
in hæmoglobin. The white blood cells are usually incr
portion of mononuclear leukocytes. Pigmented leukocyt
and, if the infection has been severe, large white cells (
to be seen heavily loaded with pigment. In some inst
pigment are seen floating in the blood serum, having been
blood cells destroyed by the parasite.

The liver, besides showing great congestion, may presen
and the capillaries may be found filled with a multitude o
all degrees of growth. The capillaries of the liver may
pigment particles.

The kidneys are enlarged and congested. They may
deposited pigment, and their capillaries may be filled by leu
pigment. The number of parasites found in the renal ve
compared to those found in the hepatic masses, is small.
diffuse nephritis may be manifest.

The spleen is swollen, soft, and its pulp is very dark.
blood cells which it contains are inhabited by the parasit
often in the stage of sporulation. So intense may be the
gestion of the spleen that it may be ruptured by sudden str

The mucous membrane of the stomach and bowels is
capillaries often contain the plasmodium.

If the bone-marrow is examined it is found to be filled
parasites and with pigment. The crescentic parasites a
numerous in these areas.

The chronic changes consist in profound anæmia,
diminution in red cells and in hæmoglobin and by the pres
red cells. The liver is deeply pigmented, often slaty in c
being deposited in the endothelial lining of the capillaries
cells of Küpffer, that is, the perivascular cells. The hep
commonly granular and a certain degree of hepatic cirrh
present.

The spleen also becomes markedly increased in siz
from the contained pigment and later, due to increase i
it becomes very firm, the "ague cake."

The kidneys are also markedly pigmented, and m
chronic diffuse nephritis. In certain cases of the cerebra
sites may be found in the vessels of the brain and a mal
been described. As in the acute form, the bone-marro
mented and the normal marrow may be replaced by red
normoblasts and megaloblasts are present in the majori
these changes are the result of æstivo-autumnal infection.

The blood is to be examined for the malarial organism microscope with an oil-immersion lens and by the use of the following methods:[1]

"There are two methods of examining the blood for plasmodia, in fresh preparations and in stained smears. Both require considerable training, as the artefacts produced by imperfect technique have often been mistaken for organisms.

"THE EXAMINATION OF FRESH BLOOD.—To examine the fresh blood, a puncture is made in the pulp of the finger and a perfectly clean cover-glass just touched to the top of the drop of blood which exudes from the puncture. The cover is then dropped without pressure on a clean slide. The diameter of the drop on the cover-glass should never exceed 2 mm., because if more be taken the corpuscles cannot spread out in a perfectly thin layer, but will overlap each other, and the preparation will be useless. The search for the organism should be made with a $\frac{1}{12}$ oil-immersion lens and a moderate illumination. The organisms are best recognized by the actively motile pigment in the clear, highly refractile cell body.

"THE EXAMINATION OF BLOOD AFTER FIXATION.—If the examination cannot be made at once, stained preparations may be made. The smear should be made on a slide or large cover-glass. It is best fixed in a mixture of formalin and strong alcohol for one minute. The proportions are 2 c.c. of a 10 per cent. solution of formalin to 100 c.c. of strong ethyl alcohol. The organisms are most easily found and studied in preparations colored with thionin. The formula for the stain is 20 c.c. of a saturated solution of thionin in 50 per cent. alcohol, added to 100 c.c. of 2 per cent. aqueous solution of carbolic acid. The stain requires several days to ripen, and then keeps indefinitely. Its action is rapid, requiring only about fifteen seconds to color the malarial organisms deeply.

"In order to demonstrate the nuclear chromatin of the malarial parasites, it is necessary to use special stains, the most useful being a modification of that originally devised by Romanowski for this purpose and improved by Giemsa. Two substances are necessary: First, an aqueous solution of 'methylene azure II, Grübler,' 8 dgm. to the litre; and second, an aqueous solution of eosin, 'extra water-soluble, Höchst.' These two stock solutions are permanent if kept in dark-colored bottles. The smears to be stained are fixed in methyl alcohol for about ten minutes. The staining mixture is prepared by adding 1 c.c. of the methylene azure II to 10 c.c. of the eosin solution. The staining mixture is poured into a Petri dish and the slide immersed in the fluid with the blood side down. The process is complete in from fifteen to thirty minutes. The slide is washed off for about ten seconds with a strong stream of distilled water, dried in the air without heat, and embedded in dammar dissolved in xylol. A simpler method has recenlty been published, which is better adapted for clinical work. The smear is fixed for two minutes or more in methyl alcohol, then stained for ten seconds with a 1 : 1000 aqueous eosin solution, the latter allowed to run off the slide, and the smear again covered with a few drops of a ½ per cent. solution of methylene azure II. In about from fifteen to thirty seconds the staining is complete. The slide should be washed in distilled water, dried, and examined directly with an oil-immersion lens, no cover-glass being necessary."

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[1] These directions are taken from the last edition of Pathology and Morbid Anatomy, by Delafield and Prudden.

Symptoms.—The symptoms of malarial infection may be divided into two classes: those due to the tertian or quartan parasites, which are much alike, and those due to the æstivo-autumnal parasites, which are very different from those produced by the more benign forms.

THE SYMPTOMS OF TERTIAN AND QUARTAN INFECTION.—The predominant symptoms of infection by these parasites are the development at regular intervals of a *chill* followed by a *fever*, and this in turn by a *sweat*.

The stage of onset begins with a feeling of *malaise*, in which *headache* and a general sensation of *wretchedness* are present. Patients who have had previous attacks are often able to recognize the fact that they will have a paroxysm in a few hours.

After the lapse of from one to five hours the *chill develops* and is often in the form of a severe rigor, in which the teeth actually chatter and the patient is entirely unable to control his muscular quivering. At this time the skin is cold, the face is pinched and often anxious in expression; but while the patient complains of being cold and presents all the external signs of a lowering of body temperature, his actual internal temperature is raised so that fever is really well developed while the so-called cold stage is still manifest. Thus, it is not uncommon for the rectal temperature to be as high as 105° while the patient is shivering and trying to "get warm." Associated with this part of the paroxysm the patient is usually *nauseated* and may actually *vomit*, so that to the depression of the chill is added the relaxation and semi-collapse of excessive nausea. Headache of the congestive type is also severe. The *urine* is copious in quantity and light in color, and the pulse small, rapid, and of high tension.

This stage of chill lasts from a few minutes to an hour or more, and is followed by the *true febrile stage*, in which the surface of the body becomes flushed and hot. In place of the cold, pinched expression the face now appears hot and flushed, the eyes may be brightened by the fever, and the pulse, which has hitherto been small and tense, becomes full and bounding. Headache, however, still persists, and active delirium may develop, but the actual temperature of the patient is rarely higher than during the stage of chill; indeed, it may be a little lower. The so-called febrile stage is not therefore any more febrile than the stage of apparent codlness, but the intense sensation of heat in the skin at this time when the physician touches the patient is noteworthy. This stage lasts from thirty minutes to several hours and comes to an abrupt ending by the development of the *stage of sweat*, in which the temperature drops to normal; the surface of the patient becomes bedewed with sweat, which may be so profuse that it is truly a "dripping sweat." The headache and general wretchedness now disappear and the patient drops to sleep exhausted bv the violence of his attack, but otherwise not ill.

The accompanying charts (Figs. 110 and 111) show the typical temperature changes:

As in all diseases, so in this one, it must be recalled that all cases do not go through these stages in exactly the same manner. Some individuals suffer from a very moderate chill and an equally moderate fever and sweat. Some suffer the paroxysm for twelve hours and some for a much shorter space of time.

There are three important physical signs which may be demonstrable in many if not all these cases. The *spleen* is found on palpation and percussion to be distinctly enlarged, extending below the level of the ribs; some *bronchial rales* are usually to be heard in the chest, and the lips are apt to be affected by *herpes* at the close or after an attack.

FIG. 110

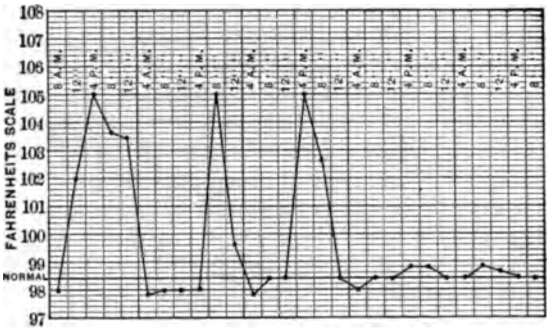


Chart showing daily paroxysm due to double tertian infection. One set of parasites segmented at 8 P.M. and the second set at 4 P.M. Paroxysm stopped by quinine on fourth day.

FIG. 111

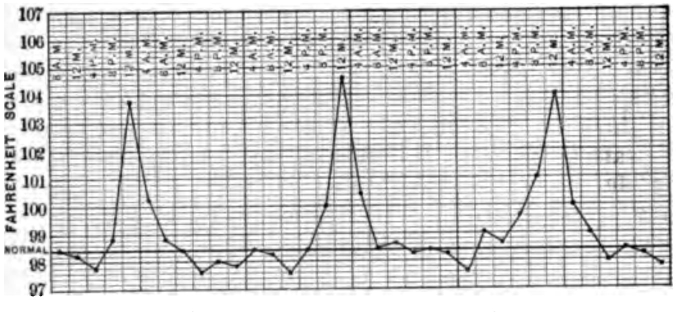


Chart showing paroxysms of tertian fever, the segmentation of the organism occurring at about 12 o'clock every other day.

The severity of all the signs depends, of course, upon the resistance of the patient and the virulence of the malarial infection.

There yet remains to be considered the periodicity of these attacks. They may occur daily, in which case they are called quotidian; or every other day, when they are called tertian; or every third day, when they are called quartan. The tertian type is due to the tertian parasite, which sporulates every forty-eight hours, so that the next paroxysm develops at the beginning of the third day; hence the term tertian, or third. When the attack occurs daily it is due to the fact that a double infection of the tertian

parasite has taken place, so that a different set of organi
day; in other words, it is a "double tertian infection." 1
frequently seen in the United States than any other type.
occurs at the end of every third day—that is, after the lap
hours, which is really therefore on the beginning of the fo
time—it is due to the quartan parasite. Sometimes, how
with this parasite is a double one, the sporulation in eacl
place separately, in which case the attacks occur on tw
with a free day following. In still other cases a triple quar
cause a daily or quotidian manifestation of the disease.
attack may therefore be due to a double tertian or a triple

On the days free from paroxysm the patient is entirel
fever, or sweats, and except for some impairment of strength
well as ever. It is for this reason that the disease is called "in

Diagnosis of Intermittent Fever.—The diagnosis of this
fever is easy in the majority of cases, but the physician sh
this conclusion until he has carefully excluded several
cause a similar temperature curve. These states are tu
lungs with chills, fever, and sweats; septicæmia and ulcer
with the same train of symptoms, and typhoid fever with
distinct character. The number of poor human beings
nauseam with quinine for malaria when they have tubercu
The history of the case, the physical signs in the lungs, an
attacks of chills and fever do not cease if quinine is given, p
is not the cause of the illness. Careful examination of th
discover sepsis by the finding of a definite focus of pus, a
ulcerative endocarditis can be discovered by the changes in
Typhoid fever can be discovered by the state of the to
bowels, the rose rash, and the Widal test of the blood. B
an absolute diagnosis is the discovery of the malarial par
by the use of the microscope. Unfortunately, this is onl
expert. (For examination of the blood for the parasite se

Prognosis of Intermittent Fever.—Intermittent malarial
considered a self-limited disease. It is true that individ
it recover without medication if they are otherwise health
able surroundings, but it is notorious that they are very lia
outbreaks of the disease when for various reasons their
impaired or the system is affected by change of climate.
means unusual for patients seemingly well of the infection
paroxysms on going to a higher altitude. If quinine is us
further infection is avoided, complete recovery usually
complication arises.

Treatment of Intermittent Fever.—In the treatment of inte
fever the fact is always to be borne in mind that in quinin
specific for the disease in that this drug, even in exceed
tion, is capable of causing the death of the malarial paras
is the cure produced by the proper administration of this d
form of remedial measure can be considered until after i

full opportunity to do its work. But, on the other hand, it must not be forgotten that quinine cannot destroy the malarial parasite until it (the quinine) has entered the blood, that it cannot enter the blood until it is absorbed, and that it is impossible for it to be absorbed if the gastroduodenal and hepatic circulation is so disturbed that catarrh of the stomach and bowels is present, making it impossible for the quinine to be taken up by the circulation. It is therefore essential, in almost every case of intermittent fever, that the bowels shall be thoroughly unloaded, preferably by full doses of calomel of which not less than 5 nor usually more than 20 grains are required, this in turn being followed by a saline purge. If, in addition to these measures, it is insisted upon by the physician that the patient shall rest in bed during the time that the quinine is being given, it is surprising how little quinine is needed to destroy the parasites; whereas, if these precautions are not taken, enormous doses may be given with no curative effect.

Quinine may be administered at two periods in connection with the paroxysm of intermittent malarial fever. One method is to administer the drug several hours before an expected paroxysm, with the hope that it may prevent sporulation, or at least destroy the spores as soon as they escape. This plan is always to be resorted to when the physician is confident that an attack is threatened and that a sufficient time will elapse to make it possible for the quinine to be absorbed. The other method consists in administering the drug in the sweating stage of a paroxysm, for the purpose of destroying the young parasites which have just escaped from the red blood corpuscles, in the hope that they may be destroyed before they can attack new cells. It is evident, therefore, that neither of these plans is contradictory, but are to be resorted to according to the period at which the physician sees the patient. It should always be the endeavor of the physician to have the quinine in the blood an hour or two before an expected paroxysm, and this means that it should be given several hours before when taken by the stomach.

When the attack is quotidian quinine must be given daily, when it is tertian it must be given every other day, and when it is quartan a full dose of quinine should be given on the day of the expected attack and smaller doses on the off days. If it is a double quartan infection the quinine should be given on the two consecutive days and only a small dose on the third day.

The quantity of quinine which is required varies of course with the severity of the infection and the rapidity of the absorption. Ten to 15 grains are usually sufficient in the milder types, but in the more severe types 30 to 60 grains are often required, although a larger quantity is usually needless if attention is given to the processes of absorption. The quinine should be given in powder in soft capsules easily dissolved.

So far as the attack itself is concerned, aside from the use of quinine, the physician may modify to some extent the effects of the paroxysm by the use of stimulants to support the circulation, and prevent congestion by using hot compresses or hot foot-baths. If the alimentary canal is overloaded with food or fecal matter, the stomach should be emptied by an emetic and the intestine by a purge or a large enema. Alcoholic stimulants, as a rule, are not advantageous. If the attack is exceedingly severe, a dose of morphine may be given hypodermically, or deodorized tincture of opium may be used. During the fever the patient may be relieved by a tepid sponging, but usually antipyretic measures are entirely unnecessary, and coal-tar products ought never to be employed. The vomiting, if excessive, may be controlled by hourly doses of a grain of oxalate of cerium with 5 grains of bismuth, and by counterirritation over the epigastrium. There is some evidence to prove that methylene blue has a destructive influence upon the malarial parasite. It may be given in the dose of 1 to 4 grains in capsules.

The Symptoms of Æstivo-autumnal Infection or Remittent Fever.—From the standpoint of severity and danger to the individual attacked, this type of malarial infection is by far the most important, although it is not as widely distributed, and therefore not as commonly met with as the tertian and quartan forms, except in certain localities which are usually tropical or semi-tropical. The term "æstivo-autumnal fever," applied to it first by the Italian investigators, Marchiafava and Celli, indicates that it occurs most commonly in the summer and fall months.

The symptoms of this type of infection differ markedly from the intermittent type of the disease in a number of particulars. Thus, it is not characterized by an intermittence of the *fever* and of the other signs of illness, but instead is *typically remittent;* that is to say, the fever and associated symptoms diminish in severity at times, but they do not entirely disappear. On the contrary, they persist in some cases to such a degree that the amount of remission is very slight indeed.

As the *gastrointestinal disturbance* due to this infection is very prone to be marked, and as the liver is usually much affected, this type of the disease is sometimes called "bilious remittent fever" or "bilious fever." *Vomiting,* which may be very persistent, and which may be markedly *bilious,* is a common symptom.

The remissions just spoken of may occur regularly, but in the majority of cases they are very irregular, both as to the time of their occurrence and to the degree of their individual severity. In some instances the *chills* and *sweats* are distinct; in others they are so ill-defined as to be scarcely noticeable. Further than this, the *febrile movement,* which is so sudden in onset and which ends so sharply by crisis in the intermittent form, is usually *gradual in onset* and equally deliberate in its fall in the remittent type. The temperature falls by lysis, not by crisis, in each paroxysm. This lack of sharply defined waves of temperature may deprive the physician of one of the most important means of recognizing that the illness is really due to the æstivo-autumnal parasite, and may mislead him into the diagnosis of typhoid fever, to which conclusion he is aided by the heavily coated tongue, the somewhat apathetic face, the enlarged spleen, the tympanitic belly, and the state of the bowels, which are lax in some instances and constipated in others, just as they are in typhoid fever. As many cases of typhoid fever have chills and sweats, the confusion is all the more readily made, particularly as a subacute bronchitis can often be discovered on ausculting the chest, just as it can be found in typhoid fever.

The severity of the symptoms varies over a wide range. In some instances the patient is only moderately ill; in other cases he is manifestly suffering from a severe malady. *Delirium* may be marked, and the *mental stupor* may be very noticeable. The *pulse* varies from 90 to 110, as it does in typhoid fever.

There still remains to be discussed that form of æstivo-autumnal infection which is so violent in its course that the term "*pernicious malarial fever*" has been applied to it. Pernicious malarial fever is very rare except in the tropics and in a few isolated places not far removed from these areas. It manifests itself in two forms:

(*a*) The *comatose form,* in which a patient is seized with symptoms resembling *intense cerebral congestion* or *apoplexy,* or develops *acute delirium* followed by semi-consciousness or *coma.* The fever is usually very high and the skin intensely hot. The patient may die in twenty-four hours without regaining consciousness, but if he survive so long a remission in the symptoms sometimes appears and recovery takes place; or, instead, a second paroxysm comes on in which death is likely to occur. These cerebral symptoms are due in part to the profound systemic disturbance caused by the infection, but chiefly to the formation of thrombosis of the cerebral vessels by a host of the parasites.

(*b*) The *algid* or *cold form,* in which the febrile movement is absent or very mild, and in its place signs of *collapse* and *exhaustion* appear, with great *coldness* of the surface of the body and a complaint of feeling cold on the part of the patient. With these symptoms of profound depression *gastrointestinal disturbances,* which are *choleraic* in type, may develop. There may be *violent vomiting* and *active serous diarrhœa,* the patient being at death's door by reason of the exhaustion. Here again the centralization of the symptoms about the intestines is due apparently to the accumulation of vast numbers of the parasites in the capillaries of the intestinal mucosa.

Complications and Sequelæ of Æstivo-autumnal Infection.—The most serious complication of infection by the æstivo-autumnal parasite is the development of *bloody urine,* which is of two forms, namely, a true hæmaturia with free blood cells in the urine, and a true *hæmoglobinuria* in which only the coloring matter of the blood is present. The former is obviously the result of a solution of continuity in the renal vessels, and the latter is equally obviously due to a destruction of a large number of red cells in the blood itself. This "black-water fever," as it is sometimes called, is practically unknown except where the æstivo-autumnal parasite is found. It occurs chiefly in certain of the Southern United States and in Africa and in Greece.

The symptoms associated with the development of the bloody urine are those which are often manifested by ordinary cases of severe remittent malarial fever. If the blood is examined before the urine becomes bloody, a large number of æstivo-autumnal parasites are usually found. On the other hand, after the bloody urine has appeared, it is commonly stated that an examination of the blood does not reveal the malarial organism.

The statement that quinine is capable of producing malarial hæmaturia is indorsed by so many practitioners of experience that it cannot be ignored, but we cannot help feeling that coincidence has been a large factor in the development of this view.

As sequelæ of malarial fever very marked anæmia, with *chronic enlargement of the spleen,* sometimes called "ague cake," develops; the *patient* is exceedingly *pallid*, the *abdomen* may be distinctly *enlarg* swelling and congestion of the liver and spleen, and th *swelling* about the *ankles*. There is *dyspnœa* on exertio *in the retina* may occur. Fever is usually not marked attacks of malarial infection superimposed upon the chro

It is quite remarkable that in young children, who suffer rial anæmia and cachexia with associated enlargement of th will take place under the administration of suitable tonic the avoidance of fresh malarial infection, and resort to a Even the spleen, which seems so hard and enlarged that its size may appear impossible, undergoes an extraordinary age, so that in adult life any evidences of it having been ch may have disappeared. Similar improvement sometime adults, but slight enlargement of the spleen is often a p of earlier malarial infection, even after the disease has absent for years.

Among the rarer complications of malaria may be m *pleurisy, ascites, intestinal hemorrhage, splenic abscess,* *of the lymphatic glands.* Cases of *orchitis* have been re military surgeons, and a rapidly developing and very pain observed in Sumatra, by Martin, who describes it as a fu mation of the testicles.

Choux has collected 147 cases of rupture of the splee He recognizes two forms: one which occurs in perso suffered very long from malaria, and whose spleens are no and one which affects the subjects of chronic malaria w enlarged and deformed by splenitis. In the latter class caused by traumatism. Playfair, during a residence o years in the East Indies, observed twenty deaths fro spleen.

Hemiplegia, either with or without aphasia, is the most ized cerebral complications. *Paraplegia* is less common is of still rarer occurrence. *Amaurosis, deafness,* and *perve* *smell* are occasionally observed. Symptoms of motor i tremor and *choreiform movements*, have also been reported tions, and a case of posthemiplegic malarial chorea has Boinat and Salebert. Paralysis of the lower extremities, d cord, occurs, and is often accompanied by loss of control and bladder. All of these disturbances may be transito some of the paralyses are rapidly fatal. *Multiple sclerosis* i quent nervous sequela, and *tetany* and *paralysis agitan* less frequent complications. Peripheral disturbances, such and anæsthesia, occur in a few cases, and supraorbital, intercostal neuralgia also complicate a small number of c

Of functional nervous complications and sequelæ, *hyste* and *insomnia* may be mentioned. The various *psychoses* a rence. Pasmanik in an analysis of 5412 cases of malaria of mental derangement. None of these patients affecte have an hereditary predisposition to insanity, and only 4.8 of the number were alcoholics.

Diagnosis of Æstivo-autumnal Fever.—Æstivo-autumnal fever must be carefully differentiated from typhoid fever and from the so-called continued thermic fever of hot climates. Its differentiation from typhoid fever is possible and should always be achieved; yet during the recent Spanish-American war the number of cases reported as malarial fever which were typhoid fever and the number called typhoid fever which were malarial was very large.

The presence of æstivo-autumnal infection is determined by the fact that the patient is, or has been, living in a region in which this disease is prevalent or at least may occur. It practically never occurs north of the State of Delaware in the United States, or in any of the northern parts of Europe. Again, its presence is determined by the discovery of the æstivo-autumnal parasite in the blood. This is not an easy test for the tyro, and the blood is not easy to obtain from the deeply seated organs in which the parasite is found. After the first week of the illness the absence of rose spots and the Widal reaction will determine the case not to be one of typhoid fever. Finally, good-sized doses of quinine will modify the fever of this type of malaria, but will not affect typhoid fever at all. The physician should make it a rule that if a case of fever does not yield to quinine in four days it is not malaria; provided, however, that by the use of calomel and other measures he has been able to put the alimentary canal in such a state that the quinine can enter the blood. If this cannot be done, then the drug should be given hypodermically.

Treatment.—The treatment of remittent fever is much more difficult than that of the intermittent form. The disease is more dangerous and often more rapid in its course. Further than this, the fever is so much more prolonged that it is of itself deleterious, and, as the patient does not have periods of intermittence in which he can make a partial recovery, strength is much more rapidly lost. The æstivo-autumnal parasite seems to be more resistant to the influence of quinine than either the tertian or quartan parasite, and, finally, it is much more difficult to cause the absorption of quinine in most cases of bilious remittent fever, both because vomiting is frequently a persistent symptom and also because absorption seems for the time being to be put aside. It is evident, therefore, that where vomiting is too persistent to permit of the administration of quinine by the stomach, it must be given hypodermically. The best way to administer it hypodermically is in the form of the hydrochlorate of quinine, which is soluble in ten parts of water, and which may be injected in the following manner: Dissolve 15 grains of hydrochlorate of quinine in 15 minims of alcohol and 2½ drachms of distilled water. Just before using, add a drop or two of dilute hydrochloric acid. The difficulty with the administration of quinine by the rectum in these cases is that absorption is too slow. But, nevertheless, the drug may be given in this manner dissolved in starch-water whenever it is advisable to get quinine into the blood by every possible avenue.

Even when it is impossible to administer quinine by the stomach, it is usually advisable to move the bowels freely, and this may be accomplished by the use of a Seidlitz powder given in divided doses, or by the employment of citrate of magnesium.

If the vomiting becomes so excessive as to be a dangerous symptom in itself, it may be controlled by hypodermic doses of morphine, or by 5 to 20 minims of spirit of chloroform given with the same amount of compound spirit of lavender or of cherry-laurel water. Counterirritation may also be applied over the epigastrium in the shape of a mustard plaster. Some cases are also benefited by the application of hot compresses over the liver. The value of large doses of calomel, amounting to 20 or even 30 or 40 grains, for their effect in overcoming hepatic torpor cannot be denied. All practitioners of experience in intensely malarial districts are agreed as to this point.

Should hæmaturia or hæmoglobinuria complicate a case, a careful consideration of the stage of the disease is essential. The value or harmfulness of quinine in malarial hæmaturia is still a " bone of contention " with many practitioners, some claiming that the quinine is capable of actually producing bloody urine, and others asserting equally positively that it is always needed. The writer has expressed the view on several occasions that some of these cases of bloody urine, complicating malarial fever, may be due to an associated parasite upon which quinine has little influence. In a certain number of cases it is probably true that the bloody urine is a sequel rather than an accompaniment of the development of the parasite in the blood. Under these circumstances, as enormous doses of quinine can have little influence upon the malarial parasite and also may irritate the kidneys, it is conceivable that the use of this drug at such a time is distinctly contraindicated; whereas, if an examination of the blood reveals the presence of the æstivo-autumnal parasite, then the quinine must be given, since the danger of producing hæmaturia by its administration is more than counterbalanced by the desirability of destroying the cause of the illness. In other words, to give quinine in some cases of malarial hæmaturia, when the specific parasite is not present, is like locking the door after the horse is stolen; while in others its administration is timely and appropriate.

Many physicians of large experience strongly urge the use of hyposulphite of sodium in doses of from 15 to 60 grains every four hours for this complication.

LATENT MALARIAL INFECTION.—*Latent malarial infection* is probably much more common than physicians believe, although the laity have an exaggerated view of its occurrence. Craig has reported the results of examining the blood of 47 men in one company of the United States Army, all of whom had been exposed to malarial infection in the Philippines, but all of whom were at least well enough to be on duty. Twenty-seven of them had the parasite in their blood, and 25 were infected by the æstivo-autumnal parasite. This persistence of infection not only possesses ordinary interest, but shows that by the distribution of returning troops to various parts of the country this parasite may be disseminated in areas hitherto uninfected. Further, as Craig points out, the development of a masked malarial infection may greatly mislead the surgeon, both before and after an operation.

PSOROSPERMIASIS.

This term is applied to an exceedingly rare condition in which psorosperms become parasites, growing in cells and producing nodules. These nodules may be large enough to be felt through the abdominal wall. The patient presents symptoms which are like typhoid fever in character. There is diarrhœa, stupor, some fever, hepatic and splenic tenderness, and feeble circulation. Autopsy in such cases has shown the presence of masses closely resembling tubercles, which are scattered over the liver, the spleen, the peritoneum, and in the kidneys.

Another form of infection by sporozoa has been described as occurring in the skin, but Stelwagon states that the condition called psorospermiasis by Darier is now known not to be due to this cause. On the other hand, Rixford and Gilchrist, in Baltimore, have recorded two cases in which tuberculosis of the skin was thought to be present for eight years. During this time the lymphatic glands were enlarged, other parts of the body became affected, and finally death ensued. Numerous nodules, looking like those of tuberculosis, were found scattered very widely through the body, and these were found to contain large numbers of sporozoa.

TRYPANOSOMIASIS.

Definition.—Two diseases, or rather two phases of one disease, are recognized in man as due to infection by *trypanosomata*. These are, first, an ill-defined fever resulting from invasion of the circulation by the *Trypanosoma gambiense*, and second, African lethargy, or sleeping sickness, due to the presence of the same parasite in the cerebrospinal fluid.

The trypanosomata are flagellated protozoa, and were first discovered by Gruby in 1843 in frogs, and by Doflein in 1845 in rats and hamsters. Since that time they have been found in practically all vertebrates. These organisms were first supposed to be spirilli before their animal nature was understood. In the large majority of instances they are not pathogenic. As far as our present knowledge goes, the varieties represent distinct types, confined to the particular animal which they infect. At least six trypanosomata that are pathogenic occur in mammals. Thus, the *Trypanosoma Evansi* and the *Trypanosoma Brucei* are the causes of very fatal diseases, known as *surra* and *nagana* among horses, mules, camels, buffaloes, and wild animals. *Trypanosoma equiperdum* is the cause of an exceedingly fatal disease of horses in Algiers and the Mediterranean coast. *Trypanosoma equinum* is also the cause of a fatal disease of horses in South America. *Trypanosoma Theileri*, the largest of the known trypanosomata, is the cause of a serious cattle disease in South Africa. The most widely distributed is *Trypanosoma Lewisi*, the parasite infecting rats. This particular variety is very common in the rats of the United States, and has been found in practically every city where search has been made for it. Finally, we have the trypanosoma which infects man.

A trypanosoma consists of a leech-shaped, granular body from 13µ to 25µ long and from 2µ to 4µ wide. This body contains a nucleus and a rod-shaped centre known as the centrosome, or micronucleus. Along one edge of the parasite, beginning at the centrosome, is a delicate, fringe-like membrane known as the undulating membrane, upon the outer edge of which is a single flagellum extending from 7µ to 15µ beyond the anterior end of the parasite. In freshly drawn peripheral blood these parasites are seen to be in most active motion, progression being in the direction of the flagellum. In hanging-drop preparation they live several days, and as long as fifty days if the slide be kept cold and moist. McNeal and Novy have succeeded in cultivating *Trypanosoma Lewisi* and *Trypanosoma Brucei* on a culture medium of agar and defibrinated rabbits' blood. This is the first instance in which animal parasites have been obtained in pure culture.

With respect to transmission of infection by trypanosomata numerous carriers have been found. Experimentally, animals may be infected by intraperitoneal and subcutaneous injection of the parasites, or by ingestion of the parasites into the stomach. Infection may be carried by food and water, by the bite of fleas, lice, mosquitoes, ticks, and several varieties of flies. The infection of *Trypanosoma equiperdum* is conveyed during coitus.

The prevailing evidence is that human trypanosomiasis is disseminated or conveyed and inoculated by suctorial flies. The view that one fly only can act as host for a particular trypanosome is generally held, but is by no means established. The known transmitters of trypanosomiasis, of which our knowledge appears accurate, are the *Glossina palpalis* for those of trypanosoma fever and sleeping sickness, and the *Glossina morsitans* for surra. Of other means of natural dissemination we know nothing.

Human Trypanosomiasis (Trypanosoma Fever).—The first reported case of trypanosoma in man was made by Nepveu, and his paper contains a fair drawing of the parasite. Forde, in 1901, described the parasite in the case of a European from the Gambia River Colony suffering from an atypical fever. Since that time Manson, Dutton, Todd, and others have reported cases, nearly all of them from the Congo and Gambia River district. Manson's case occurred in a woman aged forty years. The temperature ranged from 97° in the morning to 100° in the evening. The pulse was always rapid and feeble. Erythema was a constant feature in the case, and was first observed when the fever began. Œdema was also present, and was most pronounced on the back and face. There was marked enlargement of the spleen. Duthen reports a case of a European working along the Gambia River who suffered from an irregularly remittent fever. The trypanosomata were especially plentiful during the pyrexia. The symptoms included *extraordinary weakness, œdema of eyelids, enlarged and tender spleen,* and *rapid pulse.* Malarial organisms were never found. This patient recovered under the use of arsenic.

The clinical phenomena in the reported cases are chiefly these: In some cases the parasites occur in the blood without the patient manifesting any conspicuous symptoms. In other cases there is an irregular fever which may be high, continuous, or remittent in type. It does not yield to quinine. After persisting from three days to two or three weeks, it is followed by an apyretic interval. During the course of the fever large erythematous patches occur all over the body, associated with irregularly distributed areas of cutaneous œdema. The œdema and erythema may or may not coincide. The œdema is most marked on the face, especially on the lower eyelids. The pulse is rapid and running, a peculiarity also observed in sleeping sickness. The lymph nodes enlarge and their expressed or extracted juice usually contains the parasite.

In many cases the history of an inflamed and painful insect bite can be obtained.

The parasites are found free in the peripheral blood and never in the corpuscles. They are not numerous, varying from one to twenty in a cover preparation. They may be absent for days at a time. In the case reported by Manson experimental inoculation on animals was negative, showing the distinct nature of the parasite. It now appears quite clear that infection results from the bite of a tsetse fly (*Glossina palpalis*) which is the carrier of the parasite. In Dutton's case there was the history of the patient being bitten by a rat.

The blood condition is interesting. There is a moderate degree of anæmia in all cases and a marked increase in the large mononuclear leukocytes, running as high as 22 per cent. The increase in these cells seems rather constantly associated with sporozoal infection, just as metazoal parasitism is accompanied by eosinophilia.

Treatment.—Some of the reported cases have recovered on large doses of arsenic administered in the form of sodium cacodylate or arsenate of iron given hypodermically. Methylene blue and quinine in large doses have also been tried, but are not of any particular value. The patient should be kept in bed in a warm room, and should be fed on simple and nourishing food. Treatment of the fever is symptomatic. The most important preventive measure is to keep the individual where he cannot be bitten by flies, and, if ill, where he cannot act as a centre of infection.

African Lethargy (Sleeping Sickness).—Sleeping sickness is a chronic disease characterized by increasing lethargy, and, after an exceedingly chronic course, death occurs from coma or from inanition—"a negro sleeping himself to death."

Sleeping sickness is at present confined to tropical Africa, principally along the west coast. The northern limit of its extension is the Senegal River; the southern limit is the Portuguese Colony. It is common in Senegambia, along the Gold Coast, and at Old Calabar. It has existed for a long time in the basin of the Congo River from Stanley Falls, in the heart of equatorial Africa, to the lower Congo. It has recently extended from Victoria Nyanza to the head-waters of the Nile. In the last two years the disease has assumed epidemic proportions in Uganda and many thousands of the natives have perished. In the days of the slave traffic sleeping sickness was frequently carried to the West Indies, Southern United States, Brazil, and the Bahamas, but it never succeeded in establishing a foothold in any of these places.

Etiology.—For a long time the *Filaria perstans* was supposed to be associated with sleeping sickness, principally on account of their coincidence in the infected areas. Later studies have shown that filaria has no connection with sleeping sickness, and that many areas infected by sleeping sickness do not show infection by the *Filaria perstans.*

The older views that sleeping sickness is due to poisoning, intoxication, and filaria have been superseded by the demonstration of a trypanosoma in the cerebrospinal fluid by Castellani, and the confirmation of this observation by Bruce and other members of the English Sleeping Sickness Commission, who have successfully propagated the disease in monkeys. The trypanosoma of sleeping sickness is indistinguishable from that of trypanosoma fever, and, like the latter, is also found in the blood. Apparently the advent of sleeping sickness in a patient having trypanosoma fever is determined by colonization of the parasites in the cerebrospinal fluid. The exact relation of the hypnococcus, described by Castellani and other members of the Spanish Commission, as the cause of the disease is not as yet perfectly clear; apparently it is only a terminal infection.

Sleeping sickness attacks persons of all ages. In the endemic area negroes are the most susceptible, mulattoes less so, and Moors contract the disease still less frequently. I know of but one authenticated case of sleeping sickness in a white man on record. The tsetse-fly (*Glossina palpalis*) is the intermediate host. It has been shown experimentally that this fly, after feeding on a case of sleeping sickness, is capable of conveying the dieases to healthy monkeys, and that flies from the endemic area were found to be infected even without such feeding. It is possible that the disease may exceptionally be conveyed in other ways. The negroes believe that contagion is carried by water and also by the saliva of the sick.

Pathology.—Mott has shown that the lesion of sleeping sickness is an extensive meningoencephalomyelitis. In the cord and brain extensive round-cell infiltration is found about the capillary vessels. The cerebrospinal fluid is deeper in color than normal owing to the presence of numbers of red blood cells. Besides these, numerous leukocytes and the specific trypanosoma are found. In the latter stages of the disease there is some ground for believing that there is a concomitant—in a terminal sense—streptococcus (hypnococcus) infection.

Symptoms.—The incubation period is variable, but always long. The natives believe that the disease may develop as long as seven years after exposure. As a matter of fact numerous instances are on record where the disease has appeared in negroes several years after leaving the endemic area settling in other countries. It would appear, from the meagre knowledge and of the parasite now available, that it may be found in the peripheral blood without producing any symptoms. The causes that determine colonization of the parasites in the cerebrospinal system are unknown.

The disease sometimes begins with marked psychical prodromata, including *epileptiform seizures*, *melancholia*, and even *transitory mania*. In the larger number of cases there is *headache*, *vertigo*, *puffiness of the face*, and *slight fever*. At this point the *lethargy* begins. The patient at first is *somnolent* or *stupid*, but he can easily be roused for nourishment or to attend to the calls of nature. When so awakened his *gait* is *staggering* and the moment he is released he sinks into a *deep sleep*. In the early stages there are no evidences of paralysis, tremor, or convulsion. The patellar reflexes are decreased, sometimes abolished. Gradually the lethargy deepens until finally the patient can only be aroused with the greatest difficulty, if at all, and immediately falls again into a deep sleep. Partly from the disease itself, but largely because the lethargy prevents regular nourishment, *nutrition fails*, *emaciation* becomes progressively more marked, and *bed-sores* develop. Toward the close paralyses of various muscle groups develop, *convulsions* occur, and *fatal coma* supervenes.

Diagnosis.—The symptoms are fairly characteristic, but a positive recognition of the disease is rendered easy by lumbar puncture, centrifugalization of the cerebrospinal fluid, and demonstration of the parasites by a microscopic examination of the sediment.

Prognosis.—The course of the disease is chronic. Cases may last from three to four years. The prognosis is bad. There is no record of the recovery of an authentic case of African lethargy.

Treatment.—With regard to treatment it has been found that purging in the early stages does good and in some cases temporarily arrests or delays the disease. Massive doses of arsenic are of some service and should be used on account of their supposed value in trypanosoma fever.

KALA-AZAR.

Definition.—Kala-azar or Tropical Splenomegaly, sometimes called Dum Dum Fever, is a chronic infectious disease, characterized by long-continued fever, extreme emaciation, profound anæmia, marked enlargement of the liver and spleen, and a characteristic pigmentation of the skin.

Much uncertainty has existed as to the nature of kala-azar, and our present knowledge rests largely upon the investigation of Leishman, who showed that it is a form of trypanosomiasis. (See Trypanosomiasis.) This investigator, with Marchand, James, Bentley, Rogers and others, has found the so-called Leishman bodies in persons suffering from typical kala-azar in various parts of the world, as in the Egyptian Soudan, Algiers, and in Assam. Airde has also made confirmatory investigations in China. These Leishman bodies are minute, spherical, oval forms closely resembling the chromatin masses seen in stained trypanosomes. They are found chiefly in the spleen and liver, and can be obtained during life by puncturing the spleen with a fine exploring needle, the fluid contained in them being expelled upon a glass slide and stained by the Romanowsky method.

The Leishman bodies or parasites are capable of developing into flagellated organisms closely resembling trypanosomes, but differing somewhat in the position of the flagellum.

Manson believes that the intermediate host of this parasite is some scavenger-fly which derives the parasite from the intestinal or other discharges of the patient and then infects the human being by a bite. There may be a sexual multiplication in the fly, but in the human host the parasite multiplies by fission, and Manson thinks that this multiplication is asexual.

Pathology and Morbid Anatomy.—The autopsy in a case of kala-azar shows enormous enlargement of the spleen, which is firm and friable. The liver is also greatly enlarged and toughened in texture. The bone-marrow and the organs just named are crowded with the parasites. Leishman bodies can also be found in the lymphatic glands, the suprarenal capsules, in the testicles, and in the inflammatory exudates in the pleura and peritoneum. The direct cause of death seems to be an associated dysentery or pneumonia.

Symptoms.—The symptoms in onset resemble those of malarial fever, being characterized by daily chills and fever, followed by free sweating, these symptoms recurring about the same time every afternoon. After a period of ten days or two weeks these symptoms diminish and a period of remission occurs followed after another period of ten days or two weeks by a return of the paroxysm. This may last for weeks or months. Occasionally the remissions already spoken of fail to occur, and profound inanition develops after some months. In still other cases the febrile movements are exceedingly irregular and varied. Enlargement of the liver and spleen begin early. The patient complains of languor, dyspnœa, and the general manifestations of profound anæmia. The constant symptoms are fever, enlargement of the liver and spleen, the progressive emaciation, and grave anæmia,

Treatment.—No method of treatment has as yet been discovered which produces any good results.

NEMATODES.

Ascariasis.—*Ascaris lumbricoides*, or round worms, are found in the small intestine of man more commonly than any other parasite.

They are not segmented as are the cestodes, but occur as smooth worms, not unlike an ordinary earth-worm, except that they are provided with small papillæ or hairs. The worm also possesses longitudinal striæ and transverse rings, a mouth, and an anus. They are not hermaphroditic, but occur in the form of the male and female worm.

They are met with far more frequently in children than in adults. The female worm is of a light-brown, or red, color, and is usually about 10 to 20 cm. long and 0.5 cm. thick. The male is about one-half the size of the female.

How these worms gain access to the body is not known, although it may be by ingestion. While they most commonly are found in the small intestine, they occasionally find their way from the intestine into the stomach, and cases are on record in which they have wandered into the œsophagus and mouth, and even into the nose and bronchial tubes. Cases have also been reported in which the migration of a worm into the gall passages has produced obstruction, and still other instances are recorded in which they have found their way through an ulcer, or through a perforation in the appendix vermiformis, into the peritoneal cavity. As a rule they are present in the intestines in numbers and do not occur singly. In rare cases coiled, mottled masses of lumbricoids have caused intestinal obstruction.

A form of round worm, somewhat like that found in man, is found in the intestine of cats and dogs, but it is considerably smaller in size and does not infest man.

Symptoms.—The symptoms of the presence of this worm do not differ materially from those produced by the tapeworm (which see). Occasionally, however, this worm seems to have the power of producing an irritant poison which not only causes intestinal irritation, but when absorbed may cause great nervous irritation and apparently be responsible for convulsions in young children.

Treatment.—The treatment of a patient suffering from *Ascaris lumbricoides* is abstinence from food for twelve or eighteen hours and the administration of 1 drachm of the fluid extract of spigelia, or 2 drachms of the more old-fashioned, but efficacious, fluid extract of spigelia and senna. In other cases, 5 to 15 minims of the oil of chenopodium in capsule or emulsion, or on sugar, may be administered. In still other cases from 2 to 5 grains of santonin may be given in tablets or troches. All of these drugs should be followed by castor oil or a saline purge in order to sweep out the worms while they are poisoned by the drug.

Oxyuris Vermicularis.—Under the name *Oxyuris vermicularis* or threadworm, sometimes called pin-worm, a very small nematode worm exists in the rectum of young children and is sometimes found in adults. Occasionally it infests the entire colon. The length of the female is about 10 mm., and of the male about 4 mm. This worm may be present in great numbers without producing any symptoms whatever. Some irritation about the anus may be the only disturbance produced by their presence.

Seat-worms are to be removed by the injection into the bowel of soap and water, which, after it is passed, is to be followed by a pint of warm water which has been medicated by boiling in it from ½ to 1 ounce of quassia chips.

Trichina Spiralis.—A patient infected by the parasite known as the *Trichina spiralis* is said to suffer from *trichiniasis*. This parasite was first described by Owen in 1835. It was found in the flesh of the hog by Leidy in 1847, and in a human being by Zenker in 1860, the patient, a young girl, being thought to be a sufferer from enteric fever until at autopsy the parasites were found free in the bowel and encapsulated in the muscles.

Etiology.—In practically every case the infection of a human being comes from eating the flesh of an infected hog. It is scarcely necessary to state that infection does not occur if the pork has been well cooked.

The frequency with which the disease occurs is not known, but it is not as rare as some have thought. Williams found it present 27 times in 505 unselected autopsies in Buffalo, New York.

If the muscle of a man infected by this parasite is examined, tiny little white or gray dots will be found upon its surface and through its texture; later the parasites encapsulate and look like deposits of calcareous material of about the size of miliary tubercles. If such a calcareous deposit be opened it may be found to contain the embryo of the parasite, or if the worm be dead a granular detritus only is present.

When uncooked meat containing this parasite is swallowed, the digestive juices dissolve the capsule of the parasite, and in this way the embryos are set free in the stomach. Here they rapidly develop and become sexually mature in about seven days. The female parasite gives off an immense number of embryos, so that it is estimated that one parasite may throw off from one to two thousand young. These parasites soon find their way through the wall of the intestine, enter the lymph spaces, and so reach the circulation, by which they pass to the muscles. Their favorite position for settlement is the striated muscles. They enter the muscular connective tissue and then the sarcolemma, where they coil themselves and cause a disintegration of the contractile substance. Here they become encapsulated in about six weeks, partly by the inflammatory exudate which is produced by their presence, and partly by the calcareous material which they seem to have the power of collecting. In these capsules the parasite remains alive for a very long period of time, possibly for twenty-five years. Occasionally, however, it dies, and the entire mass undergoes calcification.

When one of the domestic animals swallows meat infected in this way, the same process takes place in the muscles as occurs in the muscles of man. In the muscles of the hog the parasite may escape notice, as it often lacks the calcified capsule. Moreover, an infected hog may be in excellent health.

Pathology and Morbid Anatomy.—The lesions of trichiniasis consist in gastrointestinal irritation, overgrowth of the lymph nodes in the abdominal cavity, occasionally bronchopneumonia with great swelling of the bronchial glands, still more rarely fatty degeneration of the liver, and constantly a parasitic myositis due to the embryos invading the muscles. Almost every muscle of the body may be found infected; but where the number of trichinæ is not very great, the muscles of the neck, the intercostal muscles, and the diaphragm seem to be the parts in which the greatest aggregations occur. Furthermore, the greatest number of trichinæ are usually found near the insertions of the muscles.

Symptoms.—The symptoms consist in *muscular soreness* and *pain*, and disinclination to move. A diagnosis of muscular rheumatism is often made because of these symptoms. *Headache, puffiness of the skin about the eyes* and *nose*, and *moderate fever* are also present. Not rarely the symptoms may closely resemble those of typhoid fever, with *great prostration* and *emaciation*. For some unknown reason a *marked leukocytosis* develops, which is peculiar in the fact that the *eosinophile corpuscles* are chiefly increased. From investigations made by Opie it would appear probable that this eosinophilia is of some value from both a diagnostic and prognostic standpoint. The eosinophiles are not greatly increased, if the infection by trichinæ is excessive, and their greatest development seems to occur at about the time that the embryonal trichinæ are passing from the intestine by way of the lymphatics and blood to the muscular tissues—that is, during the third week after the ingestion of the trichinatous meat.

Diagnosis.—In a suspected case the diagnosis may be reached by taking a small piece of muscle and examining it with a microscope. The stools of the patient should be flattened to a thin layer between two sheets of glass resting upon a black background, and then examined by means of a hand magnifying glass, when the parasite may be found as small, short, glistening, thread-like bodies.

Prognosis.—The prognosis depends largely upon the severity of the infection. In the worst outbreaks the mortality may be as high as 70 per cent. Many cases recover by the end of a fortnight. Others remain ill for weeks or months before recovery takes place.

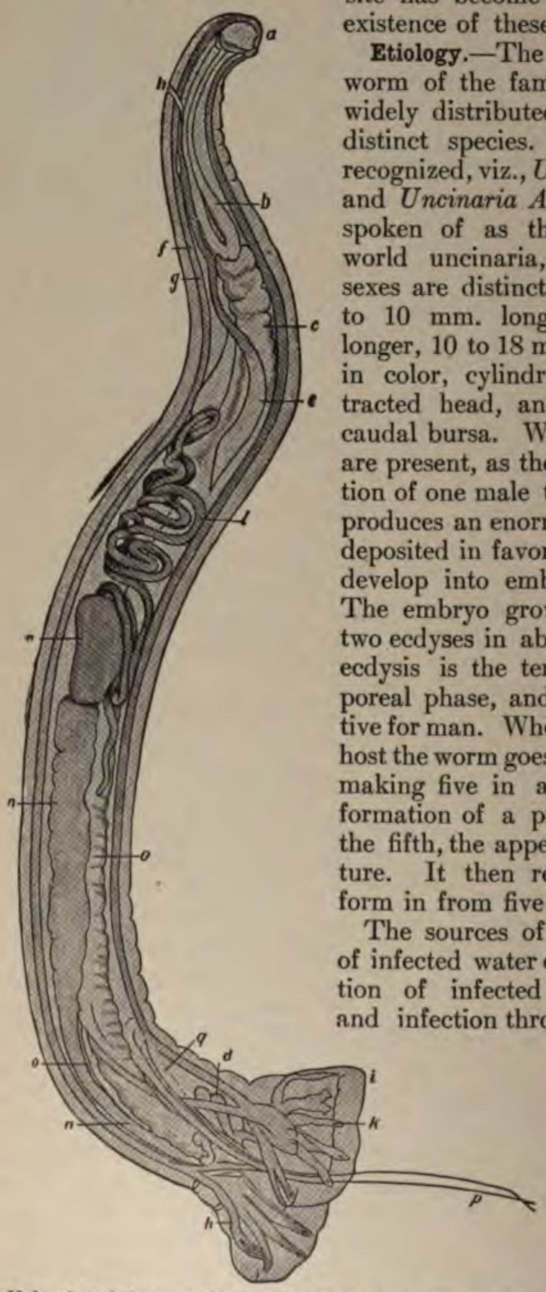
Treatment.—There is no treatment which can be directed to the removal of the parasite after it has entered the muscles. The only thing the physician can do is to give a nutritious diet, and relieve pain or other symptoms by symptomatic remedies. If the discovery is made that the patient has swallowed trichinatous pork within a few hours, then 5 grains of thymol followed by a dose of castor oil or sulphate of magnesium should be ordered to kill the parasite and sweep it out of the intestines before it can migrate into the tissues.

Uncinariasis (Ankylostomiasis). **Definition.**—Uncinariasis is an infection by different varieties of worm of the uncinaria species; it occurs not only in man, but in many of the lower animals. The parasite is often called the *Ankylostomum duodenale*, or hook-worm. The chief symptoms are severe anæmia, abdominal pains, asthenia without emaciation, and œdema. The parasite was described as uncinaria by Froelich in 1789; Dubini in 1843 gave it the name ankylostoma.

The condition is also known as "brickmakers' anæmia," "Egyptian chlorosis," "miners' anæmia," "miners' cachexia," "miners' disease," "Porto Rican anæmia," "St. Gothard's tunnel disease," "tunnel disease," "tunnel anæmia," "tropical chlorosis," and "hook-worm disease," besides a host of local names. It is one of the most ancient diseases known to man, for it was described by the Egyptians 3500 years ago.

Frequency.—Uncinariasis occurs in all the tropical and practically all the subtropical world. According to Thornton it is the greatest enemy of the human race in the tropics; greater even than plague or cholera. In portions of India 75 per cent. of the population are said to be affected. In Egypt this worm is found at nearly every postmortem. It has been the most disabling of all diseases in the Egyptian army, as well as the greatest cause for the rejection of recruits. In Ceylon its ravages are said to be more serious than those of cholera. Harris has found it in Georgia and Florida, and believes it is the common cause of the severe anæmias of the Southern United States that have hitherto been regarded as malarial. Stiles has also made very valuable studies of its characters and recurrence in the Southern United States. In Assam it is almost universal, 299 cases having been found in 300 postmortems. According to Alden, 22.5 per cent. of the total death rate of Porto Rico is ascribed to tropical anæmia due to uncinaria. In more temperate regions it has been found in nearly all our States as far north as New York. In the Cornwall and Westphalian mines the disability caused by this para-

FIG. 112



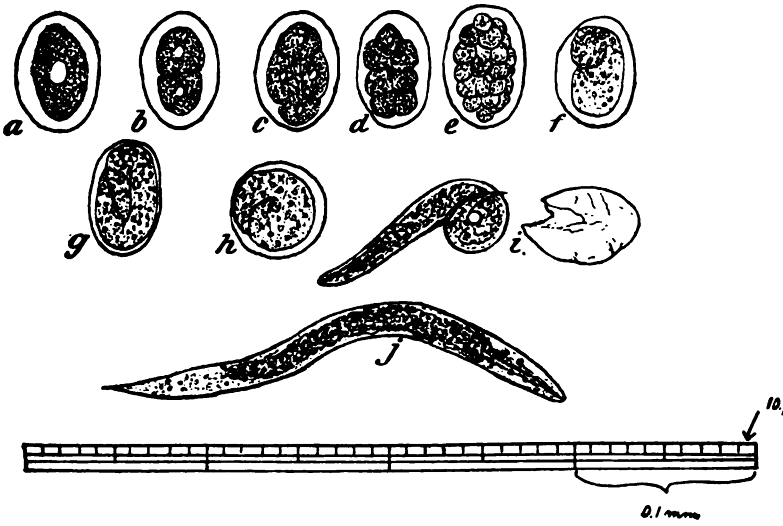
Male of Ankylostoma duodenale: *a*, head; *b*, œsophagus; *c*, gut; *d*, anal glands; *e*, cervical glands; *f*, skin; *g*, muscular layer; *h*, excretory pore; *i*, tri-lobed bursa; *k*, ribs of bursa; *l*, seminal duct; *m*, vesicula seminalis; *n*, ductus ejaculatorius; *o*, its groove; *p*, penis; *q*, penile sheath. Magnification, 20. (After Schulthess, from Ziegler.)

site has become so great as to threaten the existence of these industries.

Etiology.—The uncinaria is a nematode worm of the family *Strongylidæ*. It is very widely distributed in the animal world, in distinct species. In man two species are recognized, viz., *Uncinaria duodenale* (Dubini) and *Uncinaria Americana* (Stiles), commonly spoken of as the old-world and the new-world uncinaria, or hook-worm. The two sexes are distinct; the male worm is from 8 to 10 mm. long and the female slightly longer, 10 to 18 mm. They are grayish-white in color, cylindrical in shape, with a contracted head, and in the female a broad caudal bursa. When male and female worms are present, as they usually are in the proportion of one male to three females, the female produces an enormous number of ova. When deposited in favorable surroundings these ova develop into embryos in twenty-four hours. The embryo grows rapidly, passing through two ecdyses in about five days. The second ecdysis is the termination of the extra-corporeal phase, and the embryo is now infective for man. When taken into its appropriate host the worm goes through three more ecdyses, making five in all; the fourth marking the formation of a provisional buccal armature; the fifth, the appearance of the definite armature. It then reaches an adult or mature form in from five to six weeks.

The sources of infection are the ingestion of infected water or food, the accidental ingestion of infected earth from soiled hands, and infection through the skin during or after the second ecdysis of the worm. The mode of infection by earth is obvious. In all regions affected by uncinaria, dirt and clay eating are very common habits. There is no doubt that geophagy in infected areas is a common manner of taking the disease; but it is also true that this habit does not show itself, in many cases, until the disease is fully developed. In these cases it seems clear that the earthy matter is eaten in obedience to an instinctive craving, and that it brings relief by mechanically loosening a number of the parasites. That infection may take place through the skin, the experiments of Looss and many clinical observations, as well as accidental laboratory infections, have made quite apparent. It has often been observed that children, walking barefooted through infected ground, became infected. The disease known as Pani-Ghao, and the "ground itch" of the South, are probably skin infections by this parasite, the lesions representing the point of entrance of the parasite into the human host. Looss experimented with the young worms in a mixture of earth and charcoal. This mixture was placed on the skin of dogs without scarification or friction. The parasites were found in the intestines as rapidly as when experimental infection by the mouth was made. While making these experiments he accidentally allowed some of the mixture to remain on his own hand and himself became infected. Air-borne infection occurs very rarely, if ever.

FIG. 113



Ova and embryo of Uncinaria Americana: *a*, unicellular ovum; *b*, *c*, *d*, *e*, ova showing various stages of segmentation; *f*, *g*, ova containing larval uncinariæ; *h*, peculiarly shaped ovum; *i*, larval worm just emerged from shell; *j*, larva extended after emergence. (Stiles.)

Prophylaxis.—Prophylaxis of uncinariasis demands the exclusion of all infected persons from earth-workings. Where large bands of laborers are collected in districts in which the disease is prevalent, in mines, in tunnels, and in excavations of all kinds, systematic examination of all cases of anæmia should be made. Defecation in the workings or tunnels should be rigorously prohibited. Water-tight latrines should be provided, and the contents rendered harmless by a cheap disinfectant. In the Arlberg tunnels the pail system has been recently used with good effect. Personal prophylaxis should include careful washing of the hands before eating, and the wearing of sound shoes when working in suspected soils.

Pathology and Morbid Anatomy.—Postmortem the subcutaneous fat, the panniculus adiposus, and the mesenteric fat are fairly well preserved. The parasites are found in the jejunum, still attached to the bowel wall if the section is early, or free in the intestinal contents if the section is delayed. They vary in number from 100 to 600 or more. The jejunum is covered with old and recent pinhead bloody extravasations. The bowel is thickened in spots, and there may be small cavities in the bowel wall filled with blood and containing the heads of one or two parasites. The liver and kidneys commonly show some degree of fatty degeneration. The cause of the anæmia is probably twofold—the mechanical abstraction of a considerable amount of blood by the parasites, and a hæmolytic effect from a toxin elaborated by the worm. That hæmolysis occurs is indicated by increased iron in the liver, the occurrence of hæmatoidin in the liver and kidneys, as well as free iron in the stools.

The blood shows the ordinary changes similar to those observed in pernicious anæmia. In early cases the color-index may be low, the loss of hæmoglobin being more rapid at first than the red-cell loss. As the case advances, however, the hæmoglobin index rises and may be plus. Megaloblasts are not seen in as large numbers as in other pernicious anæmias. There is no marked leukocytosis; there is, however, a fairly constant relative increase in the eosinophiles, ranging from 3 to 30 per cent.

The feces contain the ova. They also contain considerable blood, in which differential staining will demonstrate a great many eosinophiles, showing that there is not only a general increase in these cells in the circulation, but that there is an active determination of them to the intestinal lesions. Charcot-Leyden crystals are constantly present.

Symptoms.—The symptoms vary with the number of parasites in the intestines and with the general condition of the patient. Recent observations seem to make it clear that the new-world hook-worm is not nearly so fatal as the old-world hook-worm. If there be but a few worms, the general symptoms produced are very mild. If, on the other hand, the worms run up into the hundreds, or thousands, the blood destruction is extensive. In conditions of great deterioration, in the half-starved or ill-nourished, in acute or chronic dysentery the presence of only a few of these parasites may act as a very dangerous complication.

In well-developed cases the symptoms are those of *pernicious anæmia.* The principal phenomena are *dyspeptic symptoms* with *colicky pains* in the early stages, followed by *progressive anæmia* with little or no emaciation, and with *terminal œdema.* The pain in uncinariasis is colicky in character, is one of the earliest symptoms, and is fairly constant throughout the disease. In cases in which only a few parasites are present, it may amount only to uneasiness; when there are many, it may be severe. Like all abdominal pains due to intestinal parasites it is relieved by food for the time being. The appetite is very variable; it may be voracious or it may be diminished, and curious perversions of taste, such as earth-eating, may develop.

Following the development of the colic, *anæmia* appears and rapidly becomes profound. There is very little wasting, the subcutaneous fat being fairly well preserved. The *skin* is a *lemon-white* color; the conjunctivæ and lips are exsanguinated; the scleræ pearly or muddy-white. All the subjective phenomena of profound anæmia become marked. There is *lassitude, breathlessness* on the slightest exertion, vertigo, and occasionally *dimness of vision* from retinal hemorrhages. Crises of fever occur that may last for days or weeks. Auscultation over the præcordial area reveals soft *hæmic bruits,* propagated to a remarkable distance into the great vessels. The *face* and *ankles* become *puffy,* and there may be a slight general œdema. Harris has reported a case of uncinariasis with symptoms resembling pellagra. When the disease attacks children before the age of puberty, bodily and mental growth are stunted. The pubic hair, the axillary hair, and the hair on the face is scanty or absent, the limbs are thin and undeveloped, and the children are markedly pot-bellied. Stiles describes a fish-like, staring expression of the eyes in these cases.

Diagnosis.—The diagnosis is easy, once the attention is directed to the intestinal parasites. The occupation of the patient, if it be one that predicates working in earth, is very suggestive. The anæmia is very commonly diagnosticated as malarial. There really is no diagnostic difficulty between malarial anæmia and the parasitic anæmia. This very common error has been made because, in the intensely malarial regions, the existence of this parasite has not been generally known. The disease has also been mistaken for beriberi, but there is a complete absence of paralytic symptoms in uncinariasis.

A diagnostic sign, recently described, is the occurrence of triangular black or bluish patches on the dorsum of the tongue, looking as though a pen had been wiped on it. This appearance is quite striking. Recent observations have shown that it is very constant; that it appears early, even before the advent of pronounced anæmia, and persists to the end of the disease.

The diagnosis is definitely made by the demonstration of the ova or parasites in the feces. Search should be made in as fresh specimens as possible, to avoid confusion in case embryos have occurred and quitted the ova. A small amount of the material is placed on a large glass slide diluted with distilled water, and pressed down with a thick cover. The ova of uncinaria are very striking bodies. They are clear, transparent, light gray in color; they have a delicate, transparent capsule, containing in its centre from one to six gray yolk segments with granular nuclei. In shape, they are regularly oval, with an average length of 60 microns and an average width of 35 microns. Leichtenstern has found as many as 4,216,930 ova in a single stool. Care must be taken not to mistake the egg of the *Ascaris lumbricoides* for the egg of the uncinaria. The former have a thick, gelatinous, often mammillated covering, and unsegmented protoplasm. So, too, the egg of the *Oxyuris vermicularis,* which is a thin, symmetrical shell, one side of which is almost straight and which contains an embryo, may be mistaken for the ova of the uncinaria. The egg of the whip-worm, *Trichocephalus dispar,* possesses a smooth, thick shell, apparently perforated at each end, with unsegmented protoplasm.

Stiles has recently described a rapid and effective test, where microscopic evidence cannot be obtained. The stool is placed on ordinary white blotting-paper, and allowed to stand for one hour. A rusty-red discoloration or stain develops along the edge of moisture, resembling somewhat that due to the presence of blood and the probable presence of uncinaria. Stiles directs that if uncinariasis is suspected, and it is not practicable either to make a microscopic examination or to delay matters until a specimen can be sent away for examination, still another method of diagnosis is possible. Give a small dose of thymol, followed by Rochelle salts, and collect all of the stools passed. Wash the stools thoroughly several times in a bucket, and examine the sediment for worms about half an inch long, about as thick as a hairpin or hatpin, and with one end curved back to form a hook.

Treatment.—In the treatment of uncinariasis there are two drugs of value: male fern and thymol. Of the two the most effective is thymol or its derivative, thymol urethane (thymol carbonic ether). Thymol should be given in capsule, or in emulsion with acacia, in 30 grain doses repeated in two hours for an adult. In these doses the drug occasionally causes vertigo, excitement, and smoky urine. For one or two days prior to the administration of the remedy patients should be put on liquid diet and given a brisk saline purge the night before. On account of the high location of the parasites in the intestinal tract, it will not be necessary to restrict the quantity of food. In administering thymol, it is essential that none of the solvents of the drug be given either with it or immediately after. Several cases of poisoning have occurred when alcohol or alcoholic drinks have been given with, or closely after, a dose of thymol. Similarly, ether, chloroform, glycerin, and most oils act as solvents, and may cause severe toxic symptoms owing to the absorption of the drug in bulk. In order to prevent poisoning, therefore, thymol should be followed by a saline purge, and castor oil should not be used. Weekly examinations of the stools should be made, and as long as ova or Charcot-Leyden crystals are found the use of this remedy must be repeated. Sometimes thymol is vomited, and in rare cases proves inactive. In these the male fern should be administered in the usual way. After the expulsion of the worm, the therapeutic indications are the same as for advanced anæmia from any other cause.

Filariasis (Filaria Sanguinis Hominis). Definition and History.—The group of *Filariæ* includes a number of species. The human parasite was first discovered by Demarquai in 1863 in a chylocele and demonstrated in the peripheral circulation by Lewis in 1872. The principal varieties affecting man are the following: *Filaria nocturna, Filaria diurna, Filaria perstans, Filaria Demarquaii, Filaria Ozzardi, Filaria Magalhaesi*, and *Filaria loa*. Of this group the *Filaria nocturna* and the *Filaria loa* are the only ones known to cause definite pathological conditions.

The geographical distribution of the *Filaria nocturna* is very extensive. It is found in all tropical and in most subtropical countries. Its southerly limit of observation is Brisbane. In the United States it has been found in Alabama, Louisiana, South Carolina, Pennsylvania, Illinois, and New York. In the Samoan Islands from 10 per cent. to 50 per cent. of the inhabitants are said to be infected, and in the Friendly Islands 32 per cent. In Porto Rico the native troops showed 12 per cent., and small communities may have as high as 70 per cent. of the total population infected.

Filaria Nocturna.—This parasite has two corporeal and one extracorporeal phase. First, the parent worm, whose normal habitat is the lymphatic system; second, the embryo found in the circulating blood, and third, the intermediary stage in the body of the mosquito, which has been definitely shown to be the intermediary host for this parasite. When examined in a drop of peripheral blood the embryonic form is found as a minute transparent worm, one-eightieth of an inch in length and about the diameter of a red blood corpuscle. It has a transparent sheath, or sac, somewhat longer than the body of the embryo, and is usually present in very great numbers.

The most striking characteristic of this filaria is the periodicity of its appearance. They are found only at night in the peripheral circulation, hence the name *Filaria nocturna*. In the daytime they entirely disappear and retire to the larger vessels, particularly the vessels of the lungs. The periodicity may be reversed by causing the patient to sleep in the daytime, when, after a few days, the embryos are found in the peripheral blood during the day and are absent at night.

The microscopic demonstration of the worm is easy. The blood should be drawn near midnight, when the parasites are most numerous. A large drop of blood should be taken and a thick, coarse spread made. The slides are dried in air without covers, or, if it is desired to study the parasite in motion, the blood-drop is covered with a cover-slip and ringed with vaselin. In such a preparation the filaria retains its motility for days. The worms are quite large; an inch objective will be ample for the search and demonstration.

As already stated the mosquito is the intermediary host. When a mosquito sucks the blood of a patient containing filariæ the parasites escape from their sheath while the blood is still in the stomach of the insect. Thence they pass to the thoracic muscles. In this location, in from six to seven days, the metamorphosis from embryo to the young of the adult form takes place. The minute filariæ migrate to the proboscis and are found lying in pairs in the labia, whence they are undoubtedly carried into the circulation of the first warm-blooded animal bitten by the insect. The possibility of the filariæ being passed into water when the mosquito lays her eggs, and being carried thence into the stomach in drinking water, is also to be remembered. Female mosquitoes, both of the culex and anopheles species, are capable of acting as the intermediary host.

The adult form of the parasite, commonly known as the *Filaria Bancrofti*, is a long, delicate, nematode worm. It is from three to four inches in length and the thickness of a coarse hair or bristle. The sexes are distinct. The habitat of the parent worms is the lymphatic system, commonly the thoracic duct, although any portion of the peripheral lymphatics may be invaded. There may be only one worm of each sex or there may be many.

The *Filaria diurna* presents minor differences in structure and appearance from the *Filaria nocturna*. The chief difference is in the periodicity, this parasite being found in the peripheral circulation only in the daytime. It is supposed that the *Filaria diurna* is the embryonic form of the *Filaria loa*. Nothing is known of its pathological significance.

The *Filaria perstans* is common in certain districts of West Africa, where it occurs in as much as 50 per cent. of the population. This parasite is found in the peripheral circulation at all times, day and night; hence its name. The pathological significance of this filaria is unknown. It was for a time supposed to be the cause of sleeping sickness, and was also found very commonly in association with crawcraw, an itching, pustular affection of the skin.

Pathology and Morbid Anatomy.—Filariasis causes no symptoms in the large majority of cases. The embryos are innocuous. When symptoms develop they are due to the parent worm or an immature product of the parent worm. The lesions produced are divided into two broad classes, namely, those which are due to lymphatic varix from stasis, and those due to œdema from lymphatic obstruction. The parent worms, when present in numbers, may mechanically plug the thoracic duct or one of the larger lymphatic trunks. They may also initiate a lymphangitis, with thickening and occlusion of the lymphatics. Under either circumstance, stasis and retrograde movement of the lymph current are inaugurated and eventually a compensatory lymph circulation is established. The result is engorgement of some portion of the lymphatic system and the development of a peculiar group of phenomena: lymph scrotum, lymphatic groin glands, varices of the pelvic or lumbar lymphatic trunks or of the lymphatics of the bladder, ureter, or kidney. As these varicosities grow, they become more extensive, and rupture. If the rupture be in the genito-urinary tracts, chyluria develops; if into the tunica vaginalis, a chylocele; if into the abdominal cavity, chylous ascites. These are the lesions due to lymph stasis and lymphatic varices.

In the second group the lymph stasis is associated with lymphangitis followed by obstruction, resulting in the formation of the peculiar solid œdema and the huge hypertrophies of the affected tissues known as *elephantiasis*.

The majority of cases of elephantiasis are clearly due to filarial disease. The geographical distribution of elephantiasis and filariasis are identical; elephantiasis is most common in the areas most severely affected by filaria. Lymphatic varix and lymph scrotum are not only found in the same districts as elephantiasis, but very commonly terminate in elephantiasis. The disease is also seen in cases of operative removal of lymphatic varices. In a large number of these cases the embryo filariæ cannot be demonstrated in the peripheral circulation. This is due to the fact that the parent female worm lies in the centre of the inflamed tissue and because the embryos cannot pass through the occluded portion of the lymphatic circulation. Manson's theory as to the direct causation of elephantiasis is that the parent female worm migrates to one of the peripheral trunks. While in this position she receives an injury which is followed by premature parturition and the expulsion of ova instead of embryos. These ova are five times the diameter of the embryos, so that although the embryos pass freely through the finer lymphatic radicles the ova block up the smaller lymph channels, forming minute emboli.

Following this stage of embolism, lymphangitis with inflammatory thickening occurs. When the inflammatory process subsides the deposit is in small part absorbed, but when the inflammation recurs an additional deposit is added to the remnant of the first. This concurs with the commonly observed clinical history of elephantiasis, which advances by crises of inflammation and a general symptom-complex known as elephantoid fever.

As already stated, filariasis may occur without any symptoms being present until the lesions just described develop.

Symptoms. *Elephantiasis.*—The most common location of elephantiasis is in the legs; next in the scrotum of the male and the labia of the female. It occurs in the breast, in the arms, and as interstitial or pedunculated masses in other parts of the body. These tumors occasionally grow to enormous dimensions. A scrotal tumor of 50 pounds is not at all uncommon, and one has been reported weighing 224 pounds. These large tumors develop as described with elephantoid fever. With each crisis of fever and lymphangitis there is not only an increase in the size and bulk already affected, but an extension into new territory.

Elephantoid fever is the systemic expression of the lymphangitis originating in filarial varices or in tissues already the seat of elephantiasis. The attacks recur at varying intervals of weeks or months. Exciting causes may be slight traumatism such as friction of the clothing over the occluded lymph channels. The attack begins with *rigor* and *high fever*, with all the usual manifestations of febrile disturbance, as *anorexia*, *nausea*, and, in severe cases, *delirium*. The *skin* over the inflamed lymphatic area is *hot*, *tense*, and *red*, with a marked degree of inflammatory thickening. After persisting a few days, the attack ends in a *critical sweat*. The inflammatory thickening remains. The attack may be so intense as to result in the formation of abscess. When this takes place in superficial lesions, the condition is readily recognized and the treatment is obvious. When it occurs in the deeper varices, as in the pelvis or loin, there will be deep-seated pain, tenderness, and rapidly developing sepsis.

Hæmatochyluria is usually paroxysmal and is due to the leakage of chyle into some portion of the genito-urinary tract. The common location of the leak is in the lymphatics surrounding the pelvis of the kidneys or the lymphatics of the bladder. The appearance of chyle in the urine is intermittent and is not accompanied by any increase of symptoms. The urine is opaque and varies in color from a milky-white to a deep red in proportion to the amount of blood that may be mixed with the chyle. Chylous urine coagulates on standing into a soft, jelly-like clot. In a few hours the clot contracts and forms a firm, white ball floating in a milky fluid. Microscopically, the urine contains fat, blood cells in varying amount, and occasionally embryo filaria. Chyluria may persist for many years without great deterioration of health. The only symptoms may be a dull pain in the loins or pelvis. Osler reports a case of intermittent chyluria which persisted for eighteen years without any special discomfort. When chyle escapes in large quantities, clots are occasionally formed in the bladder and may cause *urinary retention*. Medical treatment is of no avail in this condition, but the patient should be put at rest on dry diet, with as great restriction as possible in the fatty elements of his food. Under this regimen the amou notably diminished.

Varicose groin glands is a varicose condition of the inguinal lymphatics, and it is usually bilateral. It gi soft, painless swellings in both groins. They cause lymphangitis develops, when they may become very been mistaken for the buboes of plague, and are com hernia.

Lymph Scrotum.—In this condition the filarial vari lymphatics of the scrotum and is usually bilateral. It with the enlargement of the groin glands, and, like that accessions of lymphangitis. When the deeper lymphati lymphangitis may extend to the testicle, forming what orchitis.

Treatment.—The treatment of these conditions is s tumors become so large as to be a burden to the patient, matory symptoms cause great pain, their removal sho The removal of an enormous scrotum or the amputatio may be done to rid the patient of a drag that has, by its sh him to his bed or chair for years. These growths ma comparatively little risk. All surgical treatment in fi be regarded as palliative unless the parent female wor excised tissues, when the cure will be a definite one.

As far as any plan of treatment aimed at the destruct adult or embryo, is concerned, there is no remedy o value. During the attacks of filarial or elephantoid fev should be elevated, cooling lotions should be applied, an purged. After the acute symptoms subside elastic b tried, and frequently give great relief.

In countries where filarial diseases are common segr cable, but the danger of dissemination would be greatly harboring the parasites would so live as to avoid mo cautions might be advisable for sporadic cases in countri is only occasionally introduced.

Guinea-worm Disease (Dracontiasis). Definition. sometimes called *Dracunculus medinensis*, or Medina

Distribution.—This parasite is distributed throughout known in medicine long before the Christian era, and t for believing that the Biblical mention of the serpents aff near the Red Sea were these parasites. Though very the infection occurs in sharply defined areas. It exists Africa, particularly on the Gold Coast, in Abyssinia, S the Soudan. In Asia it occurs along the Caspian Sea, and in some sections of British India. It was common Indies and Tropical America during the days of the sla to have established a permanent hold only in a few Sout and in isolated spots in Brazil. In the United States this tered from time to time in imported cases, although at le been recorded in persons who had never lived in or visited tropical regions. Thus, Van Harlingen reports it as occurring in a case of a man who had never lived outside of Philadelphia; Jarvis, in the case of a man who lived at Fortress Monroe, Va., for thirty years; and Walker, in a case from Georgia.

The *Dracunculus medinensis* is a nematode worm. We have definite knowledge of the female only. The adult female worm is cylindrical in form, from 60 to 80 cm. in average length and about 2 mm. in thickness. The head of the worm is blunt, with a triangular mouth and eight papillæ. The tail is tapered and recurvated ventrally. The vascular tube and alimentary canal extend as straight canals the length of the worm, ending as blind pouches.

Charles found in the mesentery of a cadaver two female guinea worms in conjugation with two smaller worms. These worms were about 4 cm. long and attached to the females, which were quite small, about 14 cm. from the head end. It is assumed that these were males. In the adult female the uterus occupies almost the entire body and is filled with myriads of tightly-coiled embryos averaging 0.6 mm. in length.

Fedschenko and Manson have shown that the intermediate host of the guinea worm is a minute water flea (*Cyclops quadricornis*). The recently escaped embryos penetrate the cyclops, in which they pass through one stage of larval development lasting about six weeks.

It is assumed that infection occurs through swallowing the cyclops itself or the larva in drinking water. While this intermediate stage in cyclops is the usual history, Plehn has shown, experimentally, in monkeys, that direct infection by the embryos may also take place.

Negroes and the laboring classes are more frequently attacked than others, and usually, in each small area, the infected well or spring may be identified. The greater number of cases occur at the close of the heavy rains, probably because these conditions are then more favorable to the development of cyclops.

The worm is taken into the body through the stomach. The males and females probably pass through the intestine to the mesentery, where conjugation takes place. The male dies and becomes calcified or absorbed and the female migrates into the connective tissue of the host. In these migrations she usually tends toward the lower extremity and appears in the foot or the leg, although she may appear in the subcutaneous tissues of the trunk, arm, or even the head. When the subcutaneous tissue is reached complete development takes place, and when the embryos are ready for expulsion a small boil or vesicle forms, which bursts and leaves a small sinus leading down to the head of the worm. The period of incubation from the ingestion of the embryo to the appearance of the adult embryo-bearing female in the subcutaneous tissue is about one year. The migrations of the worm are not attended by pain or any other symptoms.

Symptoms.—At the time of development of the vesicle or boil there may be some slight febrile disturbance, and there is slight pain from the local irritation and inflammation. The rupture of the vesicle leaves a flattened, shallow ulcer, at the bottom of which is a small opening. At this opening the head of the worm may appear. If the ulcer be douched or sprayed with cold water a small quantity of a milky fluid exudes from the orifice, or the uterus of the worm may be protruded as a delicate tube which is seen to fill up and suddenly empty itself of a few drops of milky fluid which, examined microscopically, contains myriads of embryos. Usually when parturition is completed, or nearly so, the worm spontaneously leaves her host. In a case reported by Francis, in which five worms were observed in the feet, one complete worm containing its embryos and measuring twenty-six inches was passed in about half an hour. Usually a much longer period is required (fifteen or twenty days) before the worm emerges. Exceptionally, parturition being completed, the worm dies, becomes encysted, and can be felt as a firm, fibrous cord under the skin.

Treatment.—The older method, and the popular one with the natives, is to grasp the presenting head of the worm, fix it to a smooth stick, and gradually wind her out by twisting out a few inches every day. By this method the worm may easily be torn and a swarm of embryos liberated in the subcutaneous tissues. In the case reported by Francis, a temperature, with morning and evening variation between 98.8° and 104.5° lasting several days, occurred after rupture and retraction of a worm. These accidents have occasioned severe infections, resulting in death. Manson advises douching with cold water, application of a cold pack or cold baths to hasten expulsion of the embryos and the spontaneous emergence of the worm. Massage and electricity have been used with success. The best methods of treatment we owe to the suggestion of Emily. He advises injection of 0.1 per cent solution of mercuric chloride into the head of the worm or into the swelling. This solution causes death of the worm, which may then be easily extracted. Similarly, Aoulkes advises injection of alcohol, and Tufnel, pure carbolic acid.

Prophylaxis consists in careful filtration or sterilization of drinking water.

Strongyloides Intestinalis. **Definition.**—*Strongyloides stercoralis* is a nematode worm infecting the intestinal canal. When present in large numbers it causes a chronic diarrhœa, with anæmia and emaciation.

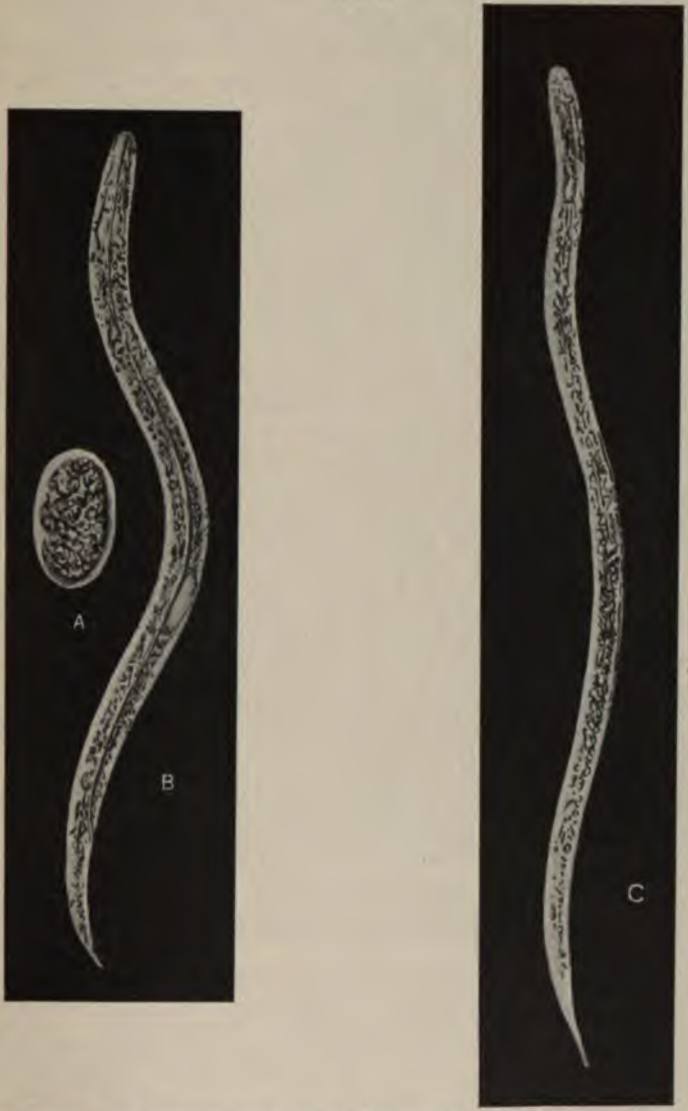
Distribution.—This parasite is widely distributed throughout the tropical and subtropical countries. It is extremely common in Cochin China, where it is supposed to be the cause of the severe diarrhœa of that country known as Cochin China diarrhœa. Powell has found it in India, in 15 out of 20 cases of anæmia. It has been observed in Martinique, Sicily, Egypt, India, Porto Rico, and the Philippine Islands. In Italy, Germany, Brazil, and California it has been frequently observed in association with uncinaria. It was first reported in the United States by Thayer, and is now known to be fairly common in all our Southern States.

Much confusion has arisen over the various nematode worms resembling this parasite. These are now believed by the majority of observers to represent morphological variations of the same worm. The following classification is given by M. L. Price:

1. The rhabditiform embryo, formerly known as *Anguillula stercoralis*. found in the fresh stools, is a slender, active nematode worm, 0.3 mm. long and 0.04 mm. broad.
2. Filariform embryo found in the stools after standing one or two days,

and supposed to develop from the rhabditiform embryos. This embryo is twice as long as the preceding form, and is also actively motile.

Fig. 114



A. Egg of Cochin-China diarrhœa worm (Strongyloides stercoralis) found in stools. B. Rhabditiform embryo of same, from the stools. C. Filariform larva of same derived, by direct transformation, from a rhabditiform embryo. The figures were drawn from life, as seen under Leitz, objective 7, ocular 3. Bulletin of the United States Marine Hospital Service, No. 10, 1903. (After Thayer.)

3. The sexually differentiated form, *Rhabditis stercoralis*, which may be developed from the preceding in five days. The male is a fine nematode

worm 0.7 mm. in length, the female 1 mm. in length, wh
extracorporeally, produces filariform embryos. Finally

4. The parthenogenetic mother-worm, *Anguillula inte*
intestinal canal at autopsy. A slender worm, 2 mm. in
nized by the string of five or six eggs in the centre of
probably takes place by the ingestion of the filariform
on uncooked vegetables. Leichtenstern has experin
incubation period to be seventeen days.

Later researches have disproved much of the path
previously attached to this parasite. It is a mistake,
the worm has no clinical significance. The principal sym
diarrhœa, without pain or temperature disturbance. S
indigestion develops and *nutrition* is very much *lowered.*
there is *anæmia* and *wasting*. Blood examination shows
and, in marked contrast with other verminous anæ
leukocytosis nor eosinophilia.

Treatment.—Thymol, given fasting, in the same ma
riasis, is the best remedy for expulsion of strongyloides.
thymol should fail or if it should be rejected, or the patie
ance to the drug, male fern should be used in large dos

Trichocephalus Dispar.—The *Trichocephalus dispa*
a "whip-worm," is occasionally found in the intestinal
male and female worms are about equal in size.
coiled in a spiral form, but the female is nearly strai
portion of the body is thicker than the anterior par
anterior filament the parasite embeds itself in the muco
intestine. This parasite is not common in the United Sta
observed in France and Southern Italy. Its chief area
the cæcum, and more than one worm is usually prese
large numbers are found. It is supposed to possess no
cance and to be incapable of producing serious sympto
have claimed that it may cause diarrhœa and anæmia of
We know little about its history of development.

CESTODES OR TAPEWORMS.

Tapeworms are very frequently found in the intest
As their name indicates, they are flat, broad, white par
of segments, each of which is rectangular in shape, but s
Each of these segments represents a single individual.
segments just named develop. The technical name for
is the "scolex," and for the segment "proglottis." By
the worm is attached to the mucous membrane of the
is no mouth in the sense that an opening exists which
an intestinal canal. Each segment of the worm is her
to say, each segment contains male and female organs

There are several varieties of tapeworms. The mo are the *Tænia mediocanellata*, sometimes called the *Tænia saginata*, or unarmed tapeworm, or beef-worm, the *Tænia solium* (pork-worm), and the *Tænia echinococcus*. Less common forms are the *Dibothriocephalus latus*, or Russian tapeworm, derived from eating infected fish; the *Tænia nana*, the *Tænia confusa*, and the "double-pored dog tapeworm," *Dipylidium caninum*. The *Tænia nana* is sometimes called the *Hymenolepis nana*, or *dwarf tapeworm*.

The *Tænia solium* usually gains its entrance into the intestinal canal of man by the ingestion of imperfectly cooked pork, the *Tænia mediocanellata* by the eating of uncooked beef, and the *Tænia echinococcus* by the ingestion of food which has been fouled by the excrement of the dog.

All tapeworms pass through three stages of existence. The segments of the worm give off eggs which are discharged from the intestinal canal of the host, enter the alimentary canal of some animal, and are hatched out as parasites which pass through the wall of the intestine, gain a place of rest in the muscles or other tissues, and there form cysts. When these muscular tissues are eaten, the parasite in the cyst once more enters the alimentary canal, becomes attached to its mucous membrane, and from it is developed the adult worm.

The *Tænia solium* may be several yards in length. At one time it was thought to be solitary; hence its name. It not infrequently happens, however, that more than one worm is present. The head, which is very small, scarcely larger than a pinhead, has a proboscis, or rostellum, about which is arranged a double row of horny hooklets. The hooklets in the anterior row are larger than those in the posterior row. Below these are four sucking disks at the sides of the head. By these means the worm attaches itself to the bowel. The segments of the worm are about 10 to 12 mm. in length and from 5 to 60 mm. wide, but they vary considerably in size; those nearest the neck of the worm being shorter and narrower than those which develop several feet away from the neck. When the egg of the *Tænia solium* is hatched out so that a scolex (or head) is set free, and this parasite becomes encysted in the muscles of a pig, the pork is said to be "measly." When it finds a resting place in the muscles of the brain, or other parts of the human being, it is known as the *Cysticercus cellulosa*. These cysts vary in size from that of a small pea to that of a bantam's egg, and are separated from the surrounding tissues by a formation of connective tissue which acts as a capsule.

The *Tænia mediocanellata* possesses a head which differs materially from the head of the *Tænia solium*. There is no rostellum or hooklets, but there are four sucking disks, which are much nearer the point of the head than they are in the *Tænia solium*. This worm further differs from the *Tænia solium* in addition in that its segments are generally broader and shorter, and the entire worm is usually much longer.

This worm may reach the length of about 25 or 30 feet, and it is not very rare for from 15 to 20 feet of a worm to be passed intact. When the scolex of this worm is found in the muscles of cattle it is called the *Cysticercus mediocanellata*.

In Germany, where imperfectly cooked pork is largely eaten, the *Tænia* *solium* is most frequently met with, but in this country, where the people eat largely of beef, the *Tænia mediocanellata* is much more common.

The *Tænia echinococcus* is very rarely met with in the United States. It is, however, exceedingly common in Australia. This worm possesses a double row of hooklets and four sucking disks. It is rare for more than three or four segments to be attached to any one head, but as the parasite is often present in numbers many disconnected segments may be discharged. Like the other forms of tapeworm, the segments increase in size as the distance from the head is increased. This worm does not inhabit the intestine of man, but produces its evil influence by reason of the entrance of its eggs into his alimentary canal, from which place they wander into other parts of the body, forming what are known as hydatid cysts. In other words, the infection of human beings by the *Tænia mediocanellata* and the *Tænia solium* is quite different from the infection of human beings by the *Tænia echinococcus*, for in the first cases the patient swallows the parasite when it has reached the second stage of its existence and is prepared to develop its segments; whereas, in the case of the *Tænia echinococcus* the patient takes food which has in some way become contaminated by the fecal discharges of the dog, which fecal discharges contain the eggs of the parasite, and from these eggs are developed cysts. A patient infected by the *Tænia echinococcus* therefore suffers from the cystic stage of development of the worm.

The hydatid cysts formed in this manner most frequently infest the liver, but almost any portion of the body may be affected. Such cysts, in the liver in particular, are always surrounded by a layer of connective tissue which is thrown out in an endeavor to circumscribe the invading parasite. The wall of hydatid cysts, therefore, is formed of two layers; the outside layer is lamellated and is sometimes called the cuticula. The inner wall of the cyst often contains muscular fibres and bloodvessels, and is called the parenchymatous layer. Not rarely the primary cysts give rise to secondary cysts called daughter-cysts, and these daughter-cysts may develop in themselves cysts which are called granddaughter-cysts.

On the inner surface of these cysts the scolices, or heads, of the worm are formed. At the posterior end of the scolex is a stem, or pedicle, by which it is attached to the wall of the brood capsule. In some instances the scolex may be found free inside of this capsule. In most cases, after the cyst has existed for a long period of time, the scolices die, the fluid is absorbed, and a granular mass remains. This granular mass may contain the hooklets, or the hooklets may be found free in the contents of the capsules, or in the primary cyst itself. Occasionally, a hydatid cyst is found which is sterile, that is, in which neither sub-cysts nor scolices are developed.

In addition to the scolices, the cysts contain a clear, limpid fluid which sometimes becomes turbid after the cyst has existed for a considerable period of time, the turbidity being due to disintegration of the lining layer of the cysts and the formation of crystals of cholesterin, and to the presence of lime-salts. Occasionally the cyst shrinks, its contents become inspissated or thickened, and the entire mass, including the connective tissue which has been formed around the cyst, may become calcified. Sometimes, too, the daughter-cysts instead of growing inside grow outside. Indeed, this variation is more commonly seen in man than it is in animals that are affected by this parasite. This is called the *echinococcus exogena.*

Under the name of *echinococcus multilocularis* a variety of echinococcus cyst is found in the liver, which is characterized by a somewhat irregular distribution of groups of small cysts walled off by connective tissue, as are the larger cysts already described. These cysts are often sterile; that is to say, they do not contain scolices or hooklets. It is probable that this formation is due to a somewhat different parasite from the ordinary *Tænia echinococcus.*

The *Bothriocephalus latus,* or *Dibothriocephalus latus,* is the largest of all human tapeworms, and has very broad, square segments. The head is egg-shaped, but possesses no disks or hooklets. On the contrary, its head is marked by long grooves by which it attaches itself to the intestine. Its neck is longer and more slender than that of other tapeworms. Two species have been described, the *Bothriocephalus cordatus* and the *Bothriocephalus cristatus.* Infection by this parasite occurs most frequently by the eating of imperfectly cooked fish, probably because the eggs develop to some extent in water and are swallowed by various fish, in whose flesh the cysts are formed just as the other scolices already described form in the flesh of the hog or ox.

Tænia nana, or dwarf tapeworm, *Hymenolepis,* is only from one-fifth to two inches in length. It has four suckers and a single row of hooklets on its head. Stiles states that it may be present singly or by thousands, and is probably more frequent than is generally thought. Its intermediate host is usually the rat, from the stools of which food is infected. In the rat the cyst stage may occur in the intestinal wall and is called a *cercocystis.* Like other tapeworms, the embryos burrow into the wall of the intestine, but do not remain there, falling back into the lumen of the bowel to reach adult development with eggs in about fifteen days. The only tæniafuge which has proved effective for the removal of this worm is aspidium.

The *Tænia cucumerina* is slightly larger than the *Tænia nana,* and its head possesses four rows of hooklets. It is not infrequently found in the ileum of dogs and cats, but rarely affects man. Its scolices inhabit the dog-louse and by means of this parasite, or by the carrying of the embryos to the mouth by the hands after handling a dog or cat, infection of a human being may take place.

Symptoms.—The symptoms produced by the presence of tapeworms in the alimentary canal are not pathognomonic. Not infrequently the worms exist for a long period of time without their host having any knowledge of their presence, and the infection is only discovered by the chance observation of one or more segments in a stool. The patient may suffer from symptoms of *gastrointestinal catarrh* produced by the irritation caused by the worm, and sometimes an *inordinate appetite* is present, but this is by no means as constant a symptom of tapeworm as most persons imagine. Not infrequently, the host of a tapeworm suffers from *anorexia* rather than

from excessive hunger. In children there may be a gc
irritation and *peevishness.*

In some instances, however, the presence of a tapewo
much more serious train of symptoms, which consist in
that may be so severe as to give rise to the suspicion that
is present. The *Bothriocephalus latus* is said to be mo
grave anæmia than any other of the tapeworms.

Treatment.—The treatment of a patient infected by
in the abstinence of all food for eighteen hours prior to
of one of the following drugs, which are known to poss
paralyzing or killing the worm that it lets go its hold,
passed from the bowel under the influence of a purge.
drachm of the oleoresin of aspidium may be given in c
to an adult, and followed in four or five hours by a sal
as citrate or sulphate of magnesium or Rochelle salts.
pelletierine given in the dose of 3 to 5 grains may be u
conditions. It is commonly given in syrupy solution, and
is put up in a small container which holds one dose. I
tierine may be followed by castor oil in place of the othe
but castor oil must not be used after aspidium is give
absorption of the drug into the body and so tends to po
A less agreeable method of destroying the worm is to adn
made of pepo, or pumpkin seeds which have been dep
coverings by the process of bruising. Several drachms
without doubt, very efficacious. The patient should al
to pass the stool through a sieve and not to seek so mu
of the worm as for the small head. The mere passage
of feet of segments does not indicate in any way that th
nently relieved unless the head, from which other segm
remains in the bowel, is also passed.

TREMATODES.

Definition.—A large number of worms belonging to t
as parasites in the body of man or of the lower animals
is so infested the condition is said to be one of *Dist*
arising from the fact that the word Distoma is oftentim
parasites.

Up to the present time no less than thirteen species
Flukes have been described as occurring in human being
belong to the family of the *Fasciolidæ,* one to the family
one to the family *Schistosomidæ.*

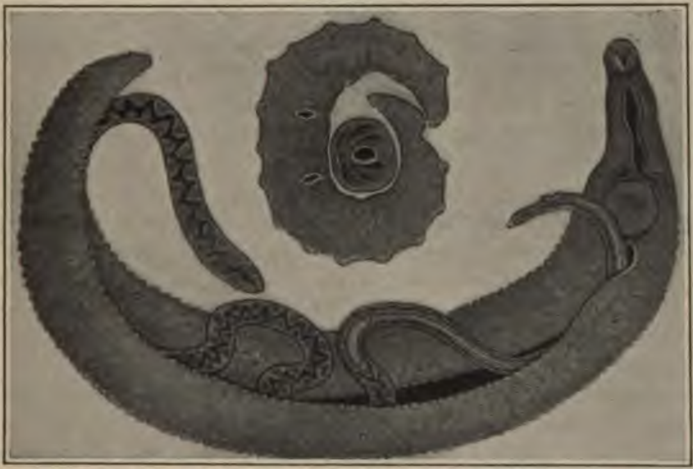
When the human being is attacked the parasite is us
genito-urinary tract, when it causes what is known as *B*
Endemic Hæmaturia. Less commonly it infests the lun
is then called *Distomatosis of the Lung, Endemic* or *Pa*
or Lung Fluke; and it is also met with in the liver, for

Liver Fluke or *Distomatosis of the Liver.* The fluke found in the genito-urinary tract is the *Schistosoma hæmatobium*, that met with in the lung is the *Paragonimus Westermanni*, sometimes called the *Distoma Ringeri* or *Distoma pulmonale*, and that discovered in the liver the *Fasciola hepatica* or instead *Dicrocoelium lanceatum*, *Opisthorchis sinensis*, the *Opisthorchis felineus*, and the *Opisthorchis noverca*.

Bilharzia Disease.—Bilharzia disease, or endemic hæmaturia, is due to the development in the body of the *Schistosoma hæmatobium*, and is characterized by hæmaturia and the formation of papillomatous tumors in the genito-urinary tract.

Etiology.—The male worm is about 4 to 15 mm. in length and 0.6 mm. in breadth. The female averages 15 to 20 mm. in length and 0.28 mm. in breadth. The male has flattened sides, rolled up on both edges so as to form a deep groove, the gynecophoric canal, in which the female lies during conjugation (Fig. 115).

FIG. 115



Male bilharzia worm carrying the female, showing the papillæ on his skin. The small figure is a cross-section showing relative position of the sexes. (Looss.)

History and Distribution.—Endemic hæmaturia has been observed in Egypt for centuries. At the present day it is said to be present in fully one-half of the population. According to Looss it is equally frequent in Uganda. It is practically limited to the African continent, although cases have been reported from Cyprus and Sicily, and Manson reports a case from the West Indies. It occurs frequently in British India, but always as an imported infection. A few cases have been reported in the United States. The parasitic nature of the disease was discovered in 1851 by Bilharz.

The ova of these worms are found in very great numbers in the urine. They are oval, and have a marked terminal spine and contain a ciliated embryo. It is supposed that the spine is the organ by means of which the embryo bores through the peripheral tissues. Ova with lateral implantation of the spine are frequently observed. Looss supposes these are examples of faulty development, and that the faulty position of the spine, limiting the mobility of the ovum, is the reason many more of this form are found in sections than free. In urine or in water the embryos very soon escape from the ovum and move about very actively by means of their cilia. In undiluted urine they die when it cools. In water they remain active for a long time.

The embryos are probably taken into the stomach in drinking-water, penetrate the gastric or intestinal wall, and they develop into mature worms. Looss surmises and brings some evidence to show that, like the uncinaria, this parasite may also penetrate the skin.

Pathology.—The affected bladder is covered with a bloody, tenacious, mucous layer; the submucosa is greatly thickened; the muscular and serous coats, as a rule, are unchanged. In older cases papillomata are found, varying in size from a small pea to large tumors filling the entire bladder. Microscopically the changes consist in marked degeneration of the epithelial layers, going on to complete destruction of the mucosa. The pseudomembranous covering then consists of ova, leukocytes, and urinary salts. In the submucosa enormous masses of ova are found, many of them calcified. The papillomatous tumors spring from this layer, and are very similar in their histological structure to nasal polypi. Similar changes occur in the rectum, urethra, ureter, seminal vesicles, prostate, and uterus. Secondarily these lesions produce stricture, urinary fistulæ, pyelitis, prostatitis, and urethritis.

The papillomatous tumors show some tendency to undergo malignant change, but by far the commoner complication of the disease is stone formation. In old cases a beginning deposit of lime-salts is found in the mucus covering the bladder wall, as small calculi embedded in the folds and loculi. In other cases large, free calculi are found with clumped masses of calcified ova as their nucleus.

Tumors and masses of ova are sometimes found in the pelvis of the kidney, rarely in its parenchyma.

Symptoms.—The symptoms vary with the intensity of the infection, the number of adult worms, and the location and extent of the lesions already described. The only constant symptom is *hæmaturia.* The amount of blood present in the urine may be so small as only to be evident on microscopic examination, or so large as to form clots of appreciable size in the bladder. As a rule, the blood is passed at the end of urination. The microscopic examination, in doubtful cases, should therefore be directed to the last few drops of urine expelled. In the large majority of cases hæmaturia will be the only symptom. In the severe infections, however, *cystitis* usually develops and becomes very troublesome. Following the development of inflammation the ordinary symptoms of *tumor* or *stone* develop. In severe cases, with diminished resistance, *suppuration* of these lesions occurs, with formation of extensive *fistulous tracts.* When the lesions are confined to or are most marked in the bowel, the early symptoms may resemble acute dysentery and, in older cases, chronic dysentery, with pain, tenesmus, and bloody and mucous stools. When tumors occur in the bowel they are readily recognized, although when situated low in the rectum they have been mistaken for hemorrhoids.

The urine contains red blood cells, leukocytes, principally eosinophiles and polynuclear cells, besides large numbers of ova. With these the ordinary evidences of an extensive chronic cystitis are also found. The number of ova present varies very widely and bears no relation to the amount of blood in the urine. When they are very few in number they may only be found in the last few drops of urine passed.

The blood changes, in severe cases, are marked. There is a pronounced fall in the number of red cells and a still greater reduction of hæmoglobin. With this there is a moderate degree of leukocytosis, the increase consisting almost entirely of eosinophile cells, which are present in proportion varying from 9 per cent. to as high as 52 per cent.

Diagnosis.—The blood condition pointing to a toxic or parasitic anæmia, with the demonstration of the ova in the urine, make the diagnosis of Bilharzia disease.

Prognosis depends on several factors, namely, the extent of the infection, the number of adult worms present, and, more important still, the conditions favoring reinfection. When all opportunity for reinfection is avoided, as by removal from the endemic area, after a time the adult worms die, and eventually all the ova are evacuated. This process may be a very long one. In some observed cases it has extended up to eight years. The prognosis also depends on the character and extent of the surgical complications and sequelæ.

Treatment.—There is no treatment that will influence either the worm or the ova in the slightest degree. All the anthelmintics have been tried and found useless. Similarly, local application of antiseptics and protoplasmic poisons to the bladder have failed. In most cases the hæmaturia does not require treatment. When it becomes severe, rest in bed should be enjoined. Cystitis should be treated on general lines by local medication, as well as the internal administration of urotropin, salol, benzoic acid, and remedies of this group. The complications of the disease, such as stricture, extensive tumors of the bladder and rectum, accessible ulcerations of the vagina or cervix, and prostatic involvement, call for appropriate surgical measures. In a general way, all the conditions which predispose to or aggravate cystitis should be avoided. These are exposure, chill, violent muscular effort, alcoholic debauches, spices, and highly seasoned food. With a view to obtaining an eventual cure, patients should if possible be removed from the endemic area. When this is not possible, proper means should be taken to ensure a good water supply. In this manner the constant reinfection of the patient is avoided. Similarly, in view of the possibility of infection through the skin, sound shoes should be insisted on and work in alluvial oozes should be avoided.

Distomatosis of the Lung (*Lung Flukes; Endemic or Parasitic Hæmoptysis*).—The lung fluke (*Paragonimus Westermanni*) is widely distributed in Japan, Formosa, Corea, and North China. It has been carried by Oriental emigrants to many other countries. Isolated cases have been reported from the United States and Mexico.

The parasite is a small, fleshy, trematode worm o
20 mm. long and 6 mm. in its transverse diam
found in the lungs, but has been observed in ot
the liver and brain. The worms discharge a vast nu
ova are dark brown, oval, 0.08 mm. long by 0.05 mm.
operculum, and contain a ciliated embryo. They are fo
in the sputum. Infection probably takes place through
although nothing is known of the extracorporeal pha
By far the larger percentage of cases is observed in youn
is supposed to predispose to the disease.

Pathology.—Patches are scattered all over the lung
the periphery, resembling hemorrhagic infarcts. On
are found to be infiltrated and honeycombed with
cavities, each of which contains one or more distoma
Occasionally large cavities are formed by coalesc
lesions.

In cases in which the parasite invades the brain, ana
found, but they are almost entirely limited to the corti

Symptoms.—The commonest symptom is chronic m
rusty, prune-juice, or bloody sputum. The amount of
may be so small as to be only demonstrable by the micr
be periodical and severe hemorrhages from the lungs.
the sputum is due not only to the blood and the blood
in it, but also to the large numbers of dark-brown ov
contains eosinophile cells, Charcot-Leyden crystals, an
course of the disease is essentially chronic. Cases las
years without much discomfort and without much
general health, excepting where marked secondary a
repeated and severe hemorrhages.

Prognosis.—The prognosis is good, excepting in t
which hemorrhage is sufficiently severe to cause a fatal
parasite attacks the brain, epileptic symptoms have be
prognosis is grave.

Treatment.—A large numer of drugs have been adm
suffering from this condition, both by the mouth
in the hope that benefit might accrue. It is eviden
the lesions renders any therapeutic measure of little v

Distomatosis of the Liver (*Liver Flukes*).—Liver
ically in certain sections of Japan. For instance, Ba
per cent. of the inhabitants of Okayama Province are i
fluke. Inouye found in various sections from 19 per c
of the population infected. Infection has been carried
Oriental emigrants. In the United States an entirely a
seen in cats and cattle and several cases have been me

The parasite commonly invades the biliary tract or
and is also found in the duodenum, the stomach, and s
tion of the biliary ducts by the parasites causes dil
catarrh. There is also overgrowth of the hepatic co

atrophy of the parenchyma. Small but constant hemorrhage from the biliary passage may cause a grave anæmia.

Postmortem the parasites are found in great numbers in the walls of the gall-bladder and biliary ducts, or free in the ducts. They lie in small, cyst-like cavities connecting with the gall-bladder or ducts.

Symptoms.—The symptoms depend on the number of worms. The first symptom is rapid enlargement of the liver, with voracious appetite. The liver may reach to the umbilicus, it is tender on palpation, and there are recurring attacks of jaundice. Sooner or later diarrhœa begins, and with it marked failure of nutrition. The patient becomes weak, emaciated, and anæmic. The diarrhœa in marked cases is severe, and the movements contain much blood besides the ova of the parasites. Later, dropsy of the legs and belly develop, and the patient dies exhausted. The course of the disease is very chronic, and likewise depends on the number of parasites. Recovery never takes place.

Treatment.—There is no treatment save the use of stimulants and good food.

With the idea of prophylaxis Inouye advises against drinking or swimming in canal water or eating raw fish or mussels. He states that in one region notably infected with the disease the mortality from distomiasis has been reduced to zero by following these simple precautions.

PARASITIC INFUSORIA.

The parasitic infusoria which are found in man are protozoa of the subclass flagellata. They are rarely met with. The *Plagiomonas urinaria* has been found in the urine of a man who suffered from chronic suppuration. The *Trichomonas vaginalis* is found in acid vaginal mucus, and the *Trichomonas hominis* has been found in the bowels and stools. They all possess but little clinical interest.

DHOBIE ITCH.

Definition.—Dhobie itch is a term applied to a large group of mycotic and bacterial, itching skin diseases affecting persons living in hot climates. These eruptions may occur anywhere on the body, but usually in the crotch, axilla, on the soles of the feet, and between the toes. The term dhobie is derived from the "dhobie" or washerman, owing to the widespread belief that the contagion is conveyed by the clothing passing through his hands.

Etiology.—Epiphytic skin diseases are exceedingly common in the tropics, and all forms of ringworm grow with the greatest freedom under the exceedingly favorable conditions of heat and moisture afforded by the climate. Three types of the disease are recognized. The first is thought to be due to the *Microsporon minutissimum;* the second type probably represents a very extensive group of different varieties of *tricophyton*, or ringworms. The third type, classed as dhobie itch, is that described by Manson as

Pemphigus contagiosus, in which he has isolated a diplococcus, the exact significance of which has not yet been fully determined.

These skin diseases have all been imported into the United States by soldiers returning from the Philippines, and there is every possibility of spreading them in the States that lie in the subtropical belt. Tropical skin infections of this type are usually spread by direct contact with infected persons or their clothing. In many instances sexual intercourse serves as a means of spreading the disease. Bathing in the waters of sluggish streams and tanks sometimes causes dhobie itch. The natives of Luzon believe that sea bathing is one of the common causes, and among men who habitually bathed in the sea I (Kieffer) saw a larger proportion of itch cases than in those who did not. The washing of underclothing in cold water, and without boiling, must also be considered a cause. Clothing washed in cold water invariably becomes overgrown with moulds when laid aside for a day or two. These mouldy clothes frequently cause ringworm infection. Ringworms are also very common among the domestic animals in the tropics, and are undoubtedly conveyed from them to man.

In the two mycotic forms the disease usually attacks the crotch and axilla. The appearance of the lesion is that of an ordinary ringworm with festooned, scaly margins. The interior of the patch is red, glossy, and bare. Secondary infections frequently occur from scratching or chafing, or from simple contact with the denuded epithelium. These diseases affect almost all ages, and are found in the native as well as in the foreigner. In the Philippines very few Americans escape without contracting one or another of them. The smarting and itching are very severe. The suffering from the chafing of the clothing may be so great as to entirely prevent the patient from moving about. Unless vigorously treated the disease persists until the cool season, when the patch heals up, leaving a scaly, pigmented area which breaks out again with the approach of the hot weather.

Pemphigus contagiosus occurs in the form of vesicles, or blebs, which break and leave a raw, red, and shiny, denuded surface, with a sharp, clear-cut zone one-sixteenth to one-eighth inch in width of undermined epithelium surrounding them. When the blebs are small its resemblance to varicella may be very close. The disease occurs in foreigners of all ages. Among the natives adults are as a rule immune, and it is principally seen in children. The eruption may be scattered over the entire body or may be limited to the crotch and axilla.

When any doubt exists of the nature of these eruptions, the mycotic elements can be readily demonstrated microscopically. Pemphigus contagiosus is to be differentiated from varicella in that the constitutional symptoms of the latter are wholly lacking.

Treatment.—The parts should be thoroughly cleaned with green soap, following which a parasiticide remedy should be used. Manson advises Vleminck's solution of the sulphuret of calcium, applied every night for three or four times. Tincture of iodine painted over the area daily is a valuable remedy. Similarly, an ointment of chrysophanic acid or the oleate of mercury is very efficient. A saturated solution of salicylic acid in collodium, applied twice daily, is a convenient and excellent remedy. The main difficulty is that in the majority of cases, as soon as the diseased area is sterilized by these remedies, it becomes reinfected by the clothing or hands of the patient. Pemphigus contagiosus should be treated by bichloride of mercury washes and a dusting powder. Prophylaxis includes careful boiling of the underclothing, and keeping the crotch and axilla as dry as possible with a good dusting powder.

CHIGGER (SAND FLEA).

The chigger, or sand flea (*Pulex penetrans*), is distributed widely over tropical and many parts of subtropical America and the West Indies. It is supposed to have been carried in 1872 from South America to Africa. At present it is widely distributed on both African coasts and in certain sections of India. It is a very common pest in the Philippine Islands, where it is known as "tungau." The chigger is a minute, reddish-brown flea, and attacks both man and animal. When impregnated the female attaches herself to the skin surfaces and burrows under the skin, head first. Ovulation takes place in the cutaneous tissue and the female increases to the size of a small pea. If unmolested the ova, when mature, are expelled through the point of entrance, through which also the female is ultimately extruded. The chiggers may vary in number from one to several hundred. They usually lodge in the feet and legs, but the hands, arms, genitals, and face may also be invaded. The bite of the insect causes little pain, and the female is usually detected when she commences to enlarge beneath the skin. There is then intolerable itching, with formation of small papules, with red, inflamed heads, and a black spot on the summit. The papules become pustular, discharge, form small ulcers, and eventually heal, leaving small, pitted scars. When the lesions are numerous, particularly when neglected in the unclean and the physically deteriorated, extensive infections and sloughing wounds may occur. Rarely tetanus and phagedenic areas develop.

Treatment.—Treatment consists in complete enucleation with a needle or the point of a fine scalpel. Chloroform, turpentine, infusion of tobacco, mercurial ointment, and the essential oils allay the itching and kill the parasites. The essential oils, particularly the oil of eucalyptus, act not only as a cure, but also as a preventive against the bites of the insects.

MYIASIS.

Infection by Larvæ of the Diptera.—Screw-worm (*Lucilia Macellaria*), the larva of the common blue-bottle flesh fly, a very common fly in the United States, West Indies, and South America, causes infection in man through the female laying her eggs on wounds in the skin and in the noses or ears of people sleeping in the open. During the campaign at Santiago de Cuba, in 1898, numerous cases of infection by this larva were seen in wounds and abrasions about the feet of the men and horses. In the tropics they have also been seen attacking the vagina of recently delivered women. The eggs deposited in these locations hat into the larva, known as the screw-worm on account c spines running around the body of the worm very m of a screw. The larvæ are about three-quarters of an are extremely active, and burrow widely, causing e all the tissues. On account of the circles of spines t cult to extract from their burrows in firm tissue. Scr the nose are very painful and exceedingly fatal. The la and ethmoidal sinuses, and eventually may even ente intolerable pain at the bridge of the nose, with a blood the nostrils. A very large percentage of the cases die infection into the sinuses or meninges. When the la they penetrate the tympanic cavity, causing severe otit meningitis. Numbers of such cases have been repo from the Rio Grande border.

Treatment.—Treatment consists in the injection o such as carbolic acid and chloroform. Better still, c on a small probe tipped with absorbent cotton, and, nose or ear is explored and each worm as it lies en is touched with the chloroform-saturated cotton. T ately and they may then be readily extracted with sm ficial wounds the destruction of the larvæ is much m

Intestinal Myiasis.—The larvæ of diptera are ve the alimentary canal of man. They usually gain en lowed on fly-blown food. No less than nineteen diffe identified in human evacuations. As a rule no sy and the first the patient knows of the existence of th haps a copious mass of them in the stools. In tropic of larvæ is very much more frequent than in te obvious reasons connected with the difficulty of pre The appearance of the larvæ is usually viewed with the patients, but, as a rule, they are entirely harmle produce some symptoms of gastrointestinal disturba diarrhœa, and abdominal pains. Free purgation i larvæ are seen in the stools, to ensure evacuation of this purpose calomel is the best drug, as it exercises but a toxic effect on the larvæ.

Dermatobia Cyaniventris.—This common Americ on the skin of man and cattle. The larvæ penetrate t producing large pustular lesions (locally known as *v* this fly there are great numbers of diptera whose lar man. In all of them the lesions are similar to that America these are principally the *Musca vomitoria.* t fly, and the bot-fly of the ox and sheep.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES IN WHICH THE CHIEF MANIFESTATIONS ARE IN THE BRAIN AND ITS MEMBRANES.

HEMORRHAGE INTO THE BRAIN, CEREBRAL THROMBOSIS, AND EMBOLISM.

Definition.—Apoplexy consists in the sudden onset of paralysis and loss of consciousness from an abrupt intracranial lesion. In its most typical form it is due to hemorrhage in the cerebrum, but it may also be due to hemorrhage into the cerebellum, into the brain-stem, or into the meninges, and it may result from embolism or from thrombosis. When it arises without being accompanied by a demonstrable brain lesion it is spoken of as an "apoplectiform attack." An inflammatory process in the central nervous system, so acute that minute hemorrhages occur in the affected area, is also spoken of as apoplectiform, as, for example, "apoplectiform bulbar paralysis."

The term "apoplexy," as commonly employed, is nearly equivalent to the popular term "stroke," and is used so indefinitely that it is better to use the more accurate terms cerebral hemorrhage, cerebral thrombosis, or embolism when describing the condition present. The symptoms produced by thrombosis and embolism are almost identical with those due to hemorrhage, and will be found discussed in the consideration of the differential diagnosis of the disease.

Etiology.—As already stated, the usual cause of apoplexy is the rupture of a bloodvessel in the brain or its meninges. This is due in the great majority of cases to changes in the bloodvessel produced by disease or by injury. These changes are described in the article on Arteriosclerosis. The immediate causes which produce rupture of an intracranial vessel are numerous, for all factors which cause a sudden increase in blood pressure may result in so great a strain on a weakened vessel wall that it gives way. Thus, apoplexy not rarely follows a paroxysm of rage, a severe nervous shock, a sudden muscular effort, as in running for a car, in straining at stool, and during sexual intercourse. The use of alcoholic and other stimulants may also cause rupture.

Frequency.—Men are more frequently attacked by apoplexy than are women, because they suffer so much more commonly from arteriocapillary fibrosis. The ratio is about as 80 to 20 per cent., according to Starr, but

Gintrac puts it at 56.6 to 43.4 per cent. Of 816 cases of cerebral hemorrhage collected by me from various sources, 454 occurred in men and 362 in women.

The period of life at which cerebral hemorrhage most commonly occurs is from fifty to eighty years of age. This is the age period during which the patient is actually most liable to this accident; but if the ages of the entire number of persons dying of apoplexy in a given series of statistics be added together and an average obtained, the largest number of cases is found between forty and sixty years, because so few persons live to eighty years that not many persons of that age are to be found in such a series. The following table is a combination of the cases of Gintrac and Breese, and shows the age incidence of cerebral hemorrhage by decades from thirty to eighty years.

Between 30 and 40 years of age	74
" 40 " 50 " "	98
" 50 " 60 " "	138
" 60 " 70 " "	172
" 70 " 80 " "	124

The question of age in its relation to apoplexy is, however, more dependent upon the state of the bloodvessels than upon the actual years of existence, for not infrequently a syphilitic of thirty years of age may suffer more from degeneration of the arteries than another man at seventy.

Available statistics do not show any increase in the frequency of cerebral hemorrhage. Thus, from 1879 to 1884, 12,408 patients were admitted to the medical wards of St. Bartholomew's Hospital, London, and of this number 79 were affected with cerebral hemorrhage. During the five years from 1897 to 1902, 12,089 medical patients were admitted to the hospital, and among them there were 62 cases of cerebral hemorrhage.

Pathology.—The changes which take place in the bloodvessels of the brain which result in apoplexy are those of arteriosclerosis as we meet it in other parts of the body; the intima becomes roughened and eroded, the muscular sheath undergoes fatty degeneration, and the fibrous sheath becomes less elastic than in health. Aneurysmal dilatations frequently develop, and these are the parts of the vessel from which hemorrhage often ensues. In the article on Arteriosclerosis it was shown that the causes of this state are syphilis, lead, gout, renal disease, and, not least important, advanced years; but of all these causes renal disease is probably the one which most frequently produces vascular rupture, because it is usually associated with cardiac hypertrophy and a high arterial tension, which increases the stress on the weakened vessel wall.

Rupture of a vessel occurs very much more frequently in certain areas than in others, as already pointed out. This is because certain vessels suffe from arterial sclerosis earlier than others, and also because of the anatomical relationship. Thus, the blood current reaches the left middle cerebral artery more directly from the heart than it does on the right side, where it first passes through the innominate artery, which diminishes its force. Durand Fardel states that 75 per cent. of the miliary aneurysms which affect cerebral vessels involve the branches of the middle cerebral artery which enter the anterior perforated space, namely, the lenticulo-striate and the lenticulo-thalamic vessels. For this reason the lenticulo-striate branch was called by Charcot the "artery of cerebral hemorrhage." About 50 to 60 per cent. of all cerebral hemorrhage is from this vessel, and therefore occurs in the internal capsule or near it. (See Hemiplegia.) The sharp spurt of blood which follows rupture of the vessel wall may break through the corpus striatum in either direction, often internally through the caudate nucleus, or it may break through the optic thalamus; and when the rupture is large and the blood pressure high, the blood thus finds its way into the lateral ventricles (see Fig. 118), into the third ventricle, and even into the fourth ventricle, where it causes death by pressure on the vital centres, if death has not already ensued from shock and the damage to the cerebral tissues.

When these "capsulo-ganglionic" vessels do not give way the cause of the symptoms is usually rupture of some of the outer branches of the Sylvian artery, producing lesions in the cortex. In still other cases, which are less frequent, the hemorrhage takes place into the pons and still more rarely into the cerebellum.

Cerebellar hemorrhages are especially prone to inundate the fourth ventricle.

A very much rarer form of apoplexy is that in which by reason of disease of the blood, or of the vessels, small oozings or extravasations take place through the vessel walls, which on subsequent examination do not reveal any rupture. This extravasated fluid finds its way alongside the vessels, and so does damage to a wide area without causing any very gross lesion in the brain tissues. Such a state may develop in the course of purpura or leukocythemia. Extravasations of blood into the meninges and cortex also occur as the result of injury.

In those cases in which the hemorrhage is arrested before it does great damage much depends upon the part of the brain which is affected.

If the hemorrhage occurs on, or in, the cortex, and is small in amount, the convulsions and paralysis which ensue may only involve part of the arm, or leg, or face, or one of the special senses, or a particular function controlled by the centre that has been destroyed, or such a monoplegia may be due to a small hemorrhage in the subcortex cutting off the fibres of the corona radiata descending from the cortical centre. But an equally small hemorrhage still lower down, where the fibres from the entire cerebral hemisphere come together in the internal capsule, will produce a complete hemiplegia (Fig. 116, lesion of ordinary hemiplegia). On the other hand, if the lesion occurs still lower down—that is, in the brain-stem, where bundles of fibres are separating from the main paths and crossing to the opposite sides to connect with cranial nerves—it will produce crossed paralysis—for example, the face is paralyzed on one side and the body on the other. Ordinary "crossed paralysis" indicates a lesion in the lower third of the pons because at this point the motor fibres for the face have crossed but the fibres for the limbs have not done so (Fig. 117, lesion of crossed paralysis). If the posterior third of the internal capsule is affected as well as the anterior and middle thirds, we find hemianæsthesia as well as motor paralysis on the opposite side (Fig. 118); and if the very posterior portion of this limb is affected the optic radiation hemianopsia is added to the symptoms. (See Fig. 1 patient survives the attack the extravasated blood c rounded by a protective wall of lymph, which un

FIG. 116

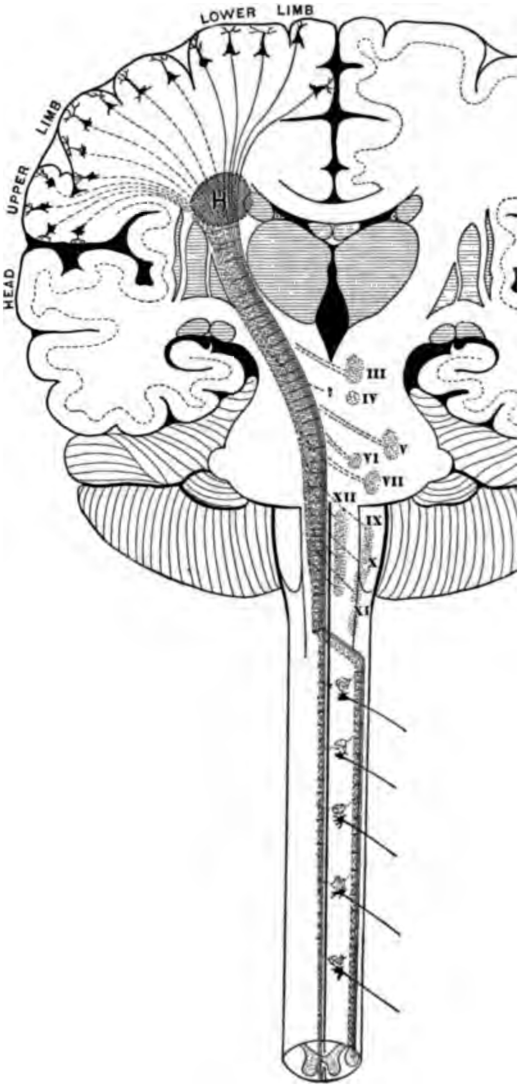


Diagram showing the fibres from the cortex forming the corona radiata, mated pass into the internal capsule. It also shows the decussation of the p passes to the right side of the spinal cord, and the direct or uncrossed tract also shows the secondary degeneration which occurs after cerebral hemorr follows the course of the motor tracts into the spinal cord. H. Site of lesi fibres going to the legs, the dotted are those going to the arms and motor numerals refer to the origins of the cranial nerves. (Modified from Van Gel while the clot softens, and contracts as its contents are being absorbed. The permanent lesion of apoplexy is thus commonly a cyst, but sometimes, absorption having been complete, only a scar remains. Not only do these changes take place at the site of the hemorrhage, when it affects the the cortex or motor fibres in the corona radiata or in the internal capsule, but degenerative alterations follow along the motor pathways through the peduncles of the cerebrum, the pons, the pyramids of the medulla, and so on into the direct and crossed pyramidal tracts of the cord. (See Fig. 116.)

FIG. 117

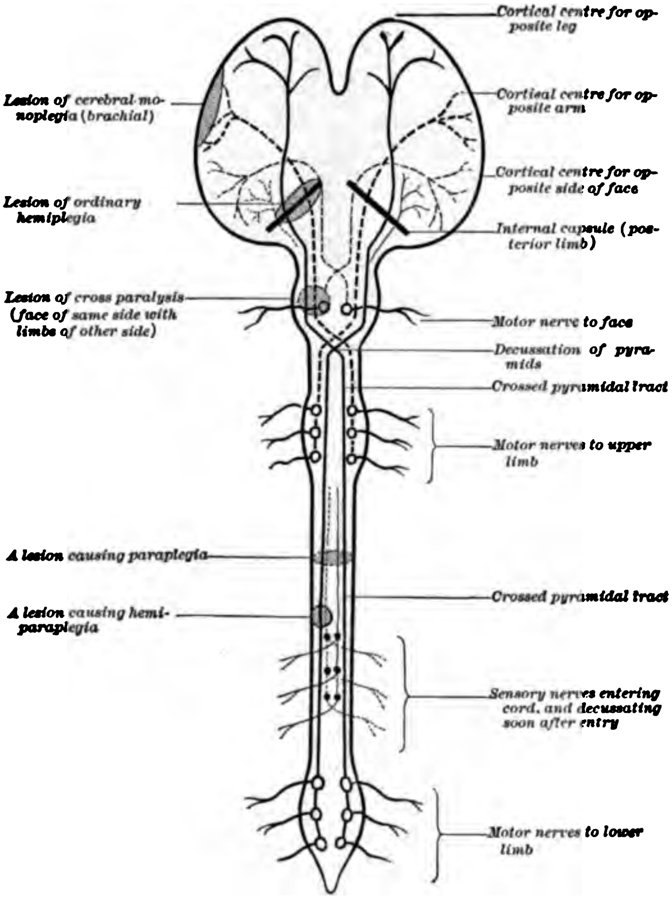
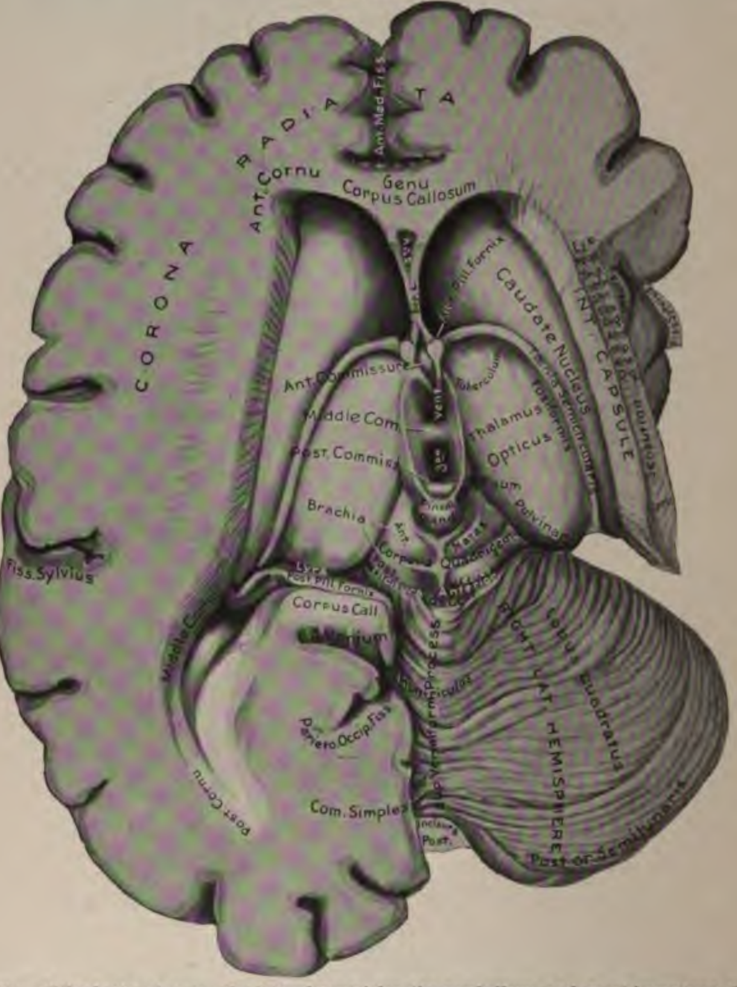

Cortical centre for opposite leg
Lesion of cerebral monoplegia (brachial)
Cortical centre for opposite arm
Cortical centre for opposite side of face
Lesion of ordinary hemiplegia
Internal capsule (posterior limb)
Lesion of cross paralysis (face of same side with limbs of other side)
Motor nerve to face
Decussation of pyramids
Crossed pyramidal tract
Motor nerves to upper limb
A lesion causing paraplegia
Crossed pyramidal tract
A lesion causing hemiparaplegia
Sensory nerves entering cord, and decussating soon after entry
Motor nerves to lower limb

Diagram showing the general arrangement of the motor tract and the effect of lesions at various points. (Ormerod.)

Symptoms.—The symptoms of apoplexy depend upon the site of the lesion and upon the suddenness and severity of the hemorrhagic extravasation, as already stated. A few cases have some *premonitory symptoms* such as *numbness or tingling* in the part of the body about to be affected, but most cases are attacked without warning. When the hemorrhage takes place from the *middle cerebral artery* the symptoms are usually as follows:

An individual who is apparently in his normal health is *suddenly seized with vertigo*, which causes him to stagger and fall. The *face* is at first *pallid* and later somewhat *congested*. The *respiration* is altered almost immediately. At first it may be *slightly gasping* and *irregular*, but soon becomes full and deep. The air is drawn into the lungs with considerable force and then equally forcibly expelled. As it enters it causes the relaxed soft palate to vibrate and as it escapes through the angle of the mouth, which is paralyzed, it produces a noise to which the term "*stertorous breathing*" has been applied. The *pulse* is *slow* and *full* and its *tension high* except for a few moments after the onset of the symptoms, when it may be rapid

FIG. 118



Cross-section of the brain, showing the lateral ventricles, the cerebellum, and, most important, the cross-section of the motor fibres in the internal capsule. (Modified from Fuller.)

and irregular from the shock. In some cases, too, in which the hemorrhage into the brain is very great and death imminent, the pulse may not become full and strong.

An examination of the patient's limbs may show that both sides are almost equally relaxed and powerless, but this is usually a temporary state due to shock, and in a very short time it will be found that the limbs on one side are moved, or at least are not quite powerless, while those on the opposite side are paralyzed. In other words, the typical paralysis of cerebral hemorrhage called *hemiplegia* is present.

The muscles of the trunk are never as completely paralyzed as those of the limbs. The muscles of the lower part of the face share in the paralysis, and for this reason the *features* will be *drawn away from the paralyzed side* because the normal balance between the muscles on the two halves of the face has been destroyed. Unlike the facial paralysis due to a lesion in the facial nucleus in the pons or in the facial nerve itself, the upper muscles escape, and so we find that the muscles of the forehead and eyes are not paralyzed; the forehead can be wrinkled and the eyes can be closed.

In the stage of onset we sometimes find the head and eyes turned sharply to one side (conjugate deviation), usually away from the paralyzed side. When this occurs it is said that the eyes "look at the lesion." The pupils are sometimes contracted, but more commonly are dilated, the pupil on the side upon which the hemorrhage has taken place being more dilated than its fellow.

Pricking or pinching the skin of the paralyzed side is not followed by any reflex contraction soon after the onset, though the deep reflexes may be present, but later, when the primary shock has passed away, it will be found that the *skin reflexes* as well as the *knee-jerk* and other *deep reflexes* are *exaggerated*, particularly upon the paralyzed side. Irritation of the sole of the foot almost invariably causes extension of the big toe (Babinski's reflex), a reversal of the normal plantar reflex, which is flexion of the toes. Ankle-clonus is also frequently present.

In cases in which the bladder and the bowel are full at the time of the "stroke," the shock of the hemorrhage may result in involuntary evacuations, but in some cases the bladder and rectum are not only retentive, in the ordinary sense, but fail to empty themselves when they become full. The bladder of an apoplectic patient should, therefore, be frequently examined, and if the urine accumulates in excess it must be withdrawn by the catheter. If the urine is examined a trace of albumin is usually found in it, even if actual renal disease is not present. The *temperature* of the body immediately after a hemorrhage is usually *subnormal*. With reaction from the primary shock, which is often of brief duration, the temperature rises from one to three degrees, the chief change being on the paralyzed side.

The *unconsciousness* of the early stage of apoplexy may last from a few hours to several days, according to the severity of the lesion. When it persists for any length of time the prognosis is correspondingly bad. In some cases the depth of the coma decreases and the patient emerges to some extent, only to sink back again into deep coma and high fever a few days later when a secondary hemorrhage takes place, perhaps bursting into the ventricle, or secondary irritation of the brain, produced by the presence of the extravasated blood, develops. In other cases in which the extravasation of blood has been limited, and the parts damaged are not of vital importance, the patient gradually improves in his mental state and progresses toward recovery. In most cases, however, the mind never completely recovers its previous acuity.

The persistency of the hemiplegia also varies greatly in different cases. It may remain absolutely unchanged, one-half of the body being helpless, or it may diminish in severity and even greatly improve to the extent that the patient can walk about and write. In most of the cases, however, in which this much to be desired result is attained, the lesion has probably been due to embolism or thrombosis rather than to an actual hemorrhage.

Many patients after an attack of apoplexy not only suffer from a degree of *mental failure*, but in addition become *exceedingly irritable* or *emotional*, crying, laughing, or getting into a furious temper at slight causes. Distinct loss of emotional control has been said to be particularly prone to occur when the lesion involves the frontal lobes.

There still remain to be considered several additional symptoms of apoplexy which are often present. The most important of these is *aphasia*. It is most common in cases in which the right side is paralyzed, because the speech centre is chiefly in the third left frontal convolution. If the patient is left-handed, however, the aphasia is present when the left side is paralyzed. The symptom aphasia varies very greatly in the time at which it is first evident and in its severity. Not infrequently it is one of the first signs of a beginning apoplexy, the speech becoming suddenly confused and indistinct. In most cases, however, the aphasia is first noticed after the patient recovers from the immediate effects of the stroke. The persistency of the aphasia varies greatly. In some cases it remains so severe that the patient has great difficulty in making himself understood. In others it improves so greatly that it may entirely disappear, or only be present when the patient becomes very tired or excited.

Another special symptom is *hemianopsia*, which is of the *homonymous* type, that is, the corresponding halves of the visual fields are darkened because the temporal half of one retina and the inner or nasal half of the other retina has lost its visual function.

Hemianæsthesia persisting after recovery from the primary shock is a rare symptom and is never complete, thereby differing from the hemianæsthesia of hysteria. The sense of heat or cold or touch may be impaired, but total loss of the senses does not take place.

When the power of recognizing objects placed in the hand is lost (astereognosis) it indicates a lesion in the superior parietal portion of the cortex on the opposite side. (See Fig. 121.)

In some cases of hemiplegia of a severe type *bed-sores* develop on the heel or buttock of the affected side. The tendency to this accident can be greatly decreased if the patient is not permitted to lie in one position for long periods of time, and if great care is taken as to the cleanliness of the skin in the places where pressure is marked.

As a sequence to an apoplexy we find not only *persistent paralysis*, but as time goes on *contractures* occur in the affected limbs. The forearm and hand, however, suffer far more than the leg. The flexor muscles being stronger than the extensors, the hand is usually found in marked flexion upon the wrist, and the fingers are turned into the palm of the hand. The leg is usually held in the position of extension so that it cannot be bent at the knee, and for this reason it is often swung with a lateral movement from the hip when the patient attempts to walk. These contractures are usually much diminished when the patient is asleep, and are due to degenerative changes in the crossed pyramidal tracts. (See Fig. 116.) Occasionally, that curious mobile spasm of the fingers or other members, called "athetosis," is a sequence of apoplexy and posthemiplegic chorea may develop. This is commonest in the hemiplegias of childhood, but occurs in adults. Full doses of strychnine may produce spastic contractions in old cases of hemiplegia. Next to signs of spasm the most common symptom as a sequel is *muscular atrophy*, which is due in part to disuse of the muscles in the paralyzed limbs and does not develop till some time after the acute stage of the attack. Charcot has reported instances in which true *trophic joint changes* took place, but they are exceedingly rare.

The symptoms of an attack of apoplexy in which the lesion has been due to rupture of a branch of the middle cerebral artery having been described, there still remain to be considered those additional symptoms which develop when other parts of the cranial contents are affected by the giving way of other vessels.

When a *vessel in the dura mater* is ruptured, usually as the result of an injury, it is the middle meningeal artery or vein which suffers as a rule. The clot which is formed is either outside the dura mater (extradural) or beneath it (subdural). The noteworthy peculiarity of these cases is that the primary unconsciousness due to a blow speedily disappears, the patient may recover his normal mental state, and then, after an interval varying from some minutes to several hours or days, becomes heavy and dull, and finally comatose. *Spasmodic movements of the muscles* on one side of the body, followed by paralysis, may develop. If the extravasation of blood is large, the *pupil on the paralyzed side is contracted* and that on the side of the hemorrhage is *dilated*. This is called the "Hutchinson pupil." The *eyes are turned away from the lesion*, whereas in the acute stage of an ordinary apoplexy they are turned toward it. It is in this form of apoplexy that surgical interference is absolutely essential to save life. Such hemorrhages sometimes occur in the insane without a history of injury, particularly in paretics and in chronic alcoholics.

In hemorrhage from a *vessel upon the cortex*, as one of the branches of the Sylvian artery, it is important to recall the fact that muscular spasm, or a convulsion, usually ushers in the attack due to the disturbance of the cells in the motor area.

When the blood finds its way into the lateral ventricles, a *general convulsion* affecting the entire body may develop. Such cases usually pass into *deep coma* and soon die.

When the lesion is in the pons the *temperature* is usually soon *hyperpyretic*, the *pupils* are *tightly contracted*, *swallowing is difficult*, and the *respiration* is *very slow*. Death comes rapidly in these cases as a rule.

Under the name of "*ingravescent apoplexy*" a condition is met with in which the *symptoms develop very gradually*, beginning, it may be, by an attack of vertigo or aphasia, followed by the slow development of the other symptoms already described, so that several days may elapse before the entire symptom-complex of apoplexy is present.

Diagnosis.—An attack of apoplexy, or hemorrhage into the brain, must be separated from a number of conditions which may closely resemble it. Two conditions which resemble it so closely as to be inseparable in some cases are thrombosis and embolism of the cerebral vessels. The symptoms produced by these accidents will be found described below.

An ordinary attack of syncope can readily be differentiated by the pallor, the feeble pulse, the weak heart of a fainting attack, and the quick recovery of the patient after receiving some rapidly acting diffusible stimulant.

In epilepsy the peculiar initial cry, the bloody froth at the mouth, the general convulsion, and the deep unconsciousness are more constant and severe than in apoplexy, even if the hemorrhage takes place in the cortex. Epilepsy is more common in the young, apoplexy in those of advanced years, and there may be scars to indicate previous severe falls in epileptics. A history of epilepsy will practically settle the diagnosis, although the epileptic is liable to apoplexy. The respiration in the coma of apoplexy continues deep and noisy, the lips and cheeks of one side flap in the air current, showing paralysis, and weakness of one arm and leg may be ascertained. In the coma of epilepsy the breathing soon becomes quiet. Rarely in epilepsy weakness of one side of the body or of one limb may appear as the patient emerges from the coma; this postepileptic hemiplegia is ascribed to exhaustion, for it passes off in a few hours or days, but it may put the diagnosis in doubt for a time.

From the stupid stage of acute alcoholism apoplexy can be differentiated by the history of the patient, by the odor of alcohol on his breath, by the fact that both legs are moved if they are irritated by pricking, proving the absence of hemiplegia, and by the cool skin as compared to the hot, dry skin of apoplexy. It is, however, possible for an apoplectic to have induced an attack by the use of alcohol, and therefore the odor of alcohol on his breath is not of great importance from a diagnostic standpoint.

Opium poisoning is differentiated by the presence of contracted pupils, by the fact that by shouting the patient can be aroused, by the absence of paralysis, and by the presence of the corneal reflexes.

The coma of uræmia and of diabetes may also be mistaken for apoplexy, but in uræmia there may be œdema of the lower extremities, and there is a urinous odor about the body and breath of the patient. If the renal disease is of the parenchymatous type, the peculiar waxen appearance of the patient and the urine heavily loaded with albumin will make the diagnosis clear. If the uræmia is of the type caused by chronic contracted kidney, these latter signs will not be present nor will œdema be found, and as apoplexy often complicates this disease the diagnosis may be most difficult. Unless the coma is very deep one side may be moved far more than the other, revealing the hemiplegia of apoplexy. However, in uræmia the cerebral affection may be more pronounced in one hemisphere, thus causing a hemiplegia ("uræmic apoplexy"). In diabetic coma the sweet odor of the breath and the presence of sugar and acetone in the urine will make the diagnosis possible.

In sunstroke likewise hemiplegia may be found, which is not an apoplexy in the ordinary sense.

Finally, it must not be forgotten that apoplectiform attacks not rarely develop in the course of general paralysis of the insane. In this disease the speedy return to consciousness and recovery of power in the affected limbs, with the physical signs of this disease and the mental symptoms, will render a diagnosis possible.

The separation of the paralysis due to hemorrhage from that due to thrombosis depends more upon the history of the patient than upon the symptoms actually present. As already stated, hemorrhage usually follows some effort and takes place during waking hours, whereas the paralysis of thrombosis develops during periods of quiet and rest, as during sleep, so that the patient wakes to find the palsy present. In cases of hemorrhage premonitory symptoms are not common, but in thrombosis they are nearly constant. Thrombosis is most frequent in the aged or prematurely senile, and in syphilitics. Again, thrombosis does not cause such violent symptoms nor is the onset of the symptoms so sudden, but consciousness is preserved or is not so completely lost, or if moderate coma is present it is brief in duration and is followed by mental clearness. Finally, in cases of thrombosis, the recovery of power in parts of the paralyzed side may be quite rapid, and at the end of a few days only a few muscles, as one arm or leg, are affected. Such cases are, however, often mentally feeble, emotional, and forgetful after the attack. There is often to be found a history of syphilis, of arteriosclerosis, or an infectious disease, which has predisposed the patient to a formation of a clot by causing disease in the lining of the bloodvessel or producing changes in the blood.

Embolism can be determined by the sudden onset of symptoms during the waking hours, as a rule, and by the discovery of some source of clot or foreign body, as in an endocarditis with vegetations or a septic focus elsewhere. Unconsciousness, when it develops, may be as profound, but is usually more transient than in hemorrhage (Mills), and is often entirely absent. The appearance of the patient is not so alarming as in hemorrhage, and localized or general twitching may be present in the affected limbs. Not rarely as a collateral circulation is established, the symptoms improve with great rapidity and after a few days may amount to only a partial monoplegia. It must be recalled, however, that in some cases of thrombosis and embolism the symptoms may be so like those due to hemorrhage that a differentiation is almost impossible.

The chances of the case being one of hemorrhage rather than embolism or thrombosis is as 6 to 1, according to Dana.

Prognosis.—Many cases of hemorrhage into the brain survive the first rupture, but if so they nearly always fall victims to subsequent attacks. Out of 441 cases occurring in St. Thomas' and St. Bartholomew's Hospitals,

London, 375 proved fatal, a mortality percentage c
centage is, however, far too high for private practice,
are often seen. In the severe cases in which the con
temperature low and then quite high, the paralysis s
of the bladder or bowels impaired, death will probabl
attack, and if Cheyne-Stokes breathing is present death
place within a few hours. If the hemorrhage affects the
death may come on speedily, but when the hemorrhage
often survives. When the hemorrhage is cortical the
than in the other forms unless the pressure symptom
patient not rarely dies from pulmonary œdema or pneu
current disease.

Treatment.—Apoplexy, like other forms of internal h
be materially benefited by medicinal treatment. If na
a clot to plug the bleeding vessel, the hemorrhage mu
has done so much damage that death is inevitable unless
surface or in the meninges, when surgical relief should b
pressure with which the blood escapes into the soft text
so great that if the leak is of any size the mechanical in
tissues must be very great, and for this reason the org
disabled.

Until the recent researches of Cushing, of Baltimor
significance of high arterial pressure in cases of he
sations inside the skull, it was universally taught th
a full, bounding pulse in a case of apoplexy indicate
ticularly if at the same time there was distinct venou
thought being that by this means the blood pressure wo
that there would be a corresponding decrease in the lea
tured vessel. Cushing's investigations have apparently
doubt that the high arterial pressure which is so constant
who suffer from hemorrhage inside the skull is an effort
tain the blood supply to the vital centres at the base of
if this blood supply cannot be maintained because of a fall
death speedily ensues. In other words, the maintenanc
pressure in these cases is an advantageous sign, and any
in arterial tension is an indication that the vasomotor
paralyzed and that the blood supply to the centres at th
is becoming impaired. If Cushing's studies are correctly
venesection or the administration of vascular sedatives
of lowering tension, is therefore a distinctly harmful m
and truth demands that we should admit that the ph
if anything in the way of controlling the escape of blo

For the purpose of apparently making an effort to do g
the friends who may demand activity rather than maste
mustard foot-bath may be given, and some diffusi
Hoffmann's anodyne may be used if the patient is al
atropine may be given if arterial tension falls and the su
and clammy.

If vomiting occurs, the patient should be promptly turned on one side so that free drainage from the mouth may take place, and in order that particles of food may not be drawn into the respiratory passages.

If the tongue falls back in such a manner as to make the breathing difficult it should be drawn forward by means of the fingers covered with a towel. If, by chance, the patient is convulsed, his tongue should be protected from damage by placing between the teeth a penholder or tooth-brush handle covered with a piece of muslin.

The patient's body should always be put in that position in which breathing is most easily carried on.

The treatment after the hemorrhage has ceased consists in absolute rest, in the application of an ice-bag to the head, and attention to the bowels and bladder to prevent them from becoming overdistended. Gentle purgation is probably advantageous for its influence upon the brain. If any evidence of nervous excitation exists, it may be controlled by small doses of the bromides or morphine. If any tendency to secondary reaction develops in the course of a few days, cold to the head and small doses of aconite to quiet the circulation may be administered. Later on, with the hope of diminishing the paralysis, iodide of potassium may be given in moderate doses in order that it may aid in the absorption of the extravasated blood and remove products of inflammation. There is little use in giving the iodide of potassium for the purpose of causing absorption earlier than two or three weeks after the hemorrhage. Strychnine is usually not valuable in these cases, as it is very apt to produce spasm or contracture in the parts which are paralyzed by irritating the motor tracts in the spinal cord.

From three to four weeks after the hemorrhage it is often advantageous to apply a slowly interrupted faradic current to the paralyzed muscles, with the object of maintaining their nutrition by exercise and keeping them in the best possible condition, in the hope that eventually they may receive a sufficient amount of nervous impulse from the cerebral centres to be able to respond sufficiently to permit the patient to move his limbs. Massage is another excellent means to combat the loss of power. Passive and active movements followed by a course of systematic exercises will render valuable service in combating secondary contractures of the paralyzed extremities. Great care should be taken that all stimulants which increase circulatory activity, and all foods which readily cause indigestion, be avoided, as these two factors tend to produce that most unfortunate complication, another hemorrhage.

INFANTILE CEREBRAL PARALYSIS.

Definition.—As a result of injury or disease of the brain during fetal life or soon after birth, it not rarely happens that certain portions of the cerebrum fail to develop, and as a consequence a number of very characteristic conditions are produced, which depend in their nature upon the site and size of the atrophied region.

These conditions can be grouped in three divisions: In the first there is a spastic paralysis which may be limited to one side of the body (spastic hemiplegia), or it may be bilateral (spastic diplegia). In spastic diplegia the legs may be affected alone or the arms and legs may both be involved. The second class is chiefly characterized by mental failure varying in severity from slight intellectual deficiency to absolute idiocy. In some instances the defect is manifested by epileptic attacks. The third class presents disorders of the special senses, such as blindness, deafness, mutism from deafness, and it may be epileptic seizures as well.

Etiology.—Acute infectious disease occurring in the mother during pregnancy may result in lesions in the fetal brain. Syphilis may also act in this manner. It is probable, too, that definite developmental defects may be hereditary, as when the parent or parents are epileptics, neurotic, alcoholic, or otherwise degenerate. Premature labor is a frequent cause of diplegia. A very large proportion of cases develop as a result of injury during birth because of a meningeal or cerebral hemorrhage. In a few cases the damage is due to a fall in early infancy, and in still others there develops some time during the first three years of life a cerebral thrombosis, a hemorrhage, an encephalitis, or a meningitis which is followed by the brain symptoms about to be described. Such a condition may arise as a complication or sequela of any one of the acute infectious diseases of childhood. A convulsion may be said to be the cause in certain cases, but it is probable that the lesion in the brain is responsible for this symptom rather than that it is the provoking factor. Finally, there are certain cases in which it is impossible to discover any cause whatever.

Pathology and Morbid Anatomy.—When spastic paralysis is present it is due to a lesion which involves the motor portion of the cerebral cortex and neighboring convolutions. The lesion itself in long-standing cases is sclerotic or atrophic in character and is associated with similar changes in the motor fibres, which pass from the cortex, and in the basal ganglia as well. In some cases the sclerotic change is limited to these ganglia. When there is diplegia both sides of the brain are involved. In that type of case in which mental impairment is present the atrophy and sclerosis affect the anterior convolutions of the brain, and when disorders of special sense are present it is because the perceptive centres of the senses affected are involved in the damaged area. It is readily seen, therefore, that as the lesions are distributed so are the manifestations of the disease varied.

The exact nature of the cerebral lesions has been found to be of several types: (1) A localized atrophy, or failure of development, may produce an excavation of the surface of the brain, usually due to meningeal hemorrhage at birth, with formation of a clot which indents the delicate cortex permanently. This indentation is called porencephaly (poros, the Greek word for "hole"). (2) A sclerotic process with overgrowth of connective tissue and atrophy of the nervous protoplasm may be present. (3) Imperfect development of the cerebral cells may be found. (4) Atrophy may follow cerebral softening produced by the closure of a vessel by an embolus or thrombus. (5) An inflammatory process in the pia mater may cause an adhesion to the cerebrum and so cause atrophy (meningo-encephalitis). (6) A cerebral hemorrhage may not only destroy the cerebral tissue, but cause a cyst to develop. (7) A cyst may cause atrophy from pressure. (8) Hydrocephalus, in which state the cerebral ventricles may be so distended that the brain tissues atrophy from pressure. (9) Rarely an external hydrocephalus may produce the same results.

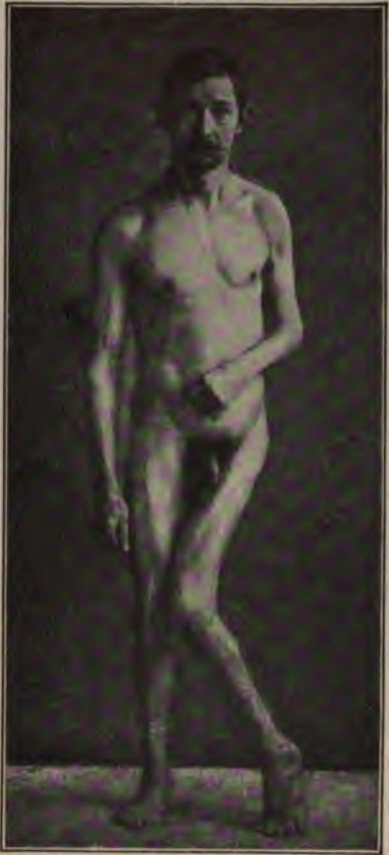
Symptoms.—The symptoms in a child affected by this accident, like those of ordinary apoplexy, vary greatly in speed of onset, in severity, and in type. When the lesion occurs at birth there are often no symptoms for several days or even weeks, except it may be an unusual limpness of the limbs and some difficulty in swallowing. In other cases *unilateral spasms* or *general convulsions* speedily develop, but these are not of long duration, and it is noticed that the child's head is not held erect, but falls from side to side, backward or forward. The convulsions may affect the entire body, or be confined to the side in which paralysis is about to develop. Associated with these convulsions there is a *marked rise in temperature*, the fever sometimes reaching as high as 105°. After the convulsion there may be *post-convulsive coma*, which may last several days. Gradual improvement now takes place, and as the child returns to consciousness it is found that there is *loss of power upon one side of the body*. Shortly afterward it is also noticed that the arm upon the affected side is not only paralyzed, but that it is in a somewhat spastic state. The leg suffers in a similar manner. Later on, *clubfoot* and a sharply flexed hand develop from secondary contractures. The paralyzed *limbs* fail to develop as they should, become *atrophied*, and are often considerably shorter than the limbs upon the healthy side. The *reflexes* are *exaggerated*. Sensation is not impaired.

If the child survives, the paralyzed parts gradually become markedly distorted and tenotomies may be necessary to prevent the contracture from causing so great a deformity as to make any motion impossible. Even these means may not make walking possible. If the intellectual portions of the brain are not involved, and if the patient reaches the years of puberty, it not infrequently happens that by prolonged training a very remarkable degree of ability is developed in the non-paralyzed side so that the individual can follow some pursuit which will render him self-supporting. In other instances, however, the spastic condition of the affected limbs is very marked, the hand is sharply flexed at the wrist, and athetoid movements of the fingers may be present whenever any attempt is made to move them. These athetoid movements occur very soon after the paralysis is noticed in certain cases. In others they do not develop for a long time. The state of the legs is even more noticeable, if such a thing be possible. Here we find that the parts are at once placed in strong extension if they are touched, the muscles of the calf are tightly contracted and the feet are inverted and turned inward. The thighs are abducted. When the attack comes on after the child has learned to speak, there may be marked aphasia for a time, but this symptom often gradually disappears.

Spastic diplegia may affect either the arms or the legs, usually both arms and legs, and is characterized not only by loss of power in these parts, but by rigidity, which is particularly marked in the lower extremities. The symptoms usually develop slowly, not acutely as they do in spastic hemiplegia.

Various deformities of the lower limbs occur, and the muscles of the trunk, particularly at the back, are so rigid that the child is as if fixed in a plaster cast. In other instances where the condition has developed some time after birth, the patient can sometimes walk by the aid of a cane or crutches, but in those instances in which the lesion is severe the contractures and athetoid movements, the exaggeration of the reflexes, and the imperfectly developed muscles all combine to make the child absolutely helpless. There is no loss in the control of the sphincters nor any trophic disturbances in the way of bed-sores, nor are there any sensory disturbances.

FIG. 119



Hemiplegia, with contractures. The patient had suffered since the age of two years. (Curschmann.)

Both of these types not infrequently suffer from epileptic convulsions, which may be of the Jacksonian type. In those cases which are due to lesions in the intellectual area of the brain, idiocy may be present. In those instances in which the defect of mental power is not complete the patient is called an imbecile. Such patients are often subject to violent outbursts of anger, to attacks of malicious mischief, and are often exceedingly filthy in their habits.

In the cases in which the posterior portions of the brain are affected, the disorders of special sense do not usually make themselves manifest until the child is at least a year or eighteen months old. Often prior to the discovery of any symptoms of disorder of special sense, epileptic convulsions have called attention to the fact that the cerebral development is imperfect. In some instances the disorders of vision may amount to nothing more than a hemianopsia. In others there may be total blindness, or deafness, or loss of smell and taste. In these cases also ordinary epilepsy and Jacksonian epilepsy are often present. In some instances minor epilepsy takes the place of major epileptic attacks.

When a hemiplegia develops in a child of a year or more it is usually due to hemorrhage or embolism or to the polioencephalitis of Strümpel rather than to meningeal disease.

Diagnosis.—The diagnosis in a well-developed case of infantile cerebral paralysis is not difficult. The early development of the malady (after a hard labor it may be), the marked arrest of normal development, and the epileptic convulsions, with the spastic state of the muscles, all separate this form of infantile paralysis from those forms which depend for their existence upon lesions in the spinal cord, for in the latter the paralyzed parts are flaccid. There are, however, two forms of spinal spastic palsy of childhood with which this condition can be confused, one of which is the so-called "hereditary spastic spinal paralysis," but in this disease the mental symptoms are lacking and the condition is progressive. The second state which resembles this disease is "amaurotic family idiocy," but in this malady the paralysis may be flaccid or spastic and blindness is an early symptom.

Prognosis.—It must be evident that the prognosis as to complete recovery in severe cases is anything but good. In those cases in which the mental powers are feeble and convulsions frequently recur, the outlook is bad both as to recovery and a long duration of life. If they live they are hopeless imbeciles or idiots. When the affection is confined chiefly to one side of the brain—that is, when there is hemiplegia—adult years may be reached and ordinary mental pursuits followed in many cases.

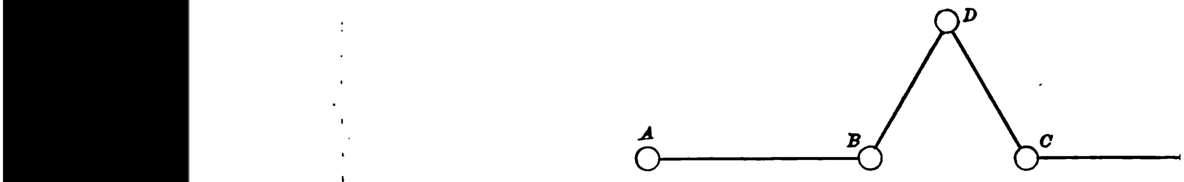
Some of these patients, moreover, can be materially improved by proper training, in which instance special senses which are not impaired, or intellectual centres which have escaped the wreck, may be developed to such an extent that a fair degree of comfort and intelligence may be attainable. The convulsions cannot be cured, as they depend upon faulty development, but they may be modified by skilful treatment, consisting in the administration of nervous sedatives, the avoidance of all causes of nervous excitement and irritation, and the moderate employment of the bromides. As a rule, better results can be obtained from hygienic methods and from mental training in an institution devoted to this purpose than can be obtained at home. At one time it was considered that operative interference might be of very great value in these cases, but we now know that little can be expected from such a plan of treatment. In those cases in which the skull has seemed to be abnormally small it was proposed that the skull should be cut, or bone should be removed, in such a way as to permit expansion of the brain. But the smallness of the skull is probably more dependent upon the size of the brain than is the size of the brain upon the condition of the skull. In some instances the parents prefer running the risk of the child's death as the result of such a grave operation rather than to have it continue a hopeless invalid, and in such cases, if there are distinct localizing symptoms, epileptic or otherwise, the question of cerebral localization and operation must be carefully considered.

Little's Disease.—The name "Little's disease" has been applied by some writers to the cerebral palsies of childhood; but is probably best restricted to cases in which there is congenital spastic rigidity of the limbs, particularly of the legs. tending to improvement. In Little's disease, thus defined, there is normal mental capacity and no epilepsy or athetosis. The condition is purely motor from defective development of the pyramidal system in the brain or, according to Dejerine, in the spinal cord.

APHASIA.

Definition and Symptoms.—Aphasia is a condition i of speech becomes impaired or arrested as the resul those parts of the brain which are concerned with th in words. For the power of speech it is necessary th have not alone the motor centres which will cause movements which give rise to certain sounds, but in be, in close association with these centres, others in w an idea must originate, and still others in which a se of words, or sounds of words, is stored. When a ch an object, such as a horse, is pointed out to him, an frequently repeated at the same moment. He theref a certain shape and form with the word "horse." F necessary that his visual apparatus shall carry to his and that his brain should store up that form as typif It is also necessary that his auditory apparatus shall certain sound or sounds, and that his brain shall ass the form that he has seen. In addition it may be th stroked the horse, and so his sense of touch has conve tain model, or form, which is associated with those his eyes and ears. When, therefore, he sees a horse a ories or imprints derived from these various sources attempts to reproduce the word "horse" by a process certain muscles which are necessary for making this so fore in one sense a complicated function, closely conn of special sense, of intellection, and with the motor neu perhaps made more clear by the following diagram:

FIG. 120



A, pathways for receiving imprints or models; *B*, centre for storage of m motor memories; *D*, concept centre; *E*, motor centres for controllin

A visual impulse, an auditory impulse, a touch i impulse, or a taste impulse, or all of them together, where they are received and stored. From *B* these are transmitted, whenever they are needed, to those which are concerned with the power of the conceptio may be transferred directly to *C*, which may transfo by mimicry, without any idea or higher intellectual p cerned with imitation. If the child thinks of a ho memories of the character of a horse from B and sends from D an impulse which cause C to send impulses to the organs of speech. It is evident, therefore, that if the sensory tracts are diseased before the centres for storing the character of external objects have received and retained models or impressions speech will be impossible, or if any part of the mechanism described in the diagram is undeveloped or damaged, the entire chain fails because one of its links is broken.

This process may, however, be even more complicated than that described. Thus, there may be stored in the storage centre not only the model or imprint of a horse but also the additional memory of the appearance of the word "horse" when in type, and in addition there may be stored the memory of certain movements which are characteristic of a horse, so that the child can imitate its movements or perhaps draw an outline of its appearance. While pantomime and drawing are not speech, they are so nearly related to it as to really form part of it. Again, it is necessary that there should be stored at C the models or memories of those muscular movements which will give rise to the sound of the word horse, for this is part of learning how to talk.

In certain cases in which the brain is diseased the patient may see and feel and hear a horse, and his concept centres may know perfectly that a horse is before him, or that the word " horse " is in print, but he cannot say the word "horse" because the centres concerned with the storage of memories of the muscular movements necessary to speak the word "horse" are destroyed. He may write the word " horse " or draw a horse, but he cannot say "horse."[1] To this form of motor aphasia the word *aphemia* is applied. If the centre in which the memories of how to write the word "horse" are destroyed the condition is called *agraphia*, and if those in which the muscular movements required to describe a horse by gesture are diseased it is said to be *amimia*. When the tracts that associate the storage centres for memories of words are interfered with, the patient skips, or jumbles, or repeats his words, and this is called *conduction aphasia;* and if he speaks one word when he means another, it is called *paraphasia*.

On the other hand, when the sensory portion of the speech mechanism is diseased the patient may be able to say the word "horse," but if he sees a horse he cannot say that he has seen it because he has lost the memories of the horse; or if he sees the word "horse" in print, he may be able to reproduce the letters in writing, in their order, but he is entirely unable to read, for he has lost the memory of the significance of these letters when so joined. This is called *alexia*, or *word blindness*, an unfortunate term, as blindness would indicate failure of visual power, which does not exist. Again, in certain cases there is loss of memory of sounds. The voice of a speaker may be heard and even imitated, but the patient understands nothing more than if an unknown language was spoken. It conveys no idea to his mind. This is called *word deafness*—another unfortunate term, because hearing in the ordinary sense of that word is perfect. *Apraxia* is still another nearly related state in which the patient fails to appreciate the purposes or uses of an object. He may see, hear, and touch a knife or a coin, but his mind cannot grasp its uses.

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[1] As a matter of fact, these powers are usually lost.

When we come to study the lesions which produce these disturbances in the ability to express an idea, we find that when the patient has aphemia or motor aphasia the damage has been done to the third left frontal convolution (Broca's convolution). In such a case he also cannot write either his own ideas or the words that he hears spoken, but he can copy. If the lesion is a severe one, his power of understanding words he sees written or hears spoken is usually impaired. In other words, he also suffers from word-blindness and word-deafness. When he has word-blindness or alexia, the lesion is at the angular gyrus. If there is a pure alexia the lesion is in the subcortical substance of the angular gyrus. Aphasia also develops when a lesion takes place from hemorrhage, embolism, or thrombosis in the knee of the internal capsule, for at this point the fibres which convey speech impulses are destroyed.

TUMORS OF THE BRAIN AND ITS MEMBRANES.

Intracranial tumors arise from the substance of the brain itself or from the membranes which surround it. A great variety of these growths have been recorded, but by far the most common are tubercle, gumma, glioma, and sarcoma. Cancer, fibroma, osteoma, neuroma, and vascular tumors also rarely occur. Echinococcus cysts may develop.

Etiology and Frequency.—The causes of these morbid growths are not understood except in the case of tubercle and gumma. Sex seems to exercise a very distinct influence, for we find that males suffer very much more frequently than females. Gowers states that out of 650 cases of brain tumor 440 occurred in males to 210 in females. Dana gives the figures at 644 to 320, and Starr's figures are nearly identical in their proportions. This great preponderance in males is not explained by either syphilis or injury, for there is no greater frequency of gumma in men than in women. On the contrary, tubercle and glioma are the growths that are particularly frequent in men. Gowers has shown that after the first six months of life till old age all ages suffer about equally. Thus, the percentage in the first decade is 18.5, in the second 14, in the third 20, in the fourth decade 18.5, and in the fifth 14. Most of the growths in childhood are tuberculous, and indeed they form 53 per cent. of all growths at all ages, if gumma be excluded.

Pathology and Morbid Anatomy.—Tumors of the brain affect its tissues in its different areas as follows, according to Gowers: In the hemispheres 297, in the cerebellum 179, in the base of the brain 76, in the pons 59, in the central ganglia 48, in the medulla 31, in the corpora quadrigemina 13, and in the crus 10.

Tuberculous tumors occur as solid, firm, round masses which are not rarely multiple. Their size varies from that of a pea to a hen's egg or even larger. The growth starts from the lymphoid sheaths of the vessels, and rapidly obliterates them. For this reason it is devoid of vessels and its tissue soon undergoes necrosis, so that on section it is cheesy and shows spots of softening. As the surface is soft, and the surrounding brain substance is also softened, the mass is clearly outlined, hence the name tuberculoma. Finally, the growth may become calcified or undergo suppuration. Tuberculous growths are often found in the cerebellum, and they also occur in the pons and cerebrum. When in the cerebrum they are usually found along the great vessels in the interpeduncular space or in the fissure of Sylvius; not rarely the growth is near a Pacchionian body, but it may be found in the depths of the centrum ovale.

Gummata of the brain rarely reach a size greater than that of a hickory-nut. They are also somewhat cheesy in appearance and have an irregular surface, which may be gelatinous, or indurated and hard, and enclosed in a fibrous capsule. These growths are thought to spring from the bloodvessels of the dura mater.

Sarcoma occurs as round, oval, or spindle-cell tumors which destroy the tissues of the brain as they grow. When they are of the gliomatous type they differ greatly from all the growths so far described, for they are not round, but extend by a process of infiltration between the nerve cells. They may be soft and mucoid (myxoglioma), or firm and fibrous (fibroglioma). Gliomas of the soft variety are liable to hemorrhage. A cystic form of glioma due to softening is not rare. Gliomata are usually single.

The secondary changes produced by these growths are of importance and depend chiefly upon the pressure of the tumor upon healthy tissues which in this way are destroyed. The very growth of the tumor inside the skull also increases intracranial pressure, and if it be so situated that it prevents the free passage of cerebrospinal fluid from the choroid plexus in the lateral ventricle through the third ventricle and the *iter a tertio ad quartum ventriculum*, then distention of the lateral ventricles or internal hydrocephalus develops. A tumor of the pons, of the corpora quadrigemina of the middle lobe of the cerebellum, or in the third ventricle may cause such obstruction. A third result of the intracranial tumor is irritation of the nerve cells of the brain and inflammation in them or in the meningeal membranes, and lastly it may cause actual thinning of the skull by the pressure induced.

Symptoms.—By far the most common symptom of brain tumor is *headache*. This headache is usually severe and is characterized by sharp exacerbations. In some instances it is dull and boring in character. In others it is sharp, stabbing, and tearing. By reason of its constancy it prevents sleep, and in its most severe paroxysms may produce *temporary aberration of mind*. The pain is widely diffused, and is particularly severe if it encroaches on the dura which is supplied with sensation by the fifth nerve. If the pain is localized it does not necessarily indicate that the growth is in that neighborhood, although in those cases in which the tumor is superficial the locality of the growth and of the pain is often identical.

Next to headache in constancy as a symptom is *vomiting*. The expulsion of the stomach contents is usually spoken of as "projectile," and in this respect it resembles the vomiting of certain forms of intestinal obstruction. Although the vomiting is severe, nausea is often absent. This symptom is supposed to be most frequent and severe when the growth is rapidly progressing. *Vertigo* also occurs and varies in severity from slight dizziness to

a degree which causes the patient to fall. Not inf
causes the patient to walk in the direction in which it
go. Vertiginous symptoms are more common in tun
than in lesions elsewhere, and often the patient falls t
very important symptom of brain tumor is *optic neur*
large proportion of cases and which often enables us t
brain tumor with the aid of the ophthalmoscope w
toms are so obscure that it is difficult to determi
patient's disease.

Optic neuritis is usually most marked in cases of tu
of the midbrain, and of the great ganglia near the bas
quently when the tumor is in the cortex or springs fro
branes, but the localizing value of neuritis is really not
both optic nerves are involved, although sometimes
one before it attacks the other. Gowers gives three
neuritis: First, irritation of the nerve fibres produced
finally causes inflammation. Second, distention of t
the lymphatic spaces of the papilla by subarachnoid
contains irritating poisons. The third cause is thoug
of the meninges, which is so frequently present, and
the optic nerve.

Slowness of thought and gradual *mental failure*
present. Sometimes *aphasia* develops. All these
prone to occur when the tumor affects the frontal
tom of aphasia is, of course, most frequently dev
frontal lobe is involved. Epileptiform convulsions
quarter of the cases of brain tumor, and are especi
instances in which the growth directly or indirectly
the cortex. So, too, there may be symptoms which fo
early stages of an apoplexy. Quite rarely actual ru
occurs, and so the symptoms are really apoplectic.

Paralysis due to brain tumor may be unilateral or
lateral it may manifest itself as a monoplegia or as
usually gradual in onset, and when hemiplegic in type
of a growth in the upper part of the pons, in the crus,
sule, or over a wide area in the cortex. Localized pa
plegia, may be due to a growth involving the cortex o
the fibres have come together so closely that even
affect the whole bundle and produce widespread par
plegia involving the arm or leg is not infrequently ass
form attacks, limited to the paralyzed part. If the g
third of the pons the ordinary form of crossed paralys
face being paralyzed on the side of the lesion, while
paralyzed on the opposite side.

A rarer form of *crossed paralysis* (Weber's syndro
tumor of one crus involving the third nerve. The ey
side opposite the lesion and the pupil dilated on the side
arm and leg, as in ordinary hemiplegia, are paralyzed u

Bilateral paralysis due to brain tumor can only occur when the growth is multiple and presses upon both sides of the brain, or when it is so situated that it can at once cut off fibres from both sides as they approach the middle area, as in the pons or in the medulla. Under these circumstances the legs are usually more affected than the arms, even though the face as well as the arms be included in the paralysis. Contractures, tonic spasms, or convulsions, either generalized or Jacksonian, may also occur. They usually indicate that the growth is situated somewhere near the cortex, where it produces other irritations.

If the tumor is in the frontal area and grows forward it may cause protrusion of the eyeballs and paralysis of the extrinsic muscles of the eye, while if it grows backward it may cause spasms and epileptiform convulsions by the irritation of the motor area of the cortex.

The development of a growth in the central region produces symptoms which are more definite than those which are found in association with growths elsewhere, because it is the motor area of the brain. Not infrequently localized convulsions, or Jacksonian epilepsy, occur in such cases and paralysis limited to the same muscles may follow such an attack. The part in which the convulsion begins and the subsequent paralysis indicate the presence of the tumor in or beneath the centre supplying the centres controlling these muscles. Disorders of sensation in the affected limb may be present, consisting of numbness, tingling, and even hemianæsthesia.

When a tumor involves the parietal region the symptoms are not definite, but pertain chiefly to common sensation and "muscle sense."

If the superior parietal lobule be invaded there is more or less loss of "muscle sense," and often the symptom astereognosis is present. When the angular gyrus are involved, word blindness may be present.

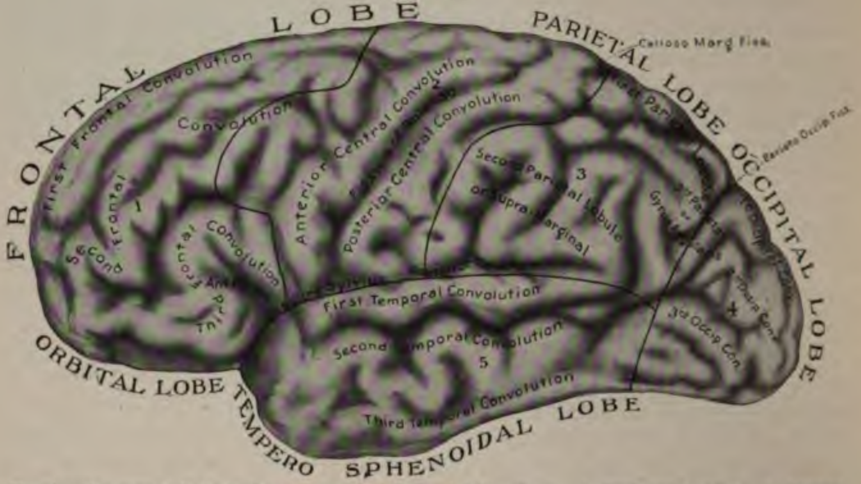
When that portion of the parietal area near the longitudinal fissure is invaded we not infrequently have spasms or convulsions in the lower extremities on the side opposite the lesion, because the growth begins to invade the part of the leg centre which is on the inner surface of the hemisphere.

Tumors of the occipital lobe, if in the cuneus or otherwise near the calcarine fissure, produce lateral homonymous hemianopsia. If the growth in the occipital lobe is sufficiently far forward to involve some of the parietal area, and so do damage to the angular gyrus, word-blindness and hemianopsia may develop, and if the invasion extends still farther, hemiataxia, hemianæsthesia, and even some hemiplegia due to involvement of some of the fibres of the internal capsule may be present.

In the first left temporal convolution brain tumor produces word-deafness, a form of sensory aphasia. Occasionally large tumors in the temporal area produce vertigo.

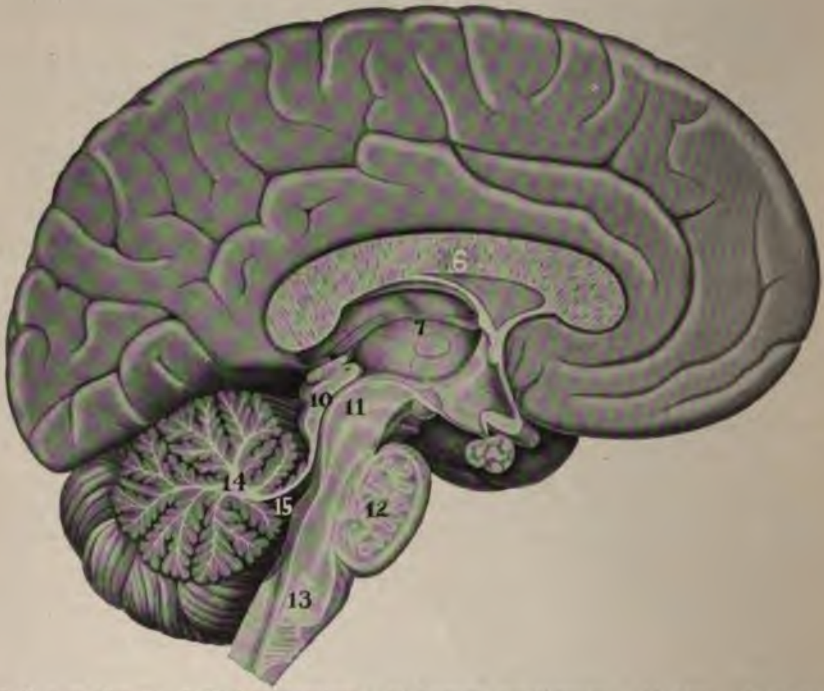
Tumors of the corpus callosum are not only very unusual, but produce symptoms which are not very definite, being primarily those of mental failure and secondarily those due to encroachment upon neighboring parts. In addition to the general symptoms of brain tumor, the growth in this region produces hemiplegia, ultimately developing into paraplegia, great mental dulness, and finally coma.

FIG. 121


FRONTAL LOBE
PARIETAL LOBE
Calloso Marg Fiss.
OCCIPITAL LOBE
Parieto Occip Fiss.
ORBITAL LOBE
TEMPERO SPHENOIDAL LOBE
First Frontal Convolution
Second Frontal Convolution
Third Frontal Convolution
Anterior Central Convolution
Posterior Central Convolution
Second Parietal Lobule or Supra Marginal
First Temporal Convolution
Second Temporal Convolution
Third Temporal Convolution
3rd Occip Con
1
2
3
4
5

Showing the areas of the brain concerned with special functions. 1. Prefrontal area. 2. Central area. 3. Parietal area. 4. Occipital area. 5. Temporal area. (Modified from Fuller.)

FIG. 122

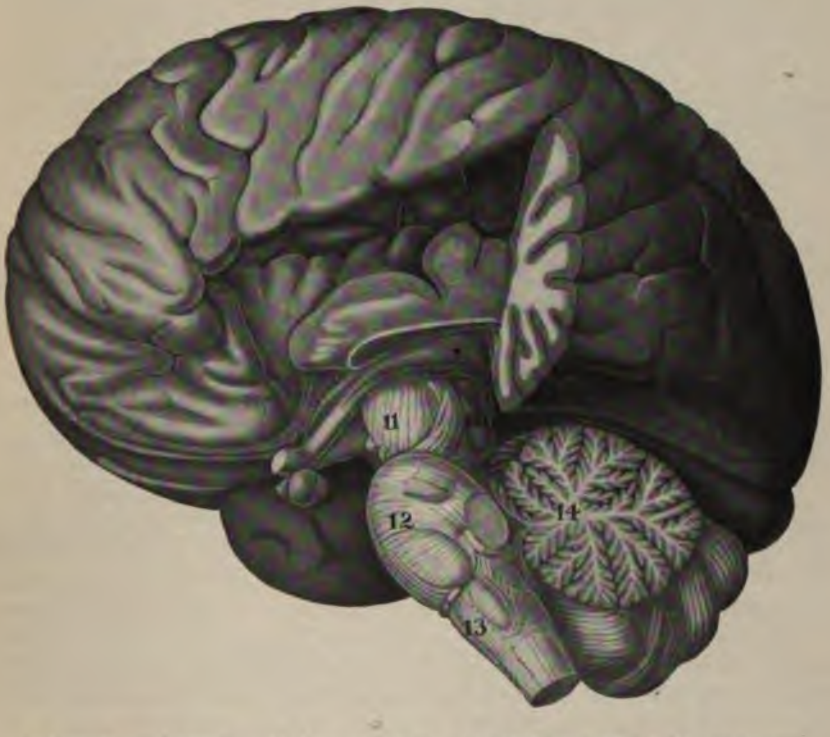


6. Corpus callosum. 7. Thalamus opticus. 10. Corpora quadrigemina. 11. Crus. 12. Pons. 13. Medulla oblongata. 14. Cerebellum. 15. Fourth ventricle. (Modified from Reichert.)

When a tumor involves the great basal ganglia or the fibres of the internal capsule, hemiplegia is the most prominent symptom, and hemianæsthesia and choreic movements may be present.

If the posterior part (pulvinar) of the optic thalamus and nearby tissues, particularly one of the optic tracts, are involved by the growth, hemianopsia may be present, but this hemianopsia may be separated from that which is due to a lesion in the occipital lobe by the presence of Wernicke's sign (hemianopic pupillary inaction).

FIG. 123



10. Corpora quadrigemina. 11. Crus. 12. Pons. 13. Medulla oblongata. 14. Cerebellum. (Modified from Reichert.)

Having from these several symptoms determined that a brain tumor is present, it still remains for the physician to determine its locality, and this can only be done by his knowledge of cerebral localization. For this study the brain can best be divided into fifteen parts, most of which can be seen on the accompanying diagrams (Figs. 121, 122, and 123).

The numbers in the text refer to the numbers in the figures and show where the growth would be situated.

TABLE OF CEREBRAL LOCALIZING SYMPTO

1. Tumors of the prefrontal area . . (See Fig. 121).	No symptoms, or Stupidity. Silliness. Emotionalism. Loss of smell on one side or both sides. Hemianopsia and optic neuritis. Protrusion of the eyeball. Paralysis of the extrinsic ocular muscles.
2. Tumors of the central area. . . . (See Fig. 121.)	Jacksonian epilepsy. Sensory disorders, tingling or hemianæsth Impaired muscle sense. Motor aphasia and agraphia. Local palsy after spasm.
3. Tumors of the parietal area . . (See Fig. 121.)	No symptoms, or Loss of muscle sense if supramarginal gyr Word-blindness if angular gyrus and infe Paralysis or spasm of the lower limbs if t cerebral area is invaded. Perhaps slight paralysis of the sixth nerve affected.
4. Tumors of the occipital lobe . . (See Fig. 121.)	Homonymous hemianopsia if the cuneus calcarine fissure and first occipital conv Failure to grasp meaning of surrounding cuneus escapes. Word-blindness and some hemianopsia if t Hemiataxia, hemianæsthesia, and partial capsule is slightly involved in the poster mous hemianopsia from involvement of
5. Tumors of temporal area . . . . (See Fig. 121.)	No symptoms if on right side. Word-deafness if the posterior part of the convolution is involved. Vertigo and forced movements, if the g irritation of internal ear.
6. Tumors of corpus callosum . . (Very rare.) (See Fig. 122.)	Gradually developing hemiplegia followe Dulness and other mental symptoms sugg Stupor } and various symptoms due to Coma } structures. Death.
7, 8, 9. Tumors of the great basal ganglia and capsule. (See Fig. 122, for 7 and Fig. 118 for 8 and 9.)	Progressive hemiplegia. Anæsthesia (?). Choreic movements if tumor involves opt part of capsule (8). Starr thinks that th No localizing symptoms if the tumor involv nucleus (9). Hemianopsia and the hemiopic pupilla if the optic tract or its endings near the thalamus and adjacent tissues are invol action of Wernicke is absent hemianops occipital lobe involving the cortex or th
10. Tumor of the corpora quadrigemina (vermis of the cerebellum) and pineal gland. Additional information as to these lesions can be had by studying Figs. 118 and 123.	Cerebellar inco-ordination. Forced movements. Ocular palsies, often symmetrical. Hemianopsia if primary optic centres o blindness if destroyed on both sides. Deafness or partial deafness if posterior t gemina are affected.

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[1] In the preparation of this table much use has been made of the facts s on Nervous Diseases.

[2] A ray of light thrown on the blind half of the retina will not produce re though the pupils react normally if the light strikes the other half of the retin

11. Tumors of crus (Very rare.)	Hemiplegia. Hemianæsthesia (?). Paralysis of oculomotor nerve upon same side as tumor (crossed paralysis).
12. Tumors in the pons	Facial palsy on same side as tumor; hemiplegia on opposite side (crossed paralysis). Trifacial paralysis on same side as tumor if below middle of pons; hemianæsthesia on opposite side (crossed sensory paralysis). Hemianæsthesia and hemiplegia if tumor is large and above middle of the pons. Conjugate deviation of eyes away from the lesion.
13. Tumors in medulla	Hemiplegia and hemianæsthesia with hypoglossal paralysis or other cranial nerve palsy on side of lesion. Glycosuria and vasomotor disturbance. Symptoms of bulbar paralysis if growth is large.
14. Cerebellar tumor	Headache, Vomiting, Vertigo, Optic neuritis } unusually severe. Reeling gait if in middle lobe. Glycosuria and cranial nerve palsies if pressing upon or in middle lobe. Bulbar symptoms (*i. e.*, cranial nerve palsies). Hydrocephalus if tumor presses on aqueduct of Sylvius.
15. Tumor in anterior fossa	Same symptoms as prefrontal tumors. Loss of smell, sight, and oculomotor paralysis. Acromegaly, optic neuritis, and temporal hemianopsia if the tumor is in middle fossa and involves optic chiasm.

Diagnosis.—As already stated, the important symptoms in the diagnosis of brain tumor are headache, vertigo, optic neuritis, convulsions, and paralysis. It must be remembered, however, that these symptoms are none of them pathognomonic of brain tumor, and that each of them is often present in other maladies. It is the combination of symptoms rather than any one of them alone that indicates or establishes the diagnosis. Headache and vertigo are due to a host of causes, as convulsions and paralysis are common conditions; it is the localization of the convulsions and of the paralysis, pointing to a distinct focus, that is significant. Hysteria may, however, cause localized palsies and localized anæsthesia. Sometimes, too, localized convulsions are not only due to tumor, but are symptomatic of a general condition such as poisoning, or of a widespread nervous disease such as paresis. The most valuable sign of brain tumor, optic neuritis, may be present in chronic contracted kidney, chronic lead poisoning, and severe anæmia, etc., but these conditions can be eliminated from the case by the absence of the other signs and symptoms of these maladies.

Among the pathological states that may produce symptoms of brain tumor is to be mentioned localized meningitis due to tuberculosis or syphilis. (See Meningitis.) In tuberculosis the degree of optic neuritis is usually slight; there is a primary tuberculous focus existing elsewhere, and, as the case progresses, evidences of a general meningeal inflammation may develop. Syphilitic meningitis is usually diffuse, not localized, and when localized is to be considered as a gumma or tumor. Another cause of such symptoms is abscess. This may be differentiated by its rapid development and by the presence of a septic focus elsewhere, as in the ear or in other parts of the body. (See Brain Abscess.)

The question as to the character of the growth is determined by the following facts in many cases. Tuberculous growths are frequently met with, particularly in children. The presence of tuberculous infection elsewhere also points to this form of tumor. So, too, the presence of a malignant growth in some other part of the body suggests that the lesion in the brain is of the same character. A history or the presence of the scars of syphilis will indicate the existence of a gumma. Gowers states that if the tumor be in the cerebellum or pons, there is some probability "of its being tubercle or glioma, and if it is in the cortex the probability that it is syphilitic is considerable." "A tumor outside the brain tissue is probably sarcoma." So, too, the disappearance of the symptoms under active antisyphilitic treatment point to syphilis as the cause. "A tumor which grows rapidly at the onset and then becomes stationary is probably tuberculous."

Prognosis.—The prognosis in all forms of brain tumor is grave. It is least so in gumma and next best in tubercle, for in the former active treatment may cure, and in the second a long period of arrest may ensue. A growth in the pons or medulla is more dangerous as to life than one in the cortex, but the presence of severe symptoms of brain tumor, be the seat of the growth what it may, is always of grave omen.

Treatment.—From what has been said of the etiology and pathology of brain tumor it must be evident that medicinal treatment can do nothing more than palliate the patient's suffering, unless the growth be syphilitic. For the relief of the headache the various coal-tar products, such as acetanilid, phenacetin, or antipyrin, may be employed, and their efficiency is usually much increased by giving simultaneously 1 or 2 grains of caffeine and 10 or 20 grains of one of the bromides, preferably the bromide of strontium or bromide of sodium. In those cases in which the headache becomes so severe as to be insupportable, hypodermic injections of morphine may be employed, but it is an interesting therapeutic fact that this drug gives less relief in the pain of brain tumor than in almost any other affection characterized by pain. Excessive vomiting is to be checked by administrations per rectum of 10 or 20 grains of chloral with 60 grains of bromide of sodium, to which may be added 30 to 60 minims of deodorized tincture of opium. So, too, epileptiform convulsions, if they are severe, may be prevented, at least in part, by the use of the bromides, or, if an aura is present, by inhalations of nitrite of amyl.

In those cases in which there is the history of syphilis, the administration of mercury and the iodide of potassium is, of course, strongly indicated, and if a gumma is producing symptoms such as convulsions, which may in themselves endanger the patient's life, the mercury should be pushed as actively as possible, being given by the mouth, by inunction, by hypodermic injection—that is, by every avenue of entrance—in the hope that it may exercise its influence upon the syphilitic growth before a convulsion sufficiently violent to cause death ensues. In other instances, when there are no acute symptoms which demand immediate interference, protiodide of mercury may be alternated with iodide of potassium, iodide of sodium, or iodide of strontium.

When the growth is due to tuberculosis the administration of cod-liver oil, iron, and arsenic, and residence in a climate in which plenty of fresh air and sunshine can be constantly obtained, is essential. In such cases great improvement in the general health may do something toward arresting the local process.

In those forms of brain tumor which are not tuberculous or syphilitic, the use of drugs, except to relieve pain, is practically useless, and operative interference offers the patient the best chance of recovery. Surgical procedures can, however, only be resorted to in those cases in which a definite localization of the tumor can be made, and where that locality is so situated that the surgeon can reach it without doing damage to vital parts. The earlier the operation is performed the better is the outlook. M. Allen Starr has collected 400 cases of brain tumor which were operated upon. In 154 instances tumor of the cerebrum was successfully removed, and the patients recovered. In 52 cases the patients died. In 16 instances in which the tumor was in the cerebellum it was removed and the patients recovered. In 8 instances death followed removal. It is interesting to note that in 91 instances of supposed cerebral tumor the growth could not be found on operation. The same failure to discover a growth was met with in 22 instances of supposed cerebellar tumor.

Of the cases which recovered 52 were sarcoma, 29 were cysts, 8 were gummata, 19 were tuberculous, and 15 were gliomas.

For the measures which are to be pursued in the removal of brain tumor, the reader is referred to the modern works on surgery.

ABSCESS OF THE BRAIN.

Definition.—Abscess of the brain is a condition in which an accumulation of pus takes place in the cerebrum, or the cerebellum, or between these parts and their covering membranes. In the latter case the brain substance forms one wall of the abscess and the membranes the other wall. The latter type of abscess differs from purulent meningitis in that the inflammatory process is primarily in the nervous tissues, and that it is limited to a comparatively small area. Abscess of the brain without involvement of the membranes is much the more common form, and the white matter suffers very much more frequently than the gray substance. Abscess rarely occurs in the central ganglia, the pons, the medulla, or the middle lobe of the cerebellum.

Etiology.—Abscess of the brain is always due to an infection by some pathogenic organism. In some instances the process is excited by an injury which affords a nidus in which the organism may develop; in other cases a septic embolus is carried from another part of the body in which there is an infected area; in still others the infection takes place more directly, as in those instances in which by fracture of the skull injury and infection both occur, or, as in the case of abscess of the middle ear and mastoid disease, the infection spreads by way of the sinus.

Middle-ear disease is the cause in a great proportion of cases, about 45 per cent. arising from this primary focus of infection and 25 per cent.

from injury. The remaining causes are infection of the other cranial bones than the mastoid or septic foci elsewhere.

Males are affected far more frequently than females, in the proportion of 3 to 1. This is largely due to the fact that males are so much more exposed to injury than females. If the cases of abscess due to middle-ear disease are studied by themselves, the proportion is almost equal; but if those due to trauma are considered by themselves, the proportion is 5 of men to 1 of women.

The disease is most frequent between the tenth and twentieth years of life. In the second decade ear disease is the common cause, and in the third decade ear disease and injury, or a distant focus of infection, are about equal in frequency as causes.

Pathology and Morbid Anatomy.—A majority of cases of brain abscess affect the right side of the brain. Out of 71 cases collected by Oppenheim 55 were in the temporal lobe, 13 in the cerebellum, 2 in the pons, and 1 in the crus.

The pathological process which results in abscess consists primarily of an encephalitis which speedily goes on to the stage of suppuration. This encephalitis may be due to any organism capable of causing an active inflammatory process. Thus, the *Streptococcus pyogenes*, the *Staphylococcus pyogenes aureus*, and the pneumococcus may be the provoking factors. In other cases the typhoid bacillus may be the cause, and even the streptothrix may produce such a lesion, as in a case seen by me in consultation with Dr. J. H. Musser. The abscess is nearly always single, but two or more pockets of pus may be present. The size of the abscess varies greatly. In some cases it is so small as scarcely to be recognized, a small collection of pus being found in the centre of an area of softening; whereas in other cases the quantity of pus may be very large, varying in quantity from a drachm to several ounces.

The abscess may be surrounded by an inflammatory fibrous wall which serves to separate it from the neighboring white matter, or it may be contained in a cavity without any such well-defined margin, the walls of the abscess being composed of softened brain tissue. The latter form is prone to spread more rapidly than the abscess which has been walled off. On the other hand, the wall of the abscess may rupture and produce sudden death. Not rarely the extension of the inflammatory process produces a meningitis if the abscess is situated near the surface of the brain, and a septic thrombosis of the nearby vessels may occur.

Symptoms.—It is of interest to note that cerebral abscess may occur without producing symptoms sufficiently typical to lead to an antemortem diagnosis. In the great proportion of cases, however, the symptoms of its existence are well developed. The most constant of these is *headache*, general or localized; it is excruciating when associated with middle-ear disease; next in constancy is *mental disturbance*, the patient being alternately irritable and dull, and often seeming to be exceedingly ill. The temperature, unlike that of septic processes elsewhere, is usually normal or subnormal, unless the abscess ruptures, when it may be hyperpyretic. The surface of the *skull* is often *hypersensitive*, and in some cases it has been possible to localize the abscess by the dulness on percussion produced by its presence. Optic neuritis is often present. None of these symptoms is in any sense pathognomonic, since all may occur in other states of disease; but when they are taken into consideration in connection with a history of middle-ear disease, injury, or the presence of a septic focus elsewhere, they possess great diagnostic value. The deafness due to destruction of the auditory centre is masked by the deafness due to the disease in the ear. *Paralysis of one side of the face* is not of much diagnostic value, since this symptom is often due to the inflammation about the facial nerve as it passes through the stylomastoid foramen.

The value of localizing symptoms depends, of course, upon the part of the brain which happens to be affected. When the infection spreads from the mastoid bone after or during otitis media, and infects the temporal lobe, there are no localizing nervous symptoms because we are in the dark as to the function of the temporosphenoidal lobe. In a few cases a lesion in the left temporosphenoidal lobe has caused aphasia. When the occipital lobe is involved the patient may present the symptom called *optical aphasia*, for in this lobe is situated the memories of objects and of words seen. Starr has observed a case of this character. The patient knows an object when it is placed before him, but cannot name it. A lesion of the temporal lobe may also produce this symptom by pressure, and it may likewise cause hemiplegia or hemianæsthesia by pressure upon the internal capsule. *Hemianopsia* may also be caused in this manner.

When the abscess is in the cerebellum the symptoms are those characteristic of cerebellar tumor, viz., a staggering gait, vertigo, and, it may be, vomiting, diplopia, and nystagmus. In some instances the patient staggers toward the diseased side. If pressure is brought to bear by the abscess upon the crus or pons there may be *paralysis of the oculomotor or facial nerves* on the side of the lesion, with increase in the knee-jerks on the opposite side.

Diagnosis.—The diagnosis of brain abscess is not difficult if the symptoms just described have been preceded by a history of injury or of a septic process elsewhere, near or remote. There are two other states with which it may be confused, namely, meningitis and thrombosis of the lateral sinus. In the former condition the onset is usually more abrupt, the headache is prone to be more severe in the early stages, and a sharp febrile movement is usually present; whereas, as has already been pointed out, in cerebral abscess fever is often absent unless the abscess ruptures. The pressure of abscess produces a slow pulse like that of cerebral compression, but in meningitis the pulse is usually very rapid. Additional symptoms of meningitis, which are of great value, are the stiffness of the muscles of the neck, the muscular twitchings, the early development of squint, and, last of all, the presence of a pathological state of the cerebrospinal fluid obtained by lumbar puncture. (See Cerebrospinal Fever.)

When thrombosis of a lateral sinus is present the febrile movement is sharp and severe, with marked remissions and exacerbations as in sepsis. There is swelling of the jugular vein on the affected side and of the conjunctiva as well, associated, it may be, with exophthalmos. Swelling, œdema, and pain on pressure over the mastoid may also be present. The use of the ophthalmoscope also reveals choked disk in many cases as an early symptom.

Prognosis.—This depends very largely upon the site of the abscess and the ability of the surgeon to evacuate and drain it. In all cases the prognosis is necessarily grave. That many patients may recover if promptly relieved is shown by recent statistics, which show that 60 per cent. of traumatic abscess recover after operation, and about 50 per cent. of abscess due to ear disease do likewise.

Treatment.—The treatment is purely surgical, and for the necessary procedures reference must be had to surgical treatises.

ACUTE CEREBRITIS OR ENCEPHALITIS.

Definition.—Acute cerebritis, sometimes called "acute encephalitis," is a condition in which there is an acute inflammation of the brain arising as a primary disease not secondary to meningitis.

Etiology.—The cause of acute cerebritis is always an infection due to the entrance of a micro-organism into the body, and in most instances the condition arises as a complication of measles, scarlet fever, smallpox, or ulcerative endocarditis, or in the convalescence from influenza. Acute alcoholism and other forms of poisoning may, by diminishing vital resistance in the brain, predispose to this condition. Possibly trauma may have a like result. Whatever may be the cause, this condition as a primary acute disease is very rare.

Pathology and Morbid Anatomy.—The inflammatory process is not widely diffused, as a rule, but is found to exist chiefly in the distribution of one or more nearly related bloodvessels. At times it affects the same areas on both sides of the brain. In some instances only the cortex is involved, while in others the process chiefly affects the white matter. A limited form of this condition is the acute inflammation of the medulla, pons, or midbrain (polioencephalitis inferior of Wernicke or acute bulbar paralysis).

The changes found in the affected parts are those characteristic of acute inflammation in all the nervous tissues, and indeed in any acute inflammation, namely, hyperæmia, out-wandering of blood cells, and minute hemorrhagic extravasations, followed by the ordinary degenerative changes in the nerve cells produced by an interference with their normal blood supply and the effects of toxæmia. The nerve-cell body or ganglion cell itself suffers from cloudy swelling, loses its sharp outlines, and its nucleus becomes indistinct or disappears. The axones and dendrites also undergo a similar change. The interstitial tissues are at first filled with small cells, and ultimately there is an overgrowth of the neuroglia cells, so that patches of sclerosis are produced. This last result is, of course, permanent, and if it takes place to any great extent may seriously impair the function of the brain. If the inflammatory process is in the cortex, adhesions to the meninges may take place.

Symptoms.—The symptoms of acute cerebritis depend to a large extent upon the portion of the brain which is chiefly affected, although the general manifestations of an acute inflammatory process in the brain are present in all cases. The patient is seized, after a few hours of *general distress*, with *headache* and *dizziness*, followed by a *chill* and, it may be, *vomiting*. These symptoms are in turn speedily followed by *fever* and *rapidity* of the *pulse* and *respirations*, and these in turn in some instances by *delirium* of a violent type. If the case is severe the patient may now pass into *coma*, and then gradually pass to death, or, after several days, or even weeks, of these symptoms, *consciousness gradually returns*, the temperature falls, and recovery takes place, although, as already stated, permanent impairment of some of the cerebral functions may persist.

The special symptoms which depend upon the areas of the brain which are affected consist in *hemiplegia*, *monoplegia*, *aphasia*, or in *word-blindness* and *word-deafness*, *hemianopsia*, or *mutism*. Any of these may become permanent. In still other cases the patient suffers from impairment of the intellectual powers or the changes in the motor cortex produce *epileptic attacks*, and in other instances the development of the sclerotic patches already named results in the production of a condition identical with disseminated sclerosis with *nystagmus*, *tremor*, and *peculiar speech*. The very rare condition called "polioencephalitis superior of Wernicke" is manifested by the presence of *ptosis*, *strabismus*, *nystagmus*, and even *optic neuritis* with *vertigo* and a *staggering gait*. In addition there may be *difficult speech* and *facial paralysis*.

Diagnosis.—Acute encephalitis may be confused with brain tumor when epileptiform convulsions and paralyses are prominent, but the absence of choked disk and the rapid onset will distinguish the former. In its "comatose" and "epileptic" forms it may resemble apoplexy or epilepsy, and a close study of the entire clinical picture is necessary, with the history, to differentiate such cases.

Prognosis.—That the prognosis in acute encephalitis in the early course of the malady must be uncertain is manifest when we consider the character of the lesions which are present. Even after the active stage of the disease is passed the outlook as to complete restoration to health is still clouded because it is not possible to tell what secondary changes may develop in the brain or its membranes. All cases of acute encephalitis are to be regarded as of much gravity. A high temperature, convulsions, profound and prolonged coma are all very unfavorable symptoms. Even in the very grave cases a remarkable degree of recovery may occur.

Treatment.—This consists in the application of an ice-bag to the head, and in the use of moderate doses of tincture of aconite if there is circulatory excitement. If the bowels are confined, an active saline purgative is useful to move them and to deplete the bloodvessels. A hot foot-bath may also be used. Phenacetin and acetanilid may be employed for the relief of pain, but they usually fail. On the other hand, the use of opium or morphine often makes the pain worse. Absolute rest in a darkened room is essential. After the acute process is over iodide of potassium may be given in the dose of 10 grains three times a day, with the hope that in this manner inflammatory exudates and adhesions may be absorbed.

THROMBOSIS OF THE VENOUS SINUSES.

Etiology.—Thrombosis of the venous sinuses is due, as are cases of thrombosis elsewhere, to an inflammation of the endothelium which lines these vessels. This inflammation may be the result of a septic infection in remote or in neighboring tissues, as, for example, in suppurative otitis media. Where the cause is sepsis the thrombus usually contains micro-organisms. The vast majority of instances depend upon suppuration in the ear.

Pathology and Morbid Anatomy.—The size of the thrombus varies very greatly. Beginning as a small clot in one sinus, it may gradually increase in size until the sinus is filled, and may even extend to adjacent sinuses and into neighboring veins. If septic in origin it may be purulent in character. The longitudinal sinus is very rarely affected. The lateral sinus is the one most commonly involved, and after it the cavernous sinus.

Symptoms.—The symptoms of thrombosis of the lateral sinus are congestion of the veins in the neighborhood of the mastoid, with swelling of the tissues covering it. There is often *pain* and *tenderness* on pressure. Not rarely the cervical glands are enlarged from infection, and wryneck may be present. *Choked disk* is frequently present upon both sides. With the infection of the sinus there is usually a history of a rigor followed by high fever, with *headache*, *vomiting*, *delirium*, and finally *coma*. The *fever* not rarely follows a septic course, rising and falling sharply. Other *evidences of septicæmia* may also be present, such as *sweating*, *diarrhœa*, and the occurrence of infarctions in such organs as the lungs, spleen, and kidneys.

In thrombosis of the cavernous sinus there is local swelling, congestion of the face about the eyes, epistaxis, and undue fulness of the retinal veins. Occasionally squint develops as a result of interference with the function of the oculomotor and abducens nerve.

Thrombosis of the longitudinal sinus is manifested by intense venous congestion in the scalp, and indeed of the entire head. Choked disk may be present and epistaxis may occur.

Prognosis.—This is unfavorable unless surgical interference gives relief, and surgical interference is practically limited to cases of disease of the lateral sinus.

Treatment.—This is purely surgical, and consists in trephining or otherwise opening both the source of infection and the sinus and thus removing the focus and the clot. For details as to the method of operation, and as to the statistics of recovery following such operations, the reader is referred to books on surgery.

CEREBRAL MENINGITIS.

Definition and Etiology.—By meningitis is meant an inflammation of the membranes covering the brain or spinal cord. From an anatomical standpoint there are three of these—the dura mater, the arachnoid, and the pia mater—but from the standpoint of the clinician and pathologist these membranes may be divided into two parts, the dura mater on the one hand and the arachnoid and pia mater (pia-arachnoid) on the other, for the dura is often inflamed by itself, but the pia and arachnoid are always affected together. When the dura is alone involved it is called pachymeningitis, and when the other membranes are affected it is called leptomeningitis.

Pachymeningitis.—Pachymeningitis may occur in an internal form, when the smooth inner layer of this membrane is inflamed (pachymeningitis interna), and in an external form, in which the outer layer is chiefly affected where it is in contact with the bone (pachymeningitis externa). The latter is the most common type by far.

Pachymeningitis externa is, in the greater proportion of cases, secondary to some traumatism or to disease of the bone. Thus, a blow on the head which fractures the skull, or necrosis of the skull, may so result. Very much more rarely an acute infection arises, as in an infectious and septic malady such as erysipelas.

Pathology and Morbid Anatomy.—The inflammatory process resembles that seen in any inflammation, namely, hyperæmia followed by swelling and cellular infiltration, and this in turn by the formation of pus which is found between the skull and the dura mater, or, in extraordinary cases, between the layers of this membrane. When the inflammatory process does not go on to suppuration, the external layer of the dura becomes thickened and adherent to the skull. If the inflammation is very severe the inner layer of the dura is affected, and the pia mater may become involved and adherent to it.

Symptoms.—These consist in those characteristic of the cause, as the primary unconsciousness from a blow, or the pain of bone disease, and in the development, as direct symptoms, of *headache, confusion of mind, delirium,* and in severe cases *convulsive seizures. Fever* may or may not be present. If pus collects, symptoms of pressure on the brain may develop, and paralysis of the opposite side of the body may ensue (*hemiplegia*).

Diagnosis.—The history of injury, of bone disease, or of some focus of infection makes the diagnosis possible.

Prognosis.—This is bad in direct proportion to the severity of the inflammatory process and the degree to which the pia mater is involved. External pachymeningitis is less grave than leptomeningitis.

Treatment.—This consists in the use of saline purgatives to relieve cranial congestion, in applying an ice-cap to the head, rest in bed in a darkened room to secure perfect quiet, and in the employment of aconite as a cardiovascular sedative if the pulse is excited. If symptoms of cerebral compression develop, as coma or paralysis, the fluid or pus must be evacuated by operation.

Pachymeningitis Interna.—Pachymeningitis interna occurs in a purulent and in a hemorrhagic form, the purulent being very rare. To the hemorrhagic type the terms "hemorrhagic internal pachymeningitis" or "hematoma of the dura mater" are sometimes applied. Even this type is rarely met with, and its existence is rarely recognizable before autopsy. It affects males far more commonly than females (4 to 1, Gowers), and is generally met with after the fiftieth year. It is also met with more commonly in the first twelve months of life than in childhood or early manhood. As a rule, in adults it develops in the course of some form of chronic insanity, particularly in the course of general paralysis of the insane, or in cases of chronic inebriety. Very rarely it has complicated the course of one of the acute infectious diseases, such as typhoid fever or smallpox. In children it may complicate scurvy.

Pathology and Morbid Anatomy.—The exact method or process by which the hemorrhagic extravasation takes place is not known. The autopsy reveals a bilateral, and rarely a unilateral, extravasation between the layers of the dura and between the dura and the arachnoid. Not only is a bloody fluid formed in these spaces, but a pseudomembrane is also present; it may be in several layers. These layers are at first red and later may be pallid, and, by adhering together at spots, form pockets in which the bloody fluid is found. When the condition has existed a long time this fluid may be decolorized and contain crystals of cholesterin. Very rarely suppuration takes place.

Symptoms.—The symptoms are in many cases, if the disease complicates chronic insanity, so suppressed, or absent, that no suspicion of the state just described is harbored. In some cases the patient develops attacks which resemble those of apoplexy, which are supposed to be due to fresh extravasations of blood. In other instances there are signs of cerebral compression, as shown by stupor, or coma, or optic neuritis. Headache and vomiting may be present. Partial hemiplegia may develop.

The prognosis is very unfavorable. Treatment is almost useless. Quiet and rest, with cold to the head, is all that can be done. In cases with strictly localized symptoms surgical intervention is justifiable.

Leptomeningitis.—Leptomeningitis is the form of meningitis which complicates the course of all of the acute infections, notably pneumonia, erysipelas, septicæmia, and less frequently variola, scarlet fever, typhoid fever, and measles. Measles produces it very commonly because this malady is often followed by otitis media, and because otitis media not rarely causes mastoid abscess, and, from this focus, infection involves the meninges or the lateral sinus. It is probable, too, that a very considerable proportion of cases of leptomeningitis are caused by infection which takes place through the nose. It can be readily understood that any infectious micro-organism which can gain access to the meninges through the openings in the skull or in the blood may cause such an inflammation, and, in addition, that the possibility, or probability, of infection is greatly increased by any disease which lowers vital resistance, such as nephritis. It must not be thought, however, that all cases of leptomeningitis due to the pneumococcus are complications of croupous pneumonia, for this micro-organism may produce a meningitis by direct infection, without the lung being affected in the least. It is probable, too, that the typhoid bacillus may, in a case which has long since convalesced from the fever, act in a similar manner. (For the relationship of leptomeningitis to pneumonia and typhoid fever the reader is referred to the articles on those diseases. Also to that on cerebrospinal meningitis.)

Meningitis is more common in children during the first decade of life than at any other period, but in these cases the inflammation usually involves the meninges at the base, whereas in adults that part of the meninges which covers the convexity is chiefly affected.

Morbid Anatomy and Pathology.—The inflammatory process may involve the whole membrane or be quite limited. The limited cases are those which arise from direct infection from a nearby focus of disease. Thus, in cases which are secondary to middle-ear disease the lesion is often unilateral, whereas in those cases in which the pneumococcus is the infecting agent the entire convexity on both sides is usually affected. In the latter type of case the effusion which develops is often large in quantity and purulent. If the cause is tuberculous the base is usually affected. (See Tuberculous Meningitis.) The actual lesions found in the meninges in these cases are noteworthy. Beginning with hyperæmia and congestion they pass on to cloudiness of the membranes affected, which is particularly well marked along the course of the bloodvessels because of the engorgement of the accompanying lymph vessels. Small spots of purulent material are dotted along these vessels which, as they increase in size, coalesce, and so considerable areas are covered by pus. When the process is severe the dura mater and the cerebral cortex may be involved by the inflammatory changes.

Symptoms.—In studying the symptoms of leptomeningitis it must be recalled that the manifestations of involvement of the cortical area are by no means pathognomonic. Every physician of experience has seen cases of typhoid fever or croupous pneumonia present evidences of meningitis, yet the autopsy has revealed no such lesion present. In other words, as pointed out when these diseases were discussed, toxins produced by the specific organisms may cause symptoms identical with those of meningeal inflammation. This is exceedingly common in the pneumonia of children.

There are, however, certain symptoms of meningitis which are certainly indicative of either inflammation or irritation of the meninges, particularly if they are associated with a disease or an injury qualified to produce meningeal involvement. These consist in *fever*, *headache*, *vomiting*, *retraction of the head*, and rarely *convulsions*. *Grinding of the teeth*, *obstinate constipation*, and an *excessive hyperæsthesia* of the skin of the arms and legs are also common symptoms. When the inflammatory process is basilar the symptoms are much more definite and reliable. In addition to those just named we find that *optic neuritis* is present, and *strabismus* and *ptosis*, due to the pressure exercised upon the cranial nerves, develop. The *pupils* may be *contracted* in the early stages because of irritation of the oculomotor nerves, and later *widely dilated* by reason of paralysis of these nerves. Fever may or may not be present, and the pulse is usually slow even if the temperature is raised. *Kernig's sign* may be present. (See Cerebrospinal Meningitis.) A *rapid loss of flesh* takes place in nearly all cases.

Diagnosis.—The symptoms of basilar leptomeningitis, whatever its cause, are usually unmistakable. A very useful aid to diagnosis is lumbar puncture, already described under Cerebrospinal Meningitis. If the cerebrospinal fluid escapes with a spurt from the needle, it is indicative of the presence of tuberculous meningitis, but by no means positive of this condition, for it sometimes happens that a similar high pressure exists in cases of purulent meningitis and of spinal tumor. If disintegrated blood is present in the cerebrospinal fluid it is an indication of the presence of pachymeningitis or injury. Fresh blood, on the other hand, is probably due to the puncture. If the fluid is perfectly clear, every inflammatory affection of the meninges except tuberculosis may be excluded. In tuberculosis it may be clear, but is often cloudy, and toward the end of the case even purulent. The normal proportion of albumin in it is 0.02 to 0.04, and if more than 0.05 is present an inflammatory process is probably going on. If the small quantity of sugar which is normally present is absent, this is a sign that inflammation is present.

Treatment.—Aside from the employment of rest and cold to the head, if fever is present or pain is suffered, we can do little for this condition except we resort to lumbar puncture for the purpose of relieving pressure.

The cerebrospinal fluid may also give us valuable information as to the presence or absence of meningitis, if it be examined microscopically and a quantitative estimation of its leukocytes is made. As a general rule, there is a marked increase in lymphocytes if the inflammatory process is tuberculous, and of polymorphonuclear cells if it is non-tuberculous. Exceptions to this rule occur, and therefore the presence of either one of these forms of leukocytes in increased number is not pathognomonic.

Microscopic examination of the cerebrospinal fluid may also be made for the purpose of discovering tubercle bacilli, or the diplococcus of pneumonia, or other pathogenic micro-organisms. The fluid for this purpose should be kept on ice for not less than twelve hours, until a small clot is formed. The web-like fibres of this clot are transferred to a cover-glass, spread in as thin a film as possible, and stained by the methods commonly employed for staining the tubercle bacillus. Where the examination must be performed at once, the fluid may be put in a centrifuge, and the sediment examined by the staining methods already described. As with examinations of the sputum in suspected tuberculosis, the finding of tubercle bacilli is a positive sign of great value, but the failure to find them by no means proves that the disease is not tuberculous. Reference to the presence of the *Diplococcus intercellularis meningitidis* has already been made in the article upon Cerebrospinal Meningitis. Occasionally the streptococcus and staphylococcus are found. In African "sleeping sickness" trypanosomes have frequently been found in the cerebrospinal fluid.

DEMENTIA PARALYTICA.

Definition.—Dementia paralytica, often called "meningoencephalitis," "paresis," or "general paralysis of the insane," is a state characterized anatomically by a widely diffused process of degeneration in the central nervous system, particularly in the cerebral cortex, with morbid changes in the pia mater. The chief symptoms in the early stages are the development of great irritability of temper, forgetfulness, carelessness as to habits, and later delusions of grandeur. Clinically it is characterized by a progressive paralysis of the body, associated with certain physical signs, and a progressive loss of mental power of a peculiar kind. The most striking symptom of this disease is the "delusion of grandeur."

Etiology.—Without doubt syphilis is a provoking cause in a large proportion of cases (75 per cent.), but it is not present in all, and in no case is it to be regarded as a direct cause, in the sense that the syphilitic virus is active in a given instance. On the contrary, it is, like locomotor ataxia, a parasyphilitic affection, that is, a sequel of that malady, appearing from five to twenty years after the initial lesion. Alcoholism, excessive sexual indulgence, and, indeed, excesses of every kind, are also without doubt factors of importance. The disease is a common one among roués. Some cases seem to follow a sunstroke or severe injury.

The disease is one of the middle period of life, between thirty and fifty years, but a number of cases have been recorded as occurring in children who have usually had hereditary syphilis.

Pathology and Morbid Anatomy.—The primary change in cases of this disease takes place in an increased blood supply to the pia mater and in the smaller vessels of the cerebral cortex, associated with degenerative changes in the bloodvessel walls. The progress of these degenerative changes results in the development of fusiform dilatations of the bloodvessels and the filling of the lymph spaces with serum. The exact cause of these changes is not understood, but Mott believes that they are due, in part at least, to the formation of a toxic substance called choline, which is not only found in the lymph spaces, but diffused through the whole cerebrospinal fluid. This poison causes congestion of the veins of the brain, and affects chiefly those which empty into the longitudinal sinus. Ultimately the quantity of lymph present in the perivascular spaces is so great that a true cerebral œdema is produced. There is an overgrowth of the neuroglia about the vessels, and this newly formed connective tissue sends fibrils down between the cells of the cortex, with the result that a true sclerosis develops. Associated with these connective-tissue changes there are degenerative changes in the cerebral neurones. The body of the neurone undergoes hyaline and then fatty degeneration, pigmentation, and finally atrophy. When the disease has been present for a long time, autopsy reveals the presence of small cysts in the white and gray matter, and so marked a decrease in the size of the convolutions and of the entire brain that it is found to be much smaller than is normal. Its surface is harder than is natural, pigmented, and adherent to the pia mater, which is also found to be the site of overgrowth of connective tissue. The ventricles contain an excess of fluid, and their lining membrane, the ependyma, is thickened. It is noteworthy that the left hemisphere is usually more affected than the right, and that the changes already described affect the frontal lobes and the areas of the motor cortex before the rest of the brain is involved.

In most cases of paresis degeneration is found in the spinal cord, so that spinal symptoms are added to the cerebral signs and form part of the clinical picture of the disease. The lateral and posterior tracts are usually affected, producing symptoms of ataxic paraplegia. Sometimes the posterior columns alone are degenerated, presenting symptoms of locomotor ataxia. Rarely disseminated sclerosis is found; and recently, in one case, the spinal cord in a case of paresis was found to be suffering from syringomyelia. In a few cases of locomotor ataxia (tabes dorsalis), fully developed and typical, the cerebral signs of paresis came on, as if the disease had finally "risen" to the brain. These cases constitute the "ascending type" of paresis.

Some authorities believe that paresis is identical in nature with tabes—that it is a "tabes of the brain." A clinical picture of paretic dementia with tabes is, therefore, met with. Rarely the spinal symptoms precede the cerebral symptoms in the development of the disease. So, too, symptoms of spastic paraplegia may develop. Sometimes autopsy reveals the fact that pachymeningitis and hæmatoma have occurred as the result of the aneurysmal dilatations of the cerebral and pial vessels, already described, leaving behind them an organized membrane beneath the dura, or a mass of encysted blood clot.

Symptoms.—When the symptoms of dementia paralytica are well developed, they are so characteristic and obtrusive that there can be little difficulty in reaching a correct diagnosis in regard to the condition from which the patient is suffering. It is only when the disease is in its stage of onset, or in an atypical form, that any doubt can be present.

As a rule, the early symptoms are recognized in retrospect rather than at the time at which they occur, unless, perchance, these symptoms are very strongly developed. It is noticed that the patient seems to be *nervously fatigued* or *mentally fagged,* and often this condition is ascribed to the excesses which have been committed in connection with venery, wine, and other forms of nervous stress. The *temper* is usually irritable, and the friends notice that the patient takes offence at remarks which ordinarily he would not notice. At times he is remarkably forgetful. Naturally tidy as to his habits and dress, he becomes *careless* and *slovenly.* Occasionally *sleeplessness* will be complained of.

Although, before the onset of the symptoms, he may be apparently kind and faithful to his family, he begins to be brutal in his conduct toward his wife and children, and perhaps returns to the alcoholic and sexual excesses which laid the foundation for his disease many years before. The *speech becomes indistinct,* hesitating, and if the tongue is protruded a very fine tremor may be seen in it. There is also a very marked tremor of the hands. Ataxia of station (Romberg's sign) is a common symptom. The *pupils* are generally unequal and irregular, and the *pupillary light reflex* may be lost, while reaction to accommodation is maintained. In other words, the Argyll-Robertson pupil is present, for the same reason that it is present in cases of locomotor ataxia, because Meynert's decussation, or other fibres, involved in the light reflex arc are affected by the degenerative process already described. The loss of consensual reflex is also frequently observed.

The symptoms of onset are often prolonged over the period of many months, and sometimes for several years, depending upon the rapidity with which the pathological changes in the brain develop. The disease is progressive and ultimately the symptoms of the later stages are developed. These symptoms may consist in *delusions,* which are usually composed of *extravagant ideas.* Thus, the patient may, on the one hand, believe that he is some great historical character, or that he is a ruling potentate, or, again, that he is possessed of fabulous riches. In one instance, for example, within the writer's knowledge, the patient took a room at a prominent hotel, after having provided himself with large sums of money, and from the balcony outside of the room showered the crowd beneath with coins of different values, with the idea that his wealth was limitless. In another instance, the manager of a small plant for making steel became imbued with the idea that his company had obtained and could fill contracts for the delivery of manufactured steel on a scale far beyond those ever attempted by any corporation, although as a matter of fact the business of the concern was at its last ebb, and his delusions aided in causing its final financial collapse, through its inability to carry out the agreements which he made with other concerns. In this instance the stress of business worry combined with previous excess was an active factor in producing the disease.

As the degenerative process in the brain continues, the patient's judgment becomes profoundly impaired. He rarely is capable of continuous thought, and no longer adheres for hours at a time to his delusions. He frequently becomes exceedingly emotional, and laughs and cries without adequate cause. At times he is excessively depressed; at other times exalted, and he may occasionally become frenzied with rage, during which time he may commit some crime.

The handwriting is often characteristic. It may become illegible, either because the letters are badly formed or because important words are dropped out. Still later, loss of power occurs in the limbs until total paralysis may be present. Sensation is not so markedly disturbed, but areas of anæsthesia and analgesia may be found. The reflexes may be markedly increased or entirely lost.

Naturally, there is an impairment of the general health with the progress of the disease. Epileptiform attacks or sudden periods of unconsciousness (apoplectiform attacks) develop, accompanied by paralysis of one limb, or by hemiplegia. Death usually ends the case by the end of the third to the sixth year, the patient dying of exhaustion or of some intercurrent disease, such as pneumonia, obstruction of the bowels, or of one of the epileptiform or apoplectiform attacks.

Cases of paresis in which the delusion of grandeur is prominent are the earliest recognized, and constitute the *classic* form of the disease. There are many, however, in which depression simulating melancholia is present throughout; that is, the *depressed* form of paresis. But in a large proportion of all cases the mental symptoms are mainly those of progressive mental loss, without distinct delusions (the simple or demented form of paresis). An alternation of excitement and depression (circular form of paresis) is observed rarely; delusions similar to those of paranoia or of alcoholic insanity may mask the underlying condition.

Diagnosis.—Dementia paralytica must be separated in its early stages from neurasthenia. The patient suffering from nervous exhaustion usually studies his own symptoms in the greatest detail, and usually considers that he is an ill man, while the paretic has a very much more optimistic view and often insists that he is more than usually well, when it is manifest that his ill health is extreme. So, too, the neurasthenic rarely has complete lapses of memory

and defects of speech. From cerebral syphilis paresis
that the former disease usually manifests severe pai
aphasia due to a syphilitic arteritis in the neighborho
The fine tremor of the tongue and of the hand in p
bral syphilis. Optic neuritis is usually present in
not very common in paresis. So, too, the mental s
depression in syphilis, and not that of excitation. I
paresis is separated by the presence in sclerosis of n
tremor and by the absence of delusions.

Prognosis.—This is absolutely unfavorable. N
although temporary, and it may be prolonged; re
encouragement on the part of the friends, may occu

Treatment.—It must be evident, from the patholo
described, that treatment can do little. In paresis w
tory of syphilitic infection, the iodides and mercury
connection with hot baths. But it must be rememb
the sequence of syphilis rather than the direct res
these remedies are in no sense specific. Their genera
and they may perhaps arrest for the time being the p
changes, but they cannot cure those in existence. If
to control, it is far better both for himself and his fri
committed to an asylum where he can be properly ca
sense of being properly controlled, but of being well f
of health and general nutrition is, of course, of imp
may be treated by any one of the good hypnotics, of
and sulphonal are the best. Occasionally, hyoscin
must be taken that the carelessness of the patient i
does not result in obstinate constipation, which may
For this reason, active purgatives are often necess
excitement comes on, $\frac{1}{100}$ of a grain of hyoscine
dermically.

DISSEMINATED SCLEROSIS

Definition.—Disseminated sclerosis is characterize
of irregularly distributed patches of sclerosis in diffe
and spinal cord. Similar changes also take place
It is sometimes called "insular sclerosis" or "multi
French it is called *sclérose en plaques disseminée*
recognized as a distinct disease for a little more than

Etiology.—The essential cause of multiple sclero
affects both sexes equally, and occurs at all ages,
childhood. Its most common period of existence is, b
tieth to the fortieth year. Occasionally it has been k
the acute infectious diseases, and has been regarde
disseminated myelitis of infectious origin (Marie); but
this malady has any direct·productive effect in the
great cause of organic nervous disease, does not s
present as an etiological factor.

Pathology and Morbid Anatomy.—The lesions of disseminated sclerosis consist, as already stated, in irregularly distributed patches in which the interstitial tissues have undergone sclerotic change. These patches are found chiefly in the white matter of the brain, being comparatively rare in the gray substance of the cortex. They are irregular in outline and vary in size. In appearance they are reddish-gray and translucent. The surface of a patch is usually even with the surrounding brain tissue, but it may be slightly depressed. A sharp line of demarcation separates the diseased area from the healthy tissues. When touched, the patches seem harder than normal gray matter, but they are not usually met with until section of the brain or cord is made, when they appear in strong contrast to the surrounding white tissue. They may be found in considerable number in the lateral ventricle, in the corpus callosum, crura, and pons. The patches in the spinal cord are not so reddish in hue as in the brain, but are more gray in color and extend vertically rather than transversely; but this rule is not absolute, and at times the transverse extension of a patch may embrace the entire thickness of the cord. When one of the cranial nerves is involved, it is found to be gray in color and sclerotic for a certain portion of its length. In some instances the entire thickness of the nerve is involved. The olfactory, optic, oculomotor, trifacial, and facial nerves are the ones which are most commonly affected, and in the case of the optic nerve the favorite seat is the chiasm.

The sclerotic process in this disease does not differ very materially from that met with in sclerotic processes occurring in other organs of the body; there is an overgrowth of the true neuroglia or connective tissue, and side by side with this overgrowth a corresponding atrophy or disappearance of the nerve cells and fibres themselves takes place. When the degenerative process is well advanced, we find scattered through the connective-tissue fatty granules and nerve cells which show evidence of degenerative change. In the nerve fibres the chief change takes place in the myelin, but after the disease is far advanced the axis cylinder also becomes affected and finally is completely destroyed. In the central nervous system it is usually found that secondary degeneration in the nerve fibres above or below the seat of the original sclerotic patch is only met with when the disease has been sufficiently severe to destroy the axis cylinders. On this account secondary degenerations are not common in disseminated sclerosis. the axis cylinders surviving in the midst of fully developed sclerotic patches,

Symptoms.—The symptoms of disseminated sclerosis depend almost entirely upon the areas of the nervous system which are chiefly affected by the pathological changes just described. Among the earliest symptoms in many cases is a *loss of power in the extremities.* In the legs the symptoms may be ataxic, but usually simulate those of spastic paraplegia in a striking degree. When spastic paraplegia is present, this indicates that the sclerotic process has involved the pyramidal tracts, and the knee-jerks are found to be exaggerated. In other instances *inco-ordination of the hands*, or of one hand, may be the first manifestation of the malady, and in still others sensory disturbances in the legs or arms are first complained of by the patient. The inco-ordination of the muscles of the arms is often very marked indeed.

Often it is impossible for a patient to carry a glass of water to his lips without spilling it (intention tremor). The co-ordinated movements which are necessary in writing are impossible because of the quick or spasmodic contractions of the muscles employed for this purpose. The cause of these irregular movements is not known. According to some it is dependent upon the fact that certain fibres are affected, and impulses going along certain channels, particularly the motor, are delayed in passing through sclerotic areas, or that those which pass along fibres still unaffected are made inadequate and, as it were, embarrassed. By others it is thought that the sclerotic patches have involved afferent fibres of the cerebellar system which are concerned with muscular sense.

If sensory fibres in the dorsal columns are involved, areas of *anæsthesia* or *hemianæsthesia*, also ataxia of the extremities, will develop, the severity of these symptoms depending, of course, upon the area involved in the sclerotic process. The eye symptoms are usually well marked. One of the earliest and most constant of these is *nystagmus.* Eyesight fails through involvement of the optic nerve or because of a sclerotic patch at the optic chiasm. As the disease advances pallor of the optic disk, particularly its temporal half, and sometimes optic atrophy can be recognized on ophthalmoscopic examination. One eye is often much more affected than the other, and loss of accommodation may occur. In other instances the pupillary reflex may be lost, yet the pupil will react to accommodation. In other words, the *Argyll-Robertson pupil* is present. The affection of the external ocular muscles, aside from the production of nystagmus, consists most frequently in a failure in convergence and in conjugate deviation. More rarely a single muscle is affected and squint is produced. When the facial nerve is affected, the symptoms in the early stages may consist in *clonic spasm* of the *muscles of the face,* followed eventually by paralysis. The lesions of the cranial nerves, which produce these results, may, as already pointed out, occur in the nerves themselves or involve their nuclei.

Of all the symptoms of disseminated sclerosis perhaps the most characteristic and most frequent is the *peculiar disorder of speech* in which syllables are enunciated in a measured manner. To this mode of speech the term *staccato* or *scanning* is applied. The exact cause of this is not clear. It does not seem to be dependent entirely upon paralysis involving the tongue or the lips.

The mental condition of the patient is usually not materially altered. The *memory* may be slightly impaired and the patient seem somewhat emotional. Very occasionally actual insanity develops. *Paroxysmal attacks of vertigo* and *vomiting* are occasionally met with, and in some cases the patient is seized with attacks which closely resemble an ordinary apoplexy. These attacks, it will be remembered, sometimes develop in patients who suffer from general paralysis of the insane, and they appear either as ordinary coma, as Jacksonian epilepsy, or as a hemiplegia which is fleeting in character. All these symptoms of an apoplectiform type may recur frequently, and are usually recovered from, but occasionally death comes on during coma. It is a fact worthy of note that, notwithstanding the profound changes which take place in different portions of the nervous system, trophic changes in the muscles are rarely met with, even in advanced cases.

Diagnosis.—The most characteristic symptoms of disseminated sclerosis, as just stated, consist in the intention tremor, the staccato speech, the nystagmus, the peculiar jerking, inco-ordinated movements of the muscles of the arms and sometimes of the legs, weakness of the legs, and the gradual involvement of the cranial nerves.

The disease is to be separated from locomotor ataxia by the jerking character of the inco-ordinated movements; and by the exaggeration of the reflexes, which are in contrast to the absent reflexes of ataxia. From paralysis agitans it is separated by the fact that in the latter disease there is a finer tremor of the hand, or of the parts of the body which may be affected (the tremor is passive), by the peculiar attitude of the patient in paralysis agitans, and by the absence, as the disease progresses, of the characteristic symptoms just spoken of as peculiar to multiple sclerosis. General paralysis of the insane is distinguished by the presence, in the classic type of this disease, of delusions of grandeur, by the twitching of the muscles of the lips and tongue, which are more constant and severe than they are in multiple sclerosis, and by the other evidences of mental change. In a case of disseminated sclerosis, in which the lateral columns of the spinal cord are involved, it may be difficult to differentiate multiple sclerosis from spastic paraplegia, but as the disease progresses the development of the other symptoms of disseminated sclerosis makes the diagnosis easy. From hysteria disseminated sclerosis is to be separated by the fact that nystagmus does not appear in this functional nervous disorder, and by the inconstancy of the symptoms in many cases of hysteria. The other characteristic stigmata of hysteria may also be found. (See article on Hysteria.)

Prognosis.—The prognosis in a case of disseminated sclerosis is absolutely unfavorable as to ultimate recovery. The disease is characterized by various remissions or periods of arrest, so that death is sometimes postponed for a considerable period of time. The prognosis as to duration of life is worse in those cases in which the lesions involve nervous tissues closely associated with vital functions, as when sclerotic changes take place in the pons, or, above all, when they occur in the medulla. It is noteworthy that pregnancy or trauma increases the rapidity with which the disease progresses.

Treatment.—Everywhere in this book, when we have considered the treatment of diseases depending upon sclerotic changes or overgrowth of connective tissue it has been pointed out that our therapeutic resources are inadequate. We do not know the causes of this connective-tissue overgrowth, and so are unable to combat it, nor do we know why the cells degenerate. Neither have we any reason to believe that in the future we will discover any remedy which will cause the absorption of connective tissue when it is once formed, and it is certain that fibres which have once degenerated and have been destroyed cannot be regenerated by the action of any medicine.

DISEASES IN WHICH THE CHIEF MANIFEST THE SPINAL CORD.

LOCOMOTOR ATAXIA.

Definition.—Locomotor ataxia is a disease character
ordination of gait and station, loss of muscle sense, a
reflexes. It is often accompanied by pain. The mos
reflexes is in the patellar tendon and iris. Pathologicall
by slow progressive lesions which affect chiefly the sen
the posterior or dorsal columns of the spinal cord. I
tabes dorsalis, or posterior spinal sclerosis.

History.—Cases of locomotor ataxia were recorded as
many years ago, but it was not till 1847 that Todd cl
malady from other states of paralysis. In 1840 Stanley
the affection was associated with changes in the poste
spinal cord. In 1855 Reynolds first showed that the di
a state in which the symptoms were due to a loss of mus
loss of power in the motor nerves or muscles—a view
who made a microscopic demonstration of the site of the

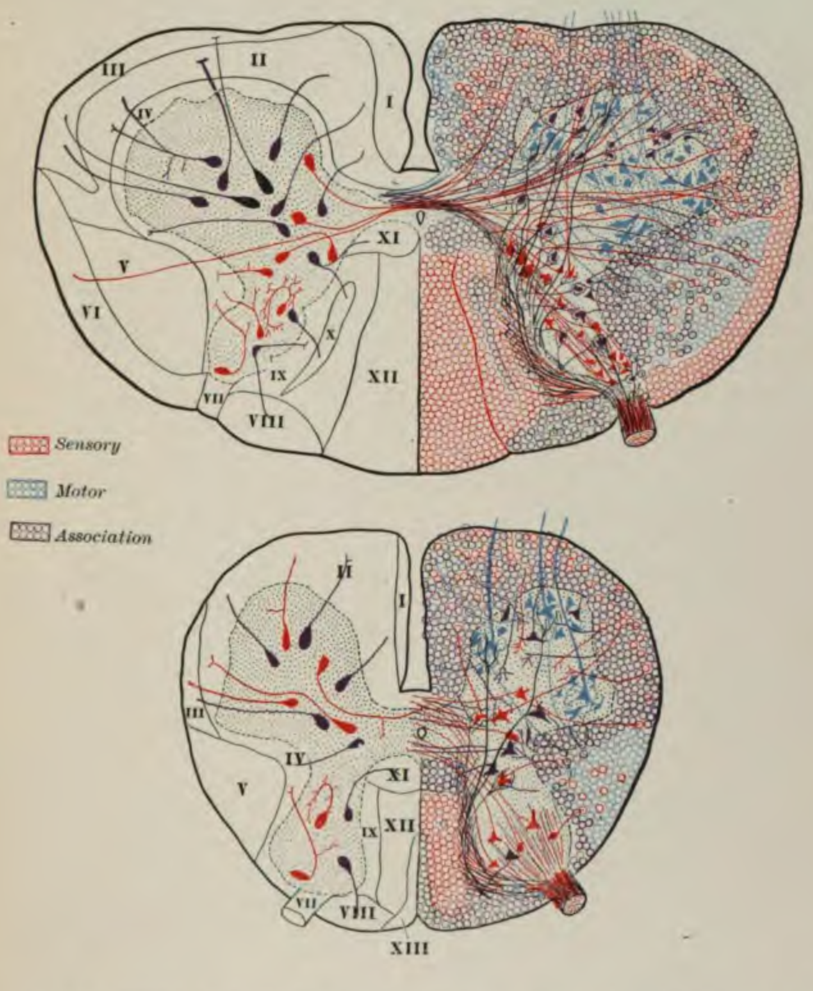
Etiology.—Males suffer more than females in the pr
In a large hospital experience of over twenty years I ha
in a woman. Half the cases develop in the decade of lif
forty years, and 80 per cent. between thirty and fifty ye
that it rarely develops after fifty years.

By far the most common cause of the disease is acqui
the syphilis is hereditary. Some writers have gone so
locomotor ataxia is due to this cause in over 90 per cent.
Gowers gives the rather moderate proportion of 77 pe
cent. It is, however, important for the student to reme
ataxia is not syphilis of the nervous system, but rather a
syphilitic infection. In many instances the ataxic sympt
years after syphilitic infection that the patient cannot
maladies have any relationship of cause and effect, the mor
frequently met with in patients who have presented v
symptoms than in those who have had severe symptom
Rarely the ataxic symptoms develop within five years o

Next to syphilis as a possible cause is traumatism. S
upon the spine, have been followed, months or years late
but it is impossible to tell whether the trauma is the
malady. Sometimes other diseases of the cord finally
ataxia.

Pathology and Morbid Anatomy.—In studying the m
pathology of locomotor ataxia it is important for the s
fact that the primary lesion of the disease is in the p
posterior roots of the spinal cord, and in the ganglia of
and not in the posterior columns of the spinal cord, as

PLATE VII.



The Cervical and Sacral Enlargements of the Spinal Cord in Cross-section—showing the various neurones in the gray matter, the direction of their axones, and the varieties of fibres in the different columns of the cord (Starr). Blue, motor-; red, sensory-; purple, association-neurones and axones.

I. Ant. median column. II. Anterolateral column. III. Gowers' anterolateral ascending column. IV. Marginal column. V. Lateral pyramidal column. VI. Direct cerebellar column. VII. Lissauer's tract. VIII. Ext. portion of column of Burdach. IX. Root zone of the column of Burdach. X. Descending comma-shaped bundle of Schultze. XI. Post. commissural tract. XII. Column of Goll. XIII. Septomarginal tract.

time. The sensory cells in the posterior ganglia, outside of the cord, are flask-shaped bodies, each of which has a process which divides into two axones. One of these goes by the posterior nerve root to the spinal cord, and the other goes to the afferent nerve, which extends to the peripheral portions of the body. (See Plate VII.) Degenerative changes take place in the proximal axone as well as in the peripheral portion of the distal axone. These changes are sclerotic and are carried into the cord, so to speak, by the proximal axone. Therefore, locomotor ataxia consists primarily in disease of the proximal axone, secondarily in disease of the distal axone, and finally in disease of those fibres in the posterior portion of the spinal cord which have their origin in the proximal axone just described. While it is true that the primary lesion is not in the cord, it is nevertheless a fact that the chief manifestations of the pathological process are to be found in this portion of the nervous system. Indeed, the changes in the spinal cord are so well developed in typical cases that the macroscopic examination suffices for the diagnosis at the autopsy.

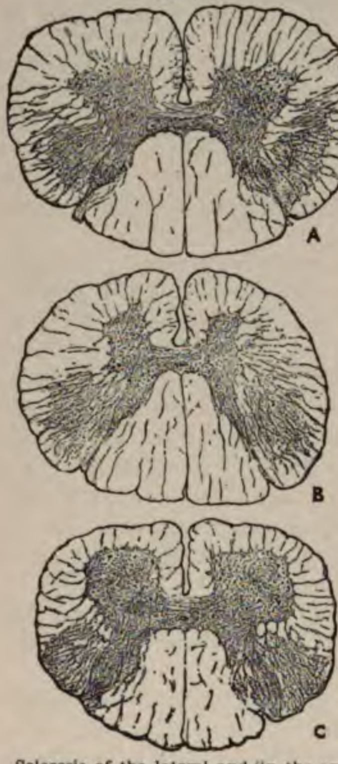
The affected portions of the cord are smaller than normal and more grayish in appearance. The distribution of those areas varies greatly. In some instances the disease is so moderate in degree and in distribution as to be difficult of recognition, except by the microscope. In others, if the malady is far advanced, the whole length of the posterior columns may be affected. There are certain parts of the cord which are particularly prone to the development of the disease. Thus, the lesions are usually well developed in the posterior columns in the lumbar region, particularly in the neighborhood of the posterior root zones, and this accounts for the fact that the legs show the earliest and most severe symptoms. As we ascend the cord, however, the lesions are chiefly found in the posteromedian columns.

In instances in which the disease affects the arms as well as the legs and is well developed, the posteroexternal columns are affected even in the cervical region (Fig. 124).

Under the microscope it is found that the connective tissue in the posterior columns of the cord has undergone hyperplasia or overgrowth. The fibrous sheath of the bloodvessels is particularly affected and is seen to be thickened and to project connective-tissue fibrils into nearby parts. The nerve fibres may have disappeared entirely or be represented by atrophied or wasted fibres. In some cases the vessels of the pia mater are also thickened, particularly in the part covering the posterior columns, and these vessels may also give off fibrils of connective tissue which add to the connective-tissue overgrowth in the superficial part of the cord.

The changes which are found in the posterior nerve roots vary greatly in their degree. In some instances they are so slight that they can be recognized only by careful microscopic examination; in other instances they are so well marked that the naked eye can detect them. Under these circumstances they appear atrophied and the connective-tissue elements may be somewhat increased. The root fibres in the cord are more affected than those outside of the cord. The ganglia are also affected by an overgrowth of connective tissue, and by an atrophy of their nerve cells. Beyond the ganglia pathological changes in the mixed nerve are rarely seen as a continuation of the process found in the nerve roots, but it is a well-recognized fact that marked primary changes are to be found in the peripheral nerves, and especially in the cutaneous branches. These changes consist in anatrophy of the myelin sheath followed by degeneration and segmentation of the axis cylinder, and they occur at the distal extremity of the nerve in greater degree than higher up. The main nerve trunks are rarely affected. It is largely because of these neural lesions that sensation in the skin and in the joints and the muscular sense are lost.

FIG. 124



Sclerosis of the lateral and (in the anterior cervical region) pyramidal tracts, with slight degeneration of the anterior coruna. A, cervical; B, dorsal; C, lumbar sections. (After Gowers.)

In certain cases the changes of locomotor ataxia may be well marked in the cord and slight in the nerve roots, and in others the nerves are chiefly involved. It is important that this fact be remembered because it serves to fix in the mind of the student the fact that locomotor ataxia is not solely a disease of the posterior columns of the cord.

While locomotor ataxia chiefly affects the spinal cord and its nerve roots, it also attacks quite frequently the cranial nerves, and of these the optic nerve suffers most severely and most frequently. Its nerve fibres atrophy and its connective tissue undergoes proliferation.

By no means rarely marked degenerative changes are found in the joints. The articulating cartilages are eroded, the joint may become filled with fluid, and for these reasons dislocations may occur. Trophic changes also occur elsewhere. (See Symptoms.)

Symptoms.—In a case of locomotor ataxia which is typical in its course the following symptoms are present, and if they are well developed it is not difficult to make a diagnosis:

The patient often states that his *feet feel "muffled,"* that is, as if he had on several pairs of thick socks. In other cases he notices that on arising at night he has difficulty in getting a proper "purchase" with his feet on the floor, or the floor may feel as if its plane is at a different angle from that pictured in his mind. These awkward sensations are due to the interference with the sensory nerve fibres, that is, with conduction of sensation to the spinal cord. He also has difficulty in walking in the dark, not only because his tactile sense is disturbed, but because his muscle sense is also impaired, with the result that he is in doubt as to the position of his limbs and as to the relative tonicity of opposing muscles. This loss of muscle sense depends upon the fact that the sensory nerves supplying the joints, tendons, and fasciæ are impaired in function and the tracts in the spinal cord which carry these impulses which reflexly co-ordinate movement are also involved. If the impulse passes this area of damage and reaches the cord it passes up the posteromedian columns without decussation, and probably goes directly to the cerebellum, which presides over balance. These columns are always diseased if the ataxia is marked, but cutaneous sensibility is often preserved, proving that the muscular and cutaneous sensations are carried by different tracts.

Closely related to the disorder of muscle sense is *inco-ordination*, which produces the *peculiar gait*. In the ataxic gait the foot is raised awkwardly and then thrown down in front of the other foot with a characteristic uncertain movement, the whole under surface of the foot striking the ground at once. This uncertainty of movement becomes still more marked if the surface over which the patient has to walk is uneven, or if a rug upon the floor requires that the feet shall be lifted slightly to clear it. In other cases the edge of the foot rests on the floor instead of the plantar surface, and in the effort to correct this position another clumsy movement is made. If the light is poor or if the patient closes his eyes, the difficulty in muscular co-ordination may be so great that he staggers and falls. As the involvement of the sensory pathways becomes more marked, support by means of a cane, nearby objects, or another person, is needful for locomotion, and finally all attempts at walking have to be given up. In cases in which the upper portions of the cord suffer, there is a similar inco-ordination of the arms, so that the patient cannot carry food to his mouth if the eyes are closed. In other instances, however, even when the legs are practically useless, the arms entirely escape. If either the arms or the legs are extended the muscles do not remain steady, but alternately contract and relax, as the patient vainly endeavors to maintain his balance. There is no actual loss of muscle strength until the disease has lasted so long that the muscles waste from disuse.

The same cause that produces the difficulty in gait also causes a *disturbance of "station."* That is to say, the patient cannot stand steadily, but sways in the endeavor to keep his balance. If his eyes are closed or if he is blindfolded he sways so widely that there is danger of his falling, and he may actually fall if he cannot co-ordinate his muscles by the use of his eyes, which will give him a conception of the relative position of surrounding objects. This instability is often very marked when the patient attempts to suddenly assume the erect posture after sitting in a chair for some time. As Romberg first called attention to this loss of station, the term "*Romberg's symptom*" is applied to this manifestation of the malady.

Still another indication of the disease in the sensory pathways in locomotor ataxia is the diminution and final *total loss of the knee-jerks* when the patellar tendon is tapped. This is called "*Westphal's symptom.*"

So far only those disorders of motion which result from the loss of muscle sense have been discussed. There yet remain to be considered the characteristic sensory symptoms themselves. These consist in *pain* and *loss of sensibility*. Pain is a very frequent and often a very early symptom, occurring in about 90 per cent. of all cases, and it may be very severe. It occurs chiefly in the legs as sharp dartings called "lightning pains." Unlike the motor symptoms, these pains are not confined to the legs and arms, but are often present in the body and even in the head, where other symptoms of this disease are rare, except in the eyes. The pains are often agonizing and occur chiefly at night. In most instances they occur in periodic attacks, then ceasing for weeks or even months. They may develop in different parts of the body at each attack. They rarely have their seat in large nerve trunks, but exist in the more minute fibres of the nerves. When they attack the stomach they are called "*gastric crises,*" a term also applied to severe attacks of vomiting in this disease. So, too, attacks of intense pain may suddenly develop in the bladder, "*vesical crises,*" and in the rectum, "*rectal crises.*" There may be marked "*girdle sensations*" in the trunk.

The *pains* are described as *darting, rending,* or *burning,* and the patient may speak of "burning toes" as his most troublesome symptom. *Intense hyperæsthesia of the skin* in the painful areas may also be present. These pains are to some extent affected by atmospheric states. I have more than once known them to be produced or exaggerated by constipation, probably because of the absorption of intestinal toxins.

It is important to bear in mind the fact that pain may be one of the earliest signs of this malady, and may vary from tingling to an agony without any of the disorders of the gait being as yet present. On the other hand, very severe ataxia may be present without any pain. In addition to these painful disorders various other disturbances of sensation also develop, such as *formication, tickling, pricking, creeping, sensations* of heat and cold, or hyperæsthesia. When the disease is well advanced, diminution of sensation in the skin or even *complete anæsthesia* may appear. The sense of pain and of touch may both be impaired. The pain sense may be lost and the heat sense retained, or, again, the tactile sense may be interpreted by the patient as pain or heat. A very interesting perversion of the function of sensation is the delay in the transmission of the sensory impulses, so that a very appreciable interval occurs between the moment at which the foot is pricked and the moment at which the patient appreciates the fact that the injury has been sustained. Obersteiner has recorded a case in which the interval was twenty-five seconds. So, too, the patient is unable to readily indicate the part touched. He may even assert that it is the left foot when it is really the right one that is irritated. To this symptom the term *allochiria* is applied. All these tests must be made, of course, with the patient blindfolded. The deeper portions of the body may be as anæsthetic as the skin, and injury to a testicle, pleurisy, and severe muscular inflammation, as after a deep injection of mercury, may be painless. Sexual power may or may not be lost.

The *changes* which take place in the *eyes* in locomotor ataxia are so constantly met with and are so valuable to us from a diagnostic standpoint, that they are worth remembering. In about 80 per cent. of the cases the *Argyll-Robertson pupil* is present, that is, the pupil reacts to accommodation, but not to light. This state depends upon a lesion, somewhere in the path of the light reflex, which includes the optic nerve on the one hand and the oculomotor nerve and nucleus on the other, with a connection between these two nerves which is not known. Some have taught that Meynert's decussation, between the primary optic centres in which the optic nerve ends and the third nerve nucleus, forms this connection and is the seat of lesion determining the Argyll-Robertson pupil. Recent studies indicate that the fibres concerned pass from the optic tract to the third nerve nucleus before the former has reached the primary optic centres.

There may be loss of accommodation in some cases. The pupils are usually myotic; they may be unequal and uneven, especially during contraction; they may also be irregular in shape.

The second important ocular symptoms are those which depend upon the nerve supply of the extrinsic muscles of the eye. *Diplopia* may develop as a fleeting or permanent symptom due to insufficiency of one of the ocular muscles, the external rectus muscle being the one most commonly affected, although there is diversity of opinion as to this point. So, too, single or double ptosis may develop and be transient or permanent. In some cases all the extraocular muscles become paralyzed so that a *complete external ophthalmoplegia* may be present. The third ocular sign of importance is *atrophy of the optic nerve,* which takes place in about 10 per cent. of all cases. It is often present before any difficulty of the gait develops, and for this reason the presence of the disease may be first recognized by the ophthalmologist rather than by the general practitioner. It is thought by some writers that this manifestation of locomotor ataxia is more prone to develop in the instances in which the arms are involved than in those cases in which the lower portions of the cord are affected. Not rarely the presence of *optic nerve atrophy* seems to be accompanied by an arrest of the sclerotic process elsewhere. The field of vision is primarily diminished, there may be loss of color vision, but sometimes the failure is marked, even from the onset, in the neighborhood of the macula. The impairment of vision which ensues may progress to *total blindness* or become arrested and consist in more or less severe impairment. Usually the process is slow, but occasionally it is so rapid that even a few days produce great changes in the visual acuity. As is easily understood, when we consider the nature of the lesions which are characteristic of the disease the loss of vision is not always unilateral.

When the optic nerve is examined by the ophthalmoscope in such cases the disk is seen to be pale and shrunken, but at times the degree of blindness is in excess of the changes in the disk. Finally, the disk becomes a pale gray.

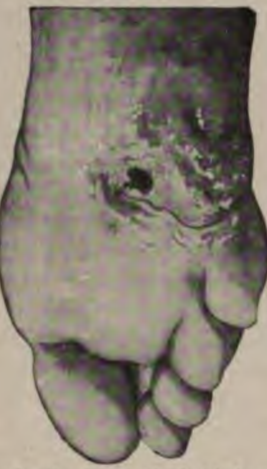
Occasionally *deafness* gradually or suddenly developes. It may be transient or fleeting.

The bladder in locomotor ataxia is often greatly impaired in its functions. The urine is often imperfectly expelled and as a result residual urine produces *cystitis.* More rarely *retention of urine* ensues. The sphincter ani is also weakened, and so control of the feces is diminished.

There still remain to be considered two results of the disease which are of interest and diagnostic importance. The first of these is the so-called "*Charcot joint,*" to which reference has already been made. Owing to the changes in the elbow, shoulder, hip, and knees the landmarks of these parts may be completely obliterated, and great swelling often is present. The second of these trophic changes is the so-called *perforating ulcer of the foot*, which may or may not be accompanied by ulcerations about the toe-nails.

Diagnosis.—As already intimated, the most valuable diagnostic symptoms and signs of locomotor ataxia are the loss of the knee-jerk, the swaying station, the Argyll-Robertson pupil, the optic atrophy, and the lightning pains. No one of these, however, enables us to make a diagnosis because of its presence. The disease must be differentiated from peripheral neuritis due to alcohol, lead, and arsenic, and from that due to typhoid fever and diphtheria. In these conditions there is loss of knee-jerk, swaying station, and often severe pains or anæsthesia it may be, but the Argyll-Robertson pupil is absent and the history of the patient as to exposure to alcohol, lead, or arsenic aids us greatly in the differentiation. To these states the term pseudotabes has been well applied.

Fig. 125



Perforating ulcer of the foot in locomotor ataxia. (Obersteiner.)

Locomotor ataxia is separated from the various forms of paraplegia by the persistence of the knee-jerk, which is usually exaggerated, and by the actual loss of power in paraplegia. From the spastic paraplegia due to lateral sclerosis true locomotor ataxia is separated by the spastic state of the muscles and the greatly increased knee-jerk.

From general paralysis of the insane locomotor ataxia may be difficult of separatoin, for in this disease the Argyll-Robertson pupil and other physical signs of locomotor ataxia may be present. As the case advances the predominance of the cerebral symptoms over the spinal symptoms becomes marked and so renders the diagnosis possible. (See Paretic Dementia.)

The fact that in rare cases of locomotor ataxia severe pains are felt in the trunk should never be forgotten, for it has happened not infrequently that they have misled the physician into a belief that caries of the vertebræ was present.

The staggering gait of cerebellar tumor can scarcely be mistaken for locomotor ataxia. If there is doubt as to its cause it can be dispelled by the absence of shooting pains, by the presence of headache and of nystagmus.

Prognosis.—The prognosis of locomotor ataxia may be best considered in two parts. So far as recovery is concerned, this is out of the question. So far as rapidity of progress is concerned, we must always be guarded in expressing an opinion. In the great majority of cases the disease lasts for years and is characterized not only by periods of rest, but of actual improvement of a very marked character in some cases. If there is a syphilitic history the use of the proper specific remedies may arrest the malady, but they cannot cure the damage already done. Cases which attack those young in years and progress rapidly are most unfavorable, but even these cases make remarkable "stops" in the advance of the affection.

Treatment.—There is probably no grave disease of the nervous system of an organic nature which, in some instances at least, yields such good results from treatment as does this one.

As may be gathered from the discussion of the pathology of locomotor ataxia, it must be evident that the physician can only palliate the symptoms of this disease, and that a complete cure is practically impossible. The most that we can do is to attempt to eliminate from the system the causes which are primarily active in the production of the characteristic lesions and so prevent further progress of the malady. What these poisons are we do not know, for, as already pointed out, even in those cases in which there is a distinct history of syphilis, it is probable that the cord disease develops rather as a parasyphilitic condition than one depending directly upon syphilis. Nevertheless, in a large proportion of cases, benefit is obtained by the pursuance of a plan of treatment which consists largely in the administration of mercury and the iodide of potassium. If the specific infection is of comparatively recent date, the mercury should be used, but in many instances this is not the case, and the iodide of potassium is the remedy of choice. The rule governing its administration, under these circumstances, is to give that quantity which the patient can take without seriously impairing his digestive functions and general health. It is a question of the effect produced rather than one of grains of drug administered. Some patients can take very large doses without symptoms of iodism; whereas, others are affected even by moderate doses. The drug should be given in ascending doses until its full physiological effects are manifested, and then the dose is to be cut down one-third or one-half, and continued for a number of months. Very often the best results are obtained if in addition inunctions with mercurial ointment are practised twice or thrice a week.

While, on the one hand, it should be our endeavor to use these two specific remedies very freely, it must also be remembered that the patient's vitality must be kept at the highest possible level by every means in our power. Poor health and digestive disturbance produced by the unwise employment of mercury or iodine probably does the patient more harm than the drugs do him good. Often it is advisable to give iodide of potassium for three months, and then to use mercury for three months. Starr asserts that the use of mercury hastens the process of optic nerve atrophy, and in those cases in which this symptom is present mercurial treatment should not be resorted to. In those cases in which there is no syphilitic history, or in which the mercurials are badly borne, arsenic may be given as a nerve tonic. Nitrate of silver was at one time thought to be advantageous, but there is nothing in our knowledge of this drug or of the disease which makes its employment in any way rational.

For the relief of the pains in the peripheral nerves, the coal-tar products are our best remedies, acetanilid, phenacetin, and antipyrin being commonly employed. In other instances the salicylates are useful. If the pain is excessive, morphine must be used.

For the twitching of the limbs, the bromides, which quiet the sensory portions of the cord, may be employed in sufficiently large doses to produce sedation.

A method of treating these cases which is of some value is that introduced by Fraenkel, of Berlin. This method is not curative in the sense that it is supposed to influence the lesions in the cord, but is employed with the object of training other nerve fibres than those originally used, so that the patient may to some extent regain his muscle sense. This plan consists in making him take certain exercises which require co-ordination. A chalk line is drawn upon the floor and he is required to follow it as closely as possible; or, a series of cup-like depressions are made in a plank, which is placed across the foot of the patient's bed. These depressions are numbered from one to ten, and he is instructed by the nurse to raise his leg and then rest his heel in the cup which she names. In this way the patient in some instances is able to speedily respond to the order, and so is trained to carry out well co-ordinated movements. Still another method consists in supplying him with a small double flight of steps provided with railings so that he cannot fall. The patient is then required to mount a few steps on one side and then descend a few on the other, using his legs to lift himself up on each step, and not pulling himself up by his hands, which rest upon the rails. In other instances still, definite spaces are marked out on the floor, and he is directed to take a stride which will bring his heel on each mark. It can be readily seen that a large number of such exercises can easily be devised if a little ingenuity is exercised. Care should be taken that the exercises are not continued so long that the patient becomes in the slightest degree exhausted. For this reason they should rarely be continued more than five minutes at a time, although they may be resorted to several times a day. Additional methods of treatment consist in the employment of massage, which is designed to maintain the nutrition of the limbs and to keep in health the bloodvessels and lymphatic system, thereby to a certain extent compensating for the lack of exercise from which the patient inevitably suffers.

The various forms of baths at home, or at health resorts, may be employed rather for the mental effect which they will exercise upon the patient than with any hope that they would be in any way curative. The great advantage in resorting to the various health resorts where baths can be obtained is that the patient goes away for the purpose of getting well and leaves his business cares behind him. The great difficulty with cool baths is that the patient usually has not sufficient power of reaction to stand them, and tepid and hot baths often seem to exercise an enervating effect. Where baths can be used with the object of aiding in the absorption of mercury and the iodides, and where they do not produce depression, they are valuable.

So far as exercise is concerned, this should be governed entirely by the strength of the patient. Under no circumstances whatever should he be permitted to become exhausted. Not infrequently severe attacks of pains in the limbs are precipitated by exercise which is sufficiently severe or prolonged to diminish the nervous vitality of the patient or to tire the nerves themselves.

Electricity may be used in the form of the galvanic current, the positive pole of the galvanic battery being placed at the nape of the neck and the negative pole at the sacrum and at the soles of the feet. The electrodes should be large so that the current will be well diffused. As a matter of fact the condition of the spinal cord and the nerve trunks is such that little real benefit can be expected from this plan of treatment except for its sedative influence.

Some years ago a method of treating locomotor ataxia by suspension was introduced. A harness was attached to the patient's head and shoulders, and by this means he was gently swung free from the chair in which he was sitting. Under these circumstances, the weight of the lower portion of his body produced some extension of the spine, and it was claimed that in this manner marked benefits were produced. Hundreds of cases were treated by this method, but it is worthy of note that it has now gone out of fashion, proving that it does not possess the value which at first was credited to it.

FRIEDREICH'S ATAXIA.

Definition.—Under this name a disease of the nervous system is rarely met with which is hereditary and which depends for its clinical manifestations upon lesions in the posterior and lateral columns of the spinal cord. It is, therefore, an ataxic paraplegia which is peculiar in that it develops in early life. Friedreich's ataxia is also called "hereditary ataxic paraplegia," "hereditary ataxia," "Friedreich's disease," and "family ataxia."

History.—The malady was first described by Friedreich in 1861 and again in 1876. In the United States the most noteworthy study is that of Everett Smith in 1885. Schultze showed in 1877 that the disease was due to a congenital defect in the cord.

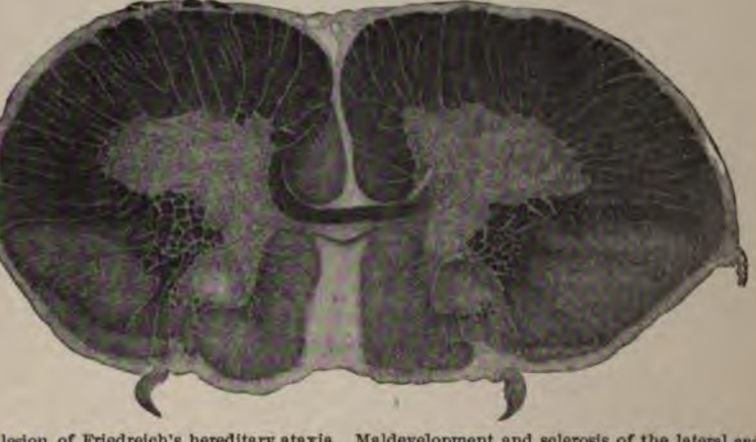
Etiology.—The exact cause is unknown. Occasionally there is a distinct family history of the disease, but often no more than one child in a family is affected. Sometimes the symptoms develop after one of the acute infectious diseases of childhood, and it is then supposed to be due to development of the evidences of imperfect growth or to damage to poorly vitalized cells which have never become well developed. In some cases the parents have an alcoholic history; in others there is a history of syphilis. Neither of these facts are, however, of real etiological importance. The influence of age is uncertain. Rarely the malady manifests itself in infancy; more commonly it develops about the sixth or eighth year; if not at this period, then at puberty, and if not at puberty, then at about twenty-one years of age. The two sexes suffer about equally. It has been shown that defective development of the cerebellum may be a part of the pathological findings in this disease. Marie, however, believes that spinal cord atrophy in these areas, when due to cerebellar disease, is a separate malady, and the symptoms due to agenesis of the cerebellum are sufficiently distinctive to constitute a separate type of hereditary ataxia.

Pathology and Morbid Anatomy.—As already stated, the lesions of Friedreich's ataxia are chiefly found in the posterior and lateral tracts of the spinal cord, and the disease may therefore be considered as a combination of two maladies so far as the lesions and symptoms are concerned.

When the spinal cord is removed from such a case at autopsy it is usually seen to be smaller than normal, and the pia mater is commonly thickened, particularly over its posterior surface.

If the cord is examined under the microscope with suitable staining (Fig. 126), it is found that the posterior dorsal columns, particularly those of Goll, the lateral pyramidal tracts, and the direct cerebellar tracts all show degenerative changes. These changes are not chiefly limited to one portion of the cord, as they are in most cases of locomotor ataxia, but extend up into the cervical region as well as in the lumbar region. The lesions are not only posterior and lateral, but anterior as well, for the direct pyramidal tract on either side of the anterior median fissure is affected. There is also atrophy of the cells in the anterior and posterior horns of the gray matter. The anterior and posterior nerve roots are also atrophied. The cells in the column of Clarke are markedly degenerated, and round-cell infiltration is present about the central canal of the cord. In this disease, as in other maladies, the loss of nervous tissue is followed by overgrowth of the neuroglia in the affected parts.

FIG. 126



The lesion of Friedreich's hereditary ataxia. Maldevelopment and sclerosis of the lateral and posterior columns. (Schultze.)

Symptoms.—As Friedreich's ataxia consists, pathologically, in lesions in the posterior and lateral columns of the cord, it necessarily follows that the symptoms are closely allied to locomotor ataxia and lateral sclerosis. The onset of the disease is characterized by *gradual loss of co-ordination,* affecting the legs before it affects the arms, which causes unsteadiness in station, so that the feet, when the patient is standing, are placed far apart to maintain the balance of the body. In some instances the first symptom is that the child falls over objects which hitherto have not been obstacles in its path. When the child walks its *gait is tottering,* and if it closes its eyes the lack of co-ordination and consequent instability is so great that it may fall. The muscles of the legs are often strongly contracted in an endeavor to maintain the upright posture, and this condition of muscular rigidity is increased by the disease in the lateral tracts. The child if stripped and left standing is seen to be continually writhing in an endeavor to adjust opposing muscles in order to maintain its equilibrium. The *knee-jerks* are *lost,* but cases are occasionally met with in which the reflexes are exaggerated. These cases closely approximate the group called hereditary cerebellar ataxia. (See below.)

Loss of power is not as early a symptom as is *inco-ordination.* It affects the legs far more severely than the arms. The extensors suffer less than the flexors, and this may place the feet in a posture like that of talipes equinus or varus. This deformity may also be caused not only by one group of muscles overcoming others by reason of their loss of power, but by the fact that if the lesions in the lateral columns of the cord predominate, a spastic paraplegia develops which may result in contractures as in ordinary ataxic paraplegia. In those instances in which the muscles of the trunk become affected curvature of the spine may develop.

The mind is not affected by the disease, but nevertheless the patient rarely develops mentally as does the normal child.

When the disease is well advanced, the movements of the lower and upper limbs become not only irregular from inco-ordination, but jerking in character, and this *jerking movement* may extend to the head and be accompanied by *tremor. Speech becomes impaired,* the words are blurred because of imperfect articulation, and the utterance may be sudden or explosive. The disorder of speech is a late symptom of the malady, and may not appear for some years after the ataxic manifestations appear. When the eyes are moved laterally or upward nystagmus may be present, and it is peculiar in that it is absent when the eyeballs are at rest. The extraocular muscles are rarely paralyzed, and the optic nerves always escape. In these respects, therefore, the disease differs very distinctly from locomotor ataxia, in which malady these parts are commonly involved. Occasionally, cases are met with in which the pupillary reflex is lost. In these cases, however, syphilis is the cause, and the case is probably one of tabes with Argyll-Robertson pupils.

The disease is usually characterized by an absence of all disturbances of sensation save that *cramp-like contractions* of the muscles in the early stages may cause the patient some *suffering.* In rare instances *severe darting pains* have been met with, or the patient has experienced numbness in the limbs. The symptoms of ataxia are usually made worse by prolonged rest.

Diagnosis.—The development of the characteristic symptoms just enumerated during the period of childhood renders the diagnosis easy, for the maladies which resemble Friedreich's ataxia are all of them affections of adult life, save multiple neuritis, which may cause, of course, pseudotabes and a disturbance of station and gait. From Marie's cerebellar hereditary ataxia Friedreich's ataxia can be separated by a study of the symptoms of that affection described below.

It must be recalled, however, that cases of Friedreich's ataxia develop which present symptoms which do not follow characteristic lines. Thus, in some cases great muscular atrophy has occurred. Nystagmus may not appear. Diplopia may be present.

Prognosis.—The prognosis is, of course, hopeless. The only thing favorable which can be said is that the disease often develops very slowly and has long periods of arrest. The child, if attacked early in life, rarely reaches adult years.

Treatment.—Treatment, aside from that devoted to the maintenance of good nutrition, is of little avail, for obvious reasons.

Marie's Cerebellar Hereditary Ataxia.—Under this name a form of hereditary ataxia has been described by Marie in which he has shown that a congenital defect exists in the cerebellum. The condition is characterized by ataxia, difficulty in speech, and nystagmus, and in these points resembles Friedreich's ataxia. It differs, however, in the presence of defective pupillary reaction and various ocular palsies with optic atrophy and exaggeration of the knee-jerks. Further, it develops in the third decade of life, whereas Friedreich's ataxia nearly always appears before the fourteenth year.

L. F Barker has recently put the matter thus: The direct cerebellar tracts of the cord which are degenerated in Friedreich's disease end in the middle lobe of the cerebellum, which is defective in Marie's type. The ataxia of both these diseases therefore results from lesions of different parts of one system. In Barker's nomenclature the spinal part of the posterior spinocerebellar system is affected in Friedreich's ataxia, while the cerebellar part of it is involved in Marie's type. An analysis of the symptoms in all the reported cases of Marie's disease has led H. T. Patrick to say that increase of knee-jerk is its sole distinguishing feature from Friedreich's ataxia. Probably the most advanced view is that the two conditions are phases of one disease.

ACUTE ANTERIOR POLIOMYELITIS.

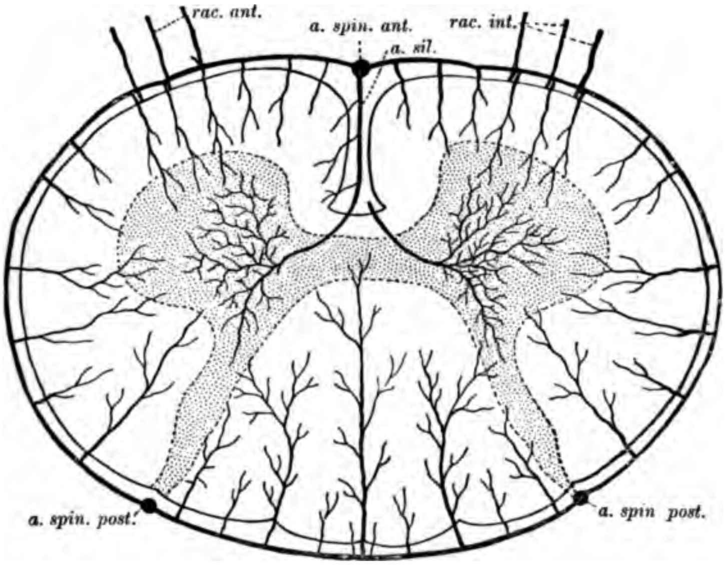
Definition.—This disease is sometimes called infantile spinal paralysis, acute infantile palsy, and acute atrophic paralysis. It is characterized by sudden loss of power in one or more of the limbs, most commonly the lower extremities. As a rule, the loss of power is complete, but occasionally it is localized in certain groups of muscles. Immediately after the development of the paralysis wasting of the muscles begins to take place and may be extreme. There is no disturbance of sensation.

Etiology.—Within recent years it has become more and more evident that acute anterior poliomyelitis is due to an infection. That the disease at times occurs in epidemic form was noted by Colmar more than sixty years ago. Since his time a large number of such epidemics have been recorded in this country and Europe. Caverly has recorded 144 cases in one epidemic near Rutland, Vermont, and in 1905 Ham reported an epidemic of 131 cases in Queensland. These epidemics are often associated with outbreaks of croupous pneumonia and influenza. The organism, if it be one, has not been isolated. Cases also occur which seem to be non-infectious. The most severe case I have ever seen followed a mother's attempt to punish a child for urinary incontinence by placing its naked back under a hydrant in midwinter. Within twenty-four hours the disease was present in full vigor.

The disease is distinctly one of early child life, the greatest number of cases developing in the first three years of life, it being very rare indeed after the tenth year. It is far more apt to develop in the summer than in winter, and has its greatest incidence in July and August. After this season it is most frequent in September and in June.

Pathology and Morbid Anatomy.—The essential lesion of this disease is an acute inflammatory process in the anterior cornua of the spinal cord, with associated hyperæmia of the membranes covering the anterior surface of the cord. The branches of the anterior spinal artery (Fig. 127) bear the brunt of the attack and are intensely engorged. Their finer branches are ruptured so that extravasations of blood take place into the gray matter. As a result of these changes the typical picture of tissues suffering from an acute inflammatory process is presented, for serum, leukocytes, and red cells crowd the nervous protoplasm. The ganglion cells of the gray matter in the anterior horns undergo marked degenerative changes. They undergo cloudy swelling and the nuclei become granular, or if the change is still more severe the nuclei disappear and the neurones lose their dendrites and become vacuolated. As a final stage the cell undergoes shrinkage, becomes a small, granular mass, and finally disappears. The damaged areas, in old cases, are occupied by connective tissue and are much shrunken, so that the affected gray horn is much smaller than its fellow. The anterior nerve fibres, which have their origin in this part of the cord, also atrophy. Associated with these changes in the anterior cornua of the cord there is often some involvement of fibres in the anterolateral tracts, because, it will be recalled, some

FIG. 127



Scheme to show the course and distribution of the terminal branches of the arterial plexus of the pia mater: *a. spin. post.*, posterior spinal arteries: *a. spin. ant.*, anterior spinal arteries· *a. sil.*, anterior median fissure; *rec. ant.*, anterior root arteries. (Van Gehuchten.)

of the fibres, or axones, which leave the anterior horns pass upward and downward in these columns to enter the anterior horns above and below to associate their function, and it is also due to the inflammatory process extending into the white columns.

The degree of the inflammatory process in the gray matter varies very greatly in different cases and may involve the cells of but a few muscles. It may affect chiefly that part of the gray matter which is most anterior or that nearer the commissure. In rare cases it would seem probable that no true inflammatory process develops in the cord, but that simple degenerative changes occur in the neurones in the anterior horns.

Symptoms.—The symptoms of acute poliomyelitis usually take the following course: A child in good health has a *restless* and *feverish night*, and seems on the next day to be somewhat out-of-sorts. In the course of twenty-four or forty-eight hours it not infrequently happens that the parents consider the child recovered from its acute illness, and it may be some days or weeks before the mother notices that one or both of the lower limbs are lacking in power. Not rarely it is found, as the child sits in its mother's lap, that one leg moves while the other hangs like a flail, or the mother notices that the child is unable to push its leg into its clothing as efficiently as it could do before it was taken ill. These may be considered as the symptoms of a comparatively moderate case.

In instances in which the onset and course of the malady is more severe, we find that fever is quite marked, often rising as high as 102.5°, and continuing at this point for several days. Occasionally, at onset, it may reach as high as 105°, and with this febrile movement there may be *headache*, *loss of appetite*, and *vomiting*. Sometimes *diarrhœa* occurs. In these instances the manifestation of loss of power is usually so marked that its presence is recognized within a few hours of its onset. Even in these cases, however, it not infrequently happens that the child is supposed to have suffered from an attack of acute gastric catarrh or indigestion until its inability to make certain movements calls attention to the palsy.

There is still a third type of cases in which *convulsions* appear at the time of onset. These convulsions may be cerebral or epileptiform in character, may be followed by deep coma lasting for many hours, and the symptoms may resemble an attack of cerebrospinal meningitis. Occasionally pain is a symptom of some importance, if, as already pointed out, the lesions in the anterior horns extend sufficiently backward to involve some of the sensory fibres beyond the commissures. These pains are usually felt about the joints. In some cases they come on not as a symptom of onset, but as a sequel, and seem to be due to an associated neuritis. The degree of the paralysis varies greatly in different cases. In some instances only one or two muscles seems to be affected. In others, the whole limb may be paralyzed, or both lower limbs and one upper limb may manifest loss of power. Even when the paralysis is quite widespread, it is rare for the cranial nerves to be affected, and equally rare for the sphincter muscles to lose power.

In some instances the disease seems to be progressive in its type, the full degree of paralysis not developing at once, but beginning in one part and then spreading to adjacent parts. Rarely one attack speedily follows another, involving a different set of muscles.

In very rare instances the paralysis may develop without any history of the symptoms of onset already described. Cases are on record in which the paralysis has been almost universal, but it is a noteworthy fact, in regard to the paralysis of acute poliomyelitis, that it is far more widespread in its early stages than later on, this being due to the fact that as the inflammation subsides certain cells which have not been irreparably damaged regain part or all of their functions, and so adequately supply the muscles under their control, or collateral muscles supply the power needed. On the other hand, it is a fact worthy of note that complete recovery of power in all the muscles affected practically never occurs. The result is, that impaired muscles may exist without great loss of power.

The period of recovery usually extends from one to three months. The muscles which fail to recover soon lose their contractility to faradism and then to galvanic electricity. The loss of faradic responses may be present as early as the eighth or ninth day, but in other instances some response is maintained, for a number of weeks. At the end of a few weeks the reactions of degeneration are observed. As would be expected from the lesions already mentioned and described when discussing the pathology of the affection, sensation is usually unimpaired. Reflex activity is, of course, diminished or lost because of the spinal lesions and the atrophy of the muscles. As secondary lesions to the paralysis we find shortening of the muscles with consequent contractures and deformities.

The legs are affected more frequently than the arms in the proportion of 3 to 1. The muscles below the knee suffer more frequently than those above the knee, and the tibial and peroneal muscles suffer more frequently than those of the calf. In the forearms the supinators usually escape, but the deltoids suffer more frequently than any other muscles in the upper extremity.

Diagnosis.—The acute poliomyelitis of childhood is readily diagnosed. Care must be taken that the muscular pains when they occur are not thought to be due to rheumatism. None of the other spinal lesions of childhood have such a characteristic onset. Cerebral palsies are usually unilateral and spastic. Pseudohypertrophic paralysis develops very gradually. The characteristic feature of the palsy in anterior poliomyelitis is the placidity.

Prognosis.—This is usually good so far as life is concerned, although if the attack has been severe vital resistance may be so diminished that other affections may readily cause the death of the child. The degree of ultimate paralysis can only be determined after two or three weeks of careful observation, when some idea as to the number of muscles which may recover can be obtained, particularly if electricity is used to determine the electrical contractility of the affected muscles.

Treatment.—In the treatment of acute poliomyelitis little can be done in the way of directly combating the disease. The child should be put at absolute rest in a quiet and darkened room. Sweet spirit of nitre and citrate of potassium should be given in small and frequent doses to diminish fever and to cause mild perspiration. It has usually been held that the administration of the salicylates, particularly salicin, is of advantage. This may be true with children who have a rheumatic or gouty heredity, but there is no reason to suppose that they exercise any specific influence upon the lesions in the cord. So, too, hot applications have been recommended to be applied to the back. It is difficult to understand how they can be of much value. Some mild counterirritant over the spine, such as a spice plaster, or a pepper plaster, may be advantageous. The whole object of the physician must be to produce nervous quiet and aid in diminishing the inflammatory process in the cord by avoiding excitement of the nervous system.

After the acute stage of onset is past, and the paralysis is present, that is to say, after sufficient time has elapsed for the acute stage of the inflammation to have passed by, or, in other words, in three or four weeks after onset, moderately large doses of strychnine may be given, but care must be taken that the doses are not so large as to produce twitching or great nervous irritability. At this time, too, the slowly interrupted faradic current may be applied to the paralyzed muscles, and particularly to those which are semiparalyzed, in the hope that in this way their nutrition may be maintained. It must not be forgotten, however, that the greatest care must be exercised that the muscles are not overfatigued, since if they are exhausted they will more rapidly atrophy than if no electricity was employed. If electricity is used before the spinal cord has recovered from the acute stage of the inflammation, it will make the condition worse. In many instances it is advisable to use electricity on one day and careful, gentle massage on the next. The electrical current should never be employed in such strength as to give the child pain or distress.

As general tonics for the nervous system the hypophosphites, glycerophosphates, cod-liver oil, and iron may be used.

Should any tendency to deformity take place, this must be treated by the methods commonly resorted to by orthopedic surgeons. Rapid recovery should not be expected in these cases. Careful treatment for months is necessary to get the best results.

CHRONIC ANTERIOR POLIOMYELITIS.

Definition.—It is evident from its name that this disease closely resembles acute anterior poliomyelitis. A very important difference lies in the fact that in the acute form the damage takes place suddenly, and then ceases to progress, some improvement occurring in most instances, whereas in the chronic form the atrophic process is slow in onset and progressive and so the symptoms get worse rather than better. When the symptoms develop during a period of from two weeks to a month the term subacute is applied, and when they come on even more slowly, so that several months are consumed in their advance, the term chronic is used. The dominant symptoms are muscular wasting with paralysis. Chronic anterior poliomyelitis is sometimes called " chronic atrophic spinal paralysis " or " progressive muscular atrophy."

Etiology.—This is unknown. Exposure to cold and wet has been thought to be a cause in some cases.

Pathology and Morbid Anatomy.—The lesions of chronic anterior poliomyelitis consist in atrophy of the nervous tissues of the anterior horns of the gray matter of the cord. Not only the cell bodies but their axones and dendrites all undergo degenerative change. There is no acute inflammatory process present as in the acute form of the disease, and no hemorrhages into the tissues about the vessels. The dominant change is a simple atrophy or wasting. The anterior nerve roots also suffer atrophic changes, and the so-called association fibres of the cord and the cells which give rise to them also atrophy. As these association fibres pass to the anterior lateral columns of the cord the degenerative process extends to them as well, and this, combined with the wasting of the anterior nerve roots, produces a shrinkage in the size of these columns, which is, however, not great enough to be recognized by the unaided eye. The atrophic process extends down these tracts and involves the motor nerve fibres all the way to the nerve plates in the muscles, which in turn undergo atrophy, the muscular fibres losing their striæ and showing fatty globules inside the sarcolemma.

Symptoms.—The symptoms of this malady vary somewhat with the portion of the spinal cord which is chiefly affected. Most commonly the earliest manifestations of the disease appear in the upper extremities, and this is usually called the "Aran-Duchenne type of the disease." It first manifests itself in the adductor muscles of one thumb. From these it extends to all the small muscles of the hand, which rapidly become wasted. Flexion of the fingers upon the hand is impossible, and as the interossei which flex the first phalanges are paralyzed, the long flexor and extensor muscles of the forearms are unopposed. As a result we find that the long flexors flex the second and third phalanges and the long extensors extend the first phalanges, giving the so-called "claw-hand" deformity. This effect is increased by the prominence of the extensor tendons, caused in part by the wasting of the smaller muscles of the hand. The hand or the shoulder on the opposite side soon suffers. Finally, all the upper arm and shoulder muscles atrophy, and later those of the upper thorax as well. Still later the lower extremities become involved. Often portions of the latissimus dorsi, the trapezius, the triceps, the pectoralis major, or other muscles escape. When the cervical muscles fail the head cannot be held erect, and when the costal muscles are atrophied the act of respiration may be solely diaphragmatic. Finally, symptoms of bulbar paralysis may ensue.

In that type in which the disease first affects the lumbosacral portion of the cord the peroneal muscles in one leg undergo paralysis and wasting. This condition then develops in the muscles of the other leg. A little later the anterior tibial muscles are affected, first on one side and then on the other. At this time there is "drop-foot" and the patient has to use his thigh muscles to raise the leg so that the toes will not strike obstructions and cause stumbling. As the pathological process in the cord advances the adductor muscles in the thighs and the gluteal muscles are paralyzed.

In still another type, sometimes called "Duchenne's type of ascending paralysis," the paralysis and wasting extend upward from the legs and speedily affect the muscles of the trunk, the arms, the forearms, and the hands so that an almost total paralysis ensues and death comes from involvement of the centres in the medulla or by reason of some intercurrent disease such as pneumonia. Sensation is not disturbed, but in a few cases a sense of discomfort may be complained of in the legs. The reaction of degeneration develops quite early in the affected parts and finally response to electrical stimulation is completely lost. The muscles present fibrillary contractions if irritated, but the bladder and rectum are not paralyzed. The paralysis is due to the wasting. The reflexes are diminished or lost in the affected muscles.

Diagnosis.—It is important to remember that chronic muscular atrophy may arise from other diseases than chronic anterior poliomyelitis, such as amyotrophic lateral sclerosis, the muscular dystrophies, peripheral neuritis, and syringomyelia.

From amyotrophic lateral sclerosis (which see) it is distinguished by the absence of spastic symptoms and lack of the exaggerated knee-jerk and of Babinski's reflex.

From muscular dystrophy by the absence of fibrillary tremor and of the reaction of degeneration in the latter condition, and by the fact that the spinal form is a disease of adult life. From syringomyelia this condition is separated by the absence of the dissociated anæsthesia and of trophic lesions in the skin.

In neuritis the distribution of the paralysis is usually symmetrical; whereas, in chronic poliomyelitis the paralyzed parts are irregularly distributed. Unless the neuritis be purely motor in character there are sensory disturbances and tenderness on pressure over the nerve trunks and in the bellies of the muscles. Moreover, in neuritis there is commonly a toxic cause recognizable.

The Charcot-Marie-Tooth type of muscular atrophy may be confused with certain forms of progressive muscular atrophy. The first of these, however, is a disease of early life, and the paralysis in the legs does not extend above the knees or above the elbows, as a rule. Further sensation is usually disturbed or impaired in the Charcot-Marie-Tooth type and preserved in this chronic muscular atrophy.

Prognosis.—The prognosis is grave. The future of the case can be determined somewhat by the rapidity of the development of the symptoms, for in the rapidly advancing cases the outlook is of course worse than in others. When the symptoms follow injury to the spine the prognosis is better than in the idiopathic cases.

Treatment.—The rapid type of cases should be treated as we treat acute poliomyelitis. The chronic forms should be cared for in a manner practically identical with that of the advanced stages of acute poliomyelitis. The treatment, therefore, consists in hygienic surroundings, nutritious food, and an abundance of fresh air and sunshine. Care should be taken that the paralyzed muscles are not exhausted by too much rubbing or exercise. Strychnine may be given in moderate doses three times a day for several weeks at a time. Fowler's solution, in moderate dose, is also useful as a tonic. Therapeutic measures, however, cannot promise much in this disease.

BULBAR PARALYSIS.

Bulbar paralysis is a term applied to a group of symptoms referable to lesions of cranial nerve nuclei in the medulla oblongata or "bulb," which affect the tongue, lips, and larynx in peculiar association, so that the condition is called "glosso-labio-laryngeal paralysis." The lesions may implicate the cranial nerve nuclei in the pons and crus, and the gray matter of the spinal cord, but atrophy of the tongue and lips is the main feature of the disease which is centred in the medulla.

Etiology.—The cause of this disease is unknown. It occurs most frequently between the thirtieth and fiftieth years.

Pathology and Morbid Anatomy.—The lesions of this malady consist in degenerative changes in the nuclei of the motor nerves which supply the tongue, lips, larynx, and pharynx. As the disease advances, additional nuclei of the cranial nerves become involved so that the pneumogastric, the facial, the motor fibres of the trifacial, and more rarely the abducens and oculomotor nerves are affected. Occasionally, it occurs as part of amyotrophic lateral sclerosis. If the reader has a clear conception of the pathology of chronic anterior poliomyelitis he will understand the pathology of this affection as well.

Symptoms.—The symptoms of chronic bulbar paralysis usually begin with *difficulty in moving the tongue* in speech so that the patient is unable to use easily letters like *v*, *n*, *r*, *f*, and *l*. The speech becomes nasal, swallowing becomes difficult, and when the muscles of the lips become affected labial sounds become imperfect, letters like *b* and *p* being difficult to pronounce. When the laryngeal muscles become paralyzed speech is lost completely. *Chewing becomes difficult*, owing to the paralysis of the tongue and lips. There is *difficulty in swallowing*, and the food not infrequently finds its way into the larynx, from which it may descend and cause septic pneumonia. Owing to the paralysis of the facial nerves, the expression of the lower portion of the face becomes altered, the lips sag, and saliva may flow over the chin. *Fibrillary contractions* of the affected muscles also occur, and the tongue lies relaxed and powerless in the floor of the mouth.

Diagnosis.—True bulbar paralysis must be separated from that still more rare affection known as myasthenia gravis. In this condition the general muscular system also suffers from feebleness, but degeneration does not take place in the affected muscles, and they do not undergo material wasting. Furthermore, the condition in myasthenia gravis is often characterized by periods of remission or partial recovery. Autopsy in those cases of myasthenia gravis which have come to a fatal termination has always failed to reveal the lesions which have been described as characteristic of true bulbar paralysis.

Prognosis.—This form of paralysis is invariably fatal.

Treatment.—The treatment of bulbar paralysis consists in the administration of tonics, and in an endeavor to maintain the patient's general health at the best possible level. Do what we will, the disease cannot be affected by any plan of treatment yet devised.

LATERAL SCLEROSIS.

Definition.—Lateral sclerosis, also called "spastic paraplegia," is a condition in which the patient suffers from stiffness or spasticity of the muscles of the lower extremities, with loss of power which ultimately amounts to distinct paralysis. The condition is characterized by great reflex irritability. There are no sensory disturbances.

History.—Türck described sclerosis of the lateral columns of the cord in 1856, and Charcot made further contributions as to the symptoms in 1865. Seguin described it still further in 1873 as "tetanoid paraplegia," an excellent term, but it remained for Erb in 1875 and 1877 to make a full exposition of the disease.

Etiology.—In many cases the cause of lateral sclerosis cannot be discovered. Syphilis may be a cause, or, to speak more correctly, the malady may be a sequel of syphilis. In some instances injuries to the back are followed by these symptoms. In one case in my experience a horse reared and fell backward on his rider, who at once found his legs paraplegic. This passed away in a few moments, but after a few months lateral sclerosis gradually developed. In another case under my care a man stood in very cold water washing sheep, and dated the beginning of his malady to that exposure. In both of these cases, however, it is quite probable that a pachymeningitis or a hemorrhage into the cord or a myelitis was the cause of the symptoms rather than a true primary lateral sclerosis. In other cases prolonged marches have seemed to produce it. It is a disease of the third and fourth decades of life. In some instances the disease seems to depend upon an hereditary defect in the lateral columns of the cord (Strümpell's family type of lateral sclerosis).

Pathology and Morbid Anatomy.—The lesions of lateral sclerosis are clear and definite. As the name of the disease implies, they are situated in the lateral or crossed pyramidal tracts of the spinal cord, and they develop chiefly in their lower portions in the early stages of the disease. The axones progressively atrophy from below upward, and this is associated with disappearance of the myelin and an overgrowth of the connective tissue.

When the disease invades the cervical region the anteromedian columns of the cord may be affected as well.

Symptoms.—In studying the symptoms of primary lateral sclerosis it must not be forgotten that they may be simulated by secondary lateral sclerosis following lesions higher up in the cerebrospinal system. Thus, it is a well-known fact that a large number of lesions in the brain or cord may result in degenerative changes in these motor pathways, and so cause spastic paraplegia. Those in the brain are hemorrhage, abscess, tumor, and softening, which, affecting the motor cortex or the motor pathways, induce a descending degeneration in the pyramidal tracts. In these cases the symptoms are usually limited to one side, but in the cerebral palsies of childhood the lesions are often bilateral. (See Infantile Cerebral Palsy.) Any lesion in the spinal cord which cuts off the fibres in the lateral tracts from their trophic cells in the brain, also results in lateral sclerosis. Thus, a transverse myelitis, disseminated sclerosis, hemorrhage into the cord, and syringomyelia may so result. Lateral sclerosis is not rarely a part of the pathology of paresis. Lesions outside the cord, such as tumors, disease of the spinal column, or thickening of the membranes may sometimes cause these symptoms.

The symptoms upon which we base the diagnosis of lateral sclerosis are the peculiar *spastic contractions* of the muscles of the legs, so that they are in a state of extension as soon as the patient attempts to move them. The attitude of the legs is that of a person with ankylosis of both knees, and the foot is apt to be extended. When the patient walks he has *difficulty in bending the knees* and the ankles, and still greater *difficulty in raising the toes* as the foot is brought forward for another step. For this reason he is prone to trip over small obstructions and to have bad falls, because his muscles are so stiff that he cannot catch himself as he loses his balance. The stress and strain of walking are therefore very great, and the muscles may become so fatigued that they ache, but this is the only sensory symptom. Crossing the legs when sitting becomes impossible, and if the patient is recumbent the knees may be approximated owing to the greater strength of the adductor muscles. This adduction of the knees also interferes with walking. If the patient's muscles are grasped they are found to be hard and tense. On further physical examination it will be found that the reflexes are all increased. *Ankle clonus* is *marked*, and the *Babinski reflex* is soon manifested. The reactions of degeneration do not appear and the muscles do not atrophy until the disease has lasted several years, when they may waste from disuse.

Finally, when the malady has continued for a very great length of time the position of the lower extremities in stiff extension may be changed to that of contracture so that they are sharply flexed at the knees and fixed in this position.

The upper extremities nearly always escape, but in the rare instances in which they are involved the extensor muscles suffer first and suffer most.

Diagnosis.—As already pointed out, the diagnosis of this disease should not be made till the causes capable of producing secondary lateral sclerosis are excluded. Occasionally hysteria may produce symptoms very like it. A typical picture of such a case will be found in my book on *Practical Diagnosis*. In such an instance the sex of the patient and the other signs of hysteria should be carefully considered before a diagnosis of lateral sclerosis is made. When spastic paraplegia is combined with muscular atrophy the condition is one of amyotrophic lateral sclerosis (which see).

Prognosis.—This is bad as to recovery, but its unfavorable character is modified by the fact that the progress is usually very slow. Often the disease lasts twenty-five years or more.

Treatment.—Unfortunately the results which may be obtained from the treatment of lateral sclerosis are not brilliant. The suggestion that nux vomica or strychnine be employed does not seem to be based on very rational views of the physiological action of this drug. Excitation of the motor tracts of the spinal cord is already present, and strychnine rather tends to increase this condition and to exaggerate the spastic condition of the lower extremities. In some instances full doses of the extract of conium are advantageous. In others large doses of chloral or one of the bromides may be employed. The gentle forms of massage may also relieve the sensation of tension and aching in the limbs. Care should be taken that the patient does not walk far enough to exhaust himself. In some instances a hot pack will give relief, particularly if it is taken at bedtime, although of course it exercises no curative influence upon the progress of the disease. Electricity is useless.

Syphilitic Spastic Spinal Paralysis.—Under the name of syphilitic spastic spinal paralysis Erb has described a form of lateral sclerosis developing within five years of the primary sore, but differing from ordinary lateral sclerosis by the presence of some lack of rectal and vesical control and mild disorders of sensation. This so-called syphilitic spinal spastic paralysis of Erb is due to a specific endarteritis, which produces embolism or thrombosis of the vessels of the spinal cord and a true myelomalacia, although some clinicians have considered it a meningomyelitis.

AMYOTROPHIC LATERAL SCLEROSIS.

Definition.—Amyotrophic lateral sclerosis is a progressive form of chronic spinal paralysis characterized by advancing muscular atrophy associated with spastic paraplegia, or, in other words, the symptoms of lateral sclerosis are present. Although the symptoms are largely spinal, modern research has shown that the motor pathway is affected throughout in advanced cases, from the beginning of its upper segment in the motor cortex to the endings of the lower segment in the peripheral nerves. The cardinal symptoms depend, however, upon degeneration of the contiguous parts of these segments, namely, the pyramidal tracts and the anterior horns of the gray matter; and the disease may be regarded as a combination of lateral sclerosis with chronic poliomyelitis. Not rarely the disease invades the medulla, and symptoms of progressive bulbar paralysis are added to the clinical picture.

Etiology.—Amyotrophic lateral sclerosis is usually met with in persons between thirty and fifty years of age. In some instances there is a history of exposure to cold or of violent exertion. In still others, it is found that the patient has been an artisan employed in the handling of such metallic poisons as mercury, lead, or arsenic, or that he has been addicted to the excessive use of alcohol. In some instances, too, the disease has apparently followed severe injuries, but in no instance has it been proved that there is actual relationship between any of these causes and the development of the disease. In all probability, these factors only become active in those cases in which there is a defective development in the central nervous system, which renders its motor elements peculiarly susceptible to damage or disease.

Pathology.—The pathological conditions in amyotrophic lateral sclerosis may be considered in three parts. The first and most important is the advancing atrophy which involves the motor neurones in the anterior horns of the gray matter of the spinal cord. Indeed, the condition is very similar to that which occurs in chronic anterior poliomyelitis or even identical with it. As a rule, the alterations take place chiefly in the cervical portions of the cord, but in some instances, particularly if the disease lasts a long time, the anterior portion of the gray matter in the lumbar region is also affected. Similar atrophic changes take place in the motor nuclei of the cranial nerves in the pons and medulla. Next in importance to these alterations in the trophic portions of the spinal cord is the atrophy and degeneration of the fibres in the lateral columns and the anterior median columns. These changes extend to the motor cells of the brain and are not limited to the lateral columns, the cortex being involved secondarily by "retrograde" degeneration from the cord and medulla upward through the pons, crura, capsule, and corona radiata. In other words, the degeneration does not begin in the motor cortex, but in the spinal cord. Following the wasting of the nervous elements of the spinal cord, there is an overgrowth of connective tissue which not only involves the lateral columns and the anterior median columns, but also the association fibres which are closely connected with the anterior horns of the gray matter.

Symptoms.—The symptoms of amyotrophic lateral sclerosis are quite characteristic, and depend in their development to some extent upon the portion of the nervous system which is first affected by the disease. In those instances in which the lesion first affects the cervical portion of the cord and the anterior horns of the gray matter the arms are first affected. These extremities manifest some *stiffness in the muscles*, and their *reflex excitability is increased*. Almost simultaneously with these symptoms there is *wasting of the muscles of one or both hands*, with associated loss of power. From the hand the paralysis extends to the forearms, or it passes directly to the muscles of the shoulder and affects those of the forearms afterward. *Fibrillary contractions* develop very early, and may be produced by tapping the muscles or by exposing them to cold. The *fingers* are often in *semi-flexion*, and if the physician endeavors to straighten them, it is found that the muscles are rigid and resistant, even though the patient has lost power in them.

When the disease develops chiefly in the pyramidal tracts of the motor columns of the cord, the evidences of spastic paralysis are the first things to impress themselves upon the observer, and it may be impossible in the early stages of the disease to separate the malady from ordinary lateral sclerosis, since the legs are stiff and move with difficulty, the *knee-jerks* are *exaggerated*, and *ankle clonus* and the *Babinski reflex* are usually present. When the patient walks the *toe is dragged along the ground*, the whole *leg is stiff*, and one foot is often placed awkwardly in front of the other, through the contraction of the adductor muscles.

Whether the disease first begins in the arms or in the legs, it is worthy of remembrance that it is often very much more marked upon one side than upon the other.

As the disease advances it sometimes happens that evidences of bulbar paralysis develops, the *speech becoming affected*, as in ordinary bulbar paralysis. *Swallowing* may also become difficult. Owing to paralysis of the uvula and palate, choking often takes place, and fluids when taken into the mouth escape through the nose. Indeed, all the symptoms of bulbar paralysis may be present, and inequality of the pupils may be noted. Tapping the affected muscles in any portion of the body usually produces marked contractions in the muscle tapped and in neighboring groups of muscles.

When *contractures* occur the hands may become greatly deformed, and the feet may be distorted into any of the forms of talipes.

In the advanced forms of the disease the muscles of the trunk and neck become atrophied, so that it is impossible for the patient to sit up, and the head falls to either side or forward. The muscles develop the *reactions of degeneration* and lose their electrical excitability. There is no loss of intelligence, but sometimes in advanced cases a mild dementia appears. When the bulbar symptoms are marked, palpitation of the heart is often present.

Diagnosis.—From bulbar paralysis amyotrophic lateral sclerosis is separated by the fact that lateral sclerosis presents marked spinal symptoms with paralysis of the upper extremities and spastic paraplegia of the lower extremities. If in a case of supposed bulbar paralysis these symptoms develop later it proves that the bulbar palsy has been due to the oncoming of amyotrophic lateral sclerosis. If the symptoms are due to the presence of a meningitis in the cervical portion of the cord, there is stiffness and loss of power in the arms, and there will also be pain of a severe character. So, too, injury or pressure upon the spinal cord in the dorsal and lumbar regions, producing a spastic paraplegia, will also produce sensory disturbances and involve the functions of the bladder and rectum.

From syringomyelia amyotrophic lateral sclerosis is separated by the presence in the former of analgesia and the rapid trophic changes which take place not only in the muscles, but in the bones, the skin, and its appendages. In many instances it is almost impossible to differentiate between amyotrophic lateral sclerosis and ordinary lateral sclerosis, and it is only by the development of the symptoms which arise from involvement of the gray matter of the cervical portion of the spinal cord and the medulla that the separation can be made.

Prognosis.—The prognosis in a case of amyotrophic lateral sclerosis is absolutely unfavorable so far as recovery is concerned. The duration of life depends upon the rapidity with which the vital centres in the medulla become involved. In some instances death comes within two years after the onset of the malady, whereas in others it lasts for a decade or even longer. The prolonged cases are usually those in which the involvement of the lateral columns seems to be the first stage of the disease. As a rule, death does not occur as the direct result of the disease, but from complications which are produced by it; as, for example, the inhalation of particles of food into the respiratory passages because of the bulbar paralysis. Rarely there is heart failure due to involvement of the cardiac centres.

Treatment.—There is no form of treatment which can be considered curative. Gentle massage and the use of electricity,with the hope that the wasting of the muscles may be diminished, has been tried by some clinicians, but it is manifest that this plan of treatment must be used with great caution, since if the trophic centres are destroyed, the muscles must necessarily undergo wasting more rapidly if they are exercised than if they are not used. If bulbar symptoms are present the patient should be fed by means of a stomach tube. The employment of nervous stimulants, such as strychnine, is inadvisable, because it exaggerates the spastic condition of the lower extremities.

MYELITIS.

Definition.—Myelitis is a term which at one time was loosely applied to all inflammatory processes in the spinal cord. Its application is becoming limited as our conception of the diseases in this part of the body becomes more definite, but it is still used to describe an inflammatory process in the cord, which is general or widely diffused or disseminated. A myelitis may be *acute*, *subacute*, or *chronic*. If it extends across a given segment of the cord it is called a "transverse myelitis;" if it is distributed in several foci through the cord, it is called "disseminated myelitis," and if it extends upward or downward, it is called an "ascending or descending myelitis."

The term "acute myelitis" is applied to those cases of sudden onset taking but a fortnight to develop. The term *subacute* is applied to those which consume from two to six weeks in onset, and the term *chronic* to those which develop so slowly that a longer time elapses before the disease is marked.

When the gray matter of the spinal cord is affected, it is called "poliomyelitis," from the Greek word *πολιος*, meaning gray.

When the brain and cord are involved it is called "encephalomyelitis," and when the gray matter about the central canal in the cord is affected it is called a "central myelitis."

Acute and Subacute Myelitis. **Etiology.**—The chief cause of this condition is without doubt an intoxication, due to the action of toxins developed during the course of acute infectious diseases. At one time it was thought that exposure, sexual excess, and severe toil were causes, but we now know that at the most they are but predisposing factors in that they diminish vital resistance. In addition to the ordinary infectious fevers, myelitis may be caused by gonorrhœa and malaria. Occasionally severe vesical infection produces acute myelitis. Myelitis has also been said to follow concussion of the spine and other injuries, but they probably act solely as predisposing agencies.

Pathology and Morbid Anatomy.—If we take transverse myelitis as a type of the various forms of this disease, we will find on opening the spinal canal that the pia mater at the level of the lesion is hyperæmic and reddened. The cord is also reddened and somewhat swollen, and its bloodvessels engorged On section of the cord the lines of demarcation between the white and gray matter are to a great degree obliterated. The cord is softened and its texture may be actually diffluent, this very soft state being, however, at least in part a postmortem change. If this part of the cord is placed under the microscope it is seen to be filled with granular cells, the bloodvessels are surrounded by extravasated leukocytes, and bacteria may be found. Small extravasations of blood into the tissue of the cord may be present from rupture of the vessels. The nerve cells are found to have undergone granular degeneration, and their axones and dendrites have also been destroyed. The axis cylinder of the nerve fibres is greatly swollen and has evidently undergone segmentation. Fatty degeneration of the myelin is found. The connective-tissue cells are swollen, and if any time has elapsed, an overgrowth of the neuroglia is present.

For a clear understanding of the cause of the symptoms met with in acute myelitis, myelomalacia, and chronic myelitis it must be remembered that the presence of a lesion in the spinal cord in the motor tracts produces a descending degeneration in that tract, because the nerve fibre is cut off from its nerve cell or neurone. If the lesion be in the sensory tracts the degeneration is ascending. The ultimate symptoms, therefore, consist not only in those which arise from the primary focus or lesion, but in those which develop as the result of these secondary changes. These changes are demonstrable within a few days after the injury, and rapidly progress so that at the end of three weeks the degeneration of the affected fibres is at its height. The overgrowth of the connective tissue is not marked until a later period. Thus, we find that the chief degenerative change below the lesion is in the anterior and lateral pyramidal tracts, and above the lesion in the dorsal columns of Goll and Burdach, in the direct cerebellar tracts, and in the columns of Gowers. In addition to these chief secondary changes, there is also, for a short distance below the primary lesion, descending degeneration in the anterior and anterolateral colums, and in certain small "fields" of the dorsal columns (oval field of Flechsig, etc.), which contain the so-called association fibres.

The ascending degeneration which takes place after a transverse lesion affects chiefly Goll's and Gowers' columns, and the higher the primary lesion the greater the degree of the degenerative process. The column of Burdach, on the other hand, while markedly degenerated near the site of the lesion, is less and less affected higher up in the cord.

Symptoms.—The symptoms of myelitis vary somewhat with the portion of the cord which is affected and with the extent of the pathological process.

When there is a *transverse myelitis* of the dorsal portion of the spinal cord the symptoms in the stage of onset consist in *wretchedness* and *moderate fever*. There may be *pain* in the *back* and *numbness* and *tingling* in the *lower extremities*. *Twitchings* or *cramp-like contractions* of the muscles in the *legs* may occur, and, *very rarely* in adults, a *convulsion* may develop. Sometimes, however, these prodromata are absent, and the first symptom complained of is *loss of power* in the *lower limbs*, which speedily develops into a *complete paraplegia*. Paraplegia may become complete in a few minutes or a few hours. In other instances of the subacute type the onset is so slow that days and even weeks may elapse before the loss of power is complete.

The paraplegia arising from a transverse myelitis in the dorsal region is usually spastic, and the deep reflexes are increased. The *legs* are *outstretched*, as in ordinary paraplegia, unless degenerative or irritative lesions arise in the lateral pyramidal tracts below the site of the transverse lesion, when they may become *flexed* by the spastic state of the muscles of the thighs. In other instances these muscles suffer from *twitchings* and *temporary contractions*, which may be strong enough to prevent the examiner from eliciting any signs of exaggeration of the deep reflexes. There is always *paralysis* of the *bladder* and *rectum* in transverse myelitis, and *retention* or *incontinence of urine and feces* may be present. Partly because of pressure and unavoidable uncleanliness, but chiefly because of trophic disorders and abnormal blood supply, *bed-sores* are prone to develop on the sacrum and buttocks. The muscles in the legs do not rapidly atrophy, because they receive trophic impulses from the cells in the anterior cornua of a lower level of the cord than that of the lesions. The *skin* of the *legs* and of the body below the level of the lesion is *anæsthetic* to all forms of irritation, and at the upper margin of this anæsthetic area there is a *girdle sensation*, and, it may be, a *band of hyperæsthesia*.

When the myelitis affects the *lumbar cord* the *paraplegia* is not only *absolute* as to voluntary movement, but as to reflex action as well, *all reflexes being lost*. A similar condition also occurs in some cases when the injury is at a higher level, provided that the cord is completely severed or the injury so severe that severance is practically complete.

If the lesion is in the *cervical cord* the *arms* are not only *paralyzed*, but *undergo atrophy* and are *flaccid*, the legs suffer from a *spastic paraplegia*, and the *arms*, *legs*, and *body* are *anæsthetic*. There may also be *dilatation* or *contraction* of the *pupils*. If the lesion is high up in the *cervical area*, then the *paralysis* of the *arms* may be *spastic* instead of flaccid, and they do not undergo atrophy. In such a case *respiration* is very *difficult*, because of the loss of power in the diaphragm and in the other respiratory muscles.

Prognosis.—While it is a fact that the prognosis in transverse myelitis is always grave, it is also a fact that partial recovery sometimes takes place. Thus, Oppenheim states that that form following gonorrhœa always gets better under good treatment, and that that form due to the acute infectious fevers has good chances for recovery. On the other hand the outlook in syphilitic cases is not good, and in septicæmia, tuberculosis, or puerperal sepsis it is bad. Again, the prognosis varies with the severity of the symptoms and the lesion, for manifestly it must be worse in complete transverse myelitis than in that form in which the destruction of the cord is not so complete. The cause of death is usually bed-sores, with exhaustion and septic cystitis.

Treatment.—An understanding of the lesions of this disease makes it evident that treatment of a curative nature is useless. Careful feeding with easily digested food, the maintenance of perfect cleanliness in the parts pressed upon in the dorsal position, and the cautious use of the catheter should be resorted to. Hyoscine may be given to stop the annoying twitchings of the muscles.

Chronic Myelitis. **Definition and Etiology.**—Chronic myelitis is, as its name implies, a chronic form of inflammatory process in the cord which develops as a result of a large number of causes. Not only may the causes of acute myelitis set up a process which may become slow and chronic in its progression, but other factors may produce it, of which the most important are impairment of its blood supply resulting from degenerative changes in the bloodvessels from atheroma or syphilitic arteritis. Chronic myelitis may also arise as a result of pernicious anæmia. In some cases, too, the primary cause lies in a meningitis of the membrane surrounding the cord, whether this meningitis be due to an infection or to injury followed by infection. When syphilis is the cause, it not rarely happens that the inflammatory process is limited to one or more parts of the spinal cord, so that the symptoms of spastic paraplegia due to disease of the lateral pyramidal tracts develops, or in other instances the symptoms of locomotor ataxia are present, because the posterior tracts in the cord are affected. Rarely but one side of the cord may be affected, producing a crossed paralysis of motion and sensation (Brown-Séquard syndrome).

Finally, the physician must recall the fact that most of the forms of chronic disease of the spinal cord, such as disseminated sclerosis, amyotrophic lateral sclerosis, and the various forms of poliomyelitis, may, in their advanced stages, resemble what has been called chronic myelitis. Indeed, it is so rare to meet with a case of chronic myelitis which cannot be placed under one of these headings that many neurologists are inclined to deny the existence of chronic myelitis as a separate malady. (See below.)

Pathology and Morbid Anatomy.—The changes in the spinal cord which are found in cases of so-called chronic myelitis are not so manifest as in the acute form when the cord is studied by the naked eye. In one class of cases the appearance is quite like that of disseminated sclerosis in that areas of overgrowth of connective tissue are found in both the white and gray matter of the cord. This overgrowth of the neuroglia is situated chiefly around the bloodvessels, the walls of which are also thickened and their lumen narrowed. When as a result of the degenerative process an axis cylinder has become distended or swollen, a small cavity is formed, and if many of these are present they may give the section of the cord a cribriform appearance. The pia mater is often found adherent to the cord.

In the other type of case a microscopic study of the cord reveals changes which are evidently the result of the several diseases of the cord already named, and which are really the cause of the symptoms presented by the patient rather than that a true primary chronic myelitis has been present. Thus, it is found that as a result of a lesion in the pyramidal tracts, a descending degeneration of the fibres in that tract takes place, or if the lesion has been in the posterior columns, an ascending degeneration ensues.

Symptoms.—When the condition of chronic myelitis follows acute myelitis, the symptoms of that state persist with gradually increasing severity until *death by exhaustion* or some intercurrent malady ensues. If, however, the process is of the slow or chronic type from the time of onset, the *primary feeling of weariness* and *weakness* on exertion passes gradually into a state of *paralysis*, more or less complete. Owing to the degenerative changes in the lateral tracts, the *paralysis* is usually *spastic*. There is often an *exaggeration* of the *deep reflexes*, with *ankle clonus*. In other instances the character of the response to reflex stimulation is entirely dependent upon the portion of the cord which is involved. The *muscles* may *gradually waste* and give the *reactions of degeneration*. Not rarely the loss of power extends gradually to the trunk and arms. *Sensory disturbances* are *common*. There may be *patches* of *anæsthesia* and *paræsthesia*, and occasionally moderately *severe pains* may be felt in the nerves in the extremities. *Vasomotor disorders* in localized areas of the skin may be present, one part being very pallid and another hyperæmic. *Bed-sores* finally develop, as in the acute form of the disease.

Diagnosis.—Chronic myelitis as a separate disease is so rare that a diagnosis of its presence should be made only after a most careful study of a case.

From disseminated sclerosis it is separated by the absence of evidence of lesions in the brain and lower cephalic centres, such as nystagmus, intention tremor, and scanning speech. From lateral sclerosis it is separated by the absence of vesical and rectal paralysis in that disease. Then, too, lateral sclerosis is not characterized by loss of sensation. Paralysis due to poliomyelitis is separated also by the absence of anæsthesia, and the fact that the paralysis of the muscles is not, as a rule, so general.

Prognosis.—Recovery does not occur. Life may be prolonged for years if the process is not progressive and no intercurrent disease attacks the enfeebled sufferer.

Treatment.—This is, of course, concerned entirely with the maintenance of good health, with the hope that a terminal infection may not occur. If there is a clear history of syphilis the iodides and mercury may be used, but they are rarely of much value in this state.

SENILE PARAPLEGIA.

Under this unsatisfactory clinical title may be described a condition occasionally met with in old persons, and probably depending upon impaired blood supply to the cord, resulting from degenerative changes in the vessels which are not severe enough to result in myelomalacia.

Symptoms.—The symptoms consist in a *moderate degree of loss of power* in the *lower limbs* so that it is difficult for the patient to move about. The *legs* may be *shuffled along the floor* instead of lifted clear of it. When the malady develops rapidly and is severe it may be impossible for a time to separate it from true myelitis.

Treatment.—The treatment consists in rest in bed, massage, and hydrotherapy. Internally, the iodides and circulatory stimulants, such as strychnine and digitalis, should be used.

MYELOMALACIA.

This term is applied to a state of the spinal cord in which it undergoes softening because of embolism or thrombosis of its bloodvessels, with the result that its blood supply is impaired. The degenerative changes consist in those which we would expect to find when necrosis of these parts occurs, namely, extravasated red and white blood cells, fat-globules, and broken axones. When the patient lives for a considerable length of time after this accident to the circulation occurs, a microscopic examination of the cord will reveal an overgrowth of connective tissue. The essential difference between this state and one of acute myelitis is that in this condition the process is necrotic; whereas, in the latter it is primarily inflammatory, and diapedesis of white and red cells takes place as a part of a vital process.

SYRINGOMYELIA.

Definition.—Syringomyelia is a condition of the spinal cord characterized by the formation of a cavity or cavities in its substance; by loss of pain sense and temperature sense, with preservation of tactile sense; by the development of progressive muscular atrophy and paralysis, and by nutritional changes in the skin, muscles, bones, and joints.

History.—Although a very rare disease, it was described before many other very common maladies, the state of the spinal cord having been first noted in 1546 by Etienne and given its name by Ollivier in 1824. We are, however, able to diagnosticate the affection by reason of researches of Schultze and other later investigators.

Etiology.—This is unknown. In some cases it is probably dependent upon a congenital defect. In other instances it has been thought to be due to disease of the spinal arteries, and in some cases Van Gieson has shown that it has developed from a perforating hemorrhage into the cord itself.

Pathology and Morbid Anatomy.—When the spinal cord in a case of syringomyelia is examined macroscopically, the membranes are found to be normal, but the surface of the cord may be irregular and portions of it protrude, while at other places retraction of its surface seems to be present. A closer examination of the areas of bulging may reveal fluctuation, and from such areas, if they be punctured, a clear serous fluid may run quite freely. This cystic state may extend very considerable distances up and down the cord, and may extend so far transversely as almost to cut the cord in two. The cavity is usually largest in the cervical and upper dorsal regions of the cord, but it may be confined to the lower part of the cord. On the other hand, as just stated, it may extend from the end of the cord even to the pons. The cavity may have large dimensions as to length and be so wide as to convert the cord into a thin-walled tube.

It is because of these extraordinary changes that the Greek words *syrinx*, a tube, and *myelon*, marrow of the spine, is applied to it. So complete may be the excavating process that when the cord is severed from the medulla the fluid may escape and the cord flatten out like a ribbon. In some cases there are several cavities superimposed. On cross-section the cavity is usually found to be just back of the central canal in the gray commissure and in the posterior cornua, or it may be present where the central canal should be. Occasionally it affects the anterior horns or the lateral or posterior white columns.

The wall of the cavity consists in a well-developed mass of neuroglia (gliomatosis), which in its growth encroaches upon surrounding tissues and may cause definite symptoms before its centre becomes broken down. Some neurologists, on that account, prefer to call this disease, at least in its earliest stages, spinal gliomatosis, or gliosis.

The loss of pain sense and temperature sense which is so characteristic has been ascribed to pressure by the neuroglia mass upon the fibers conducting these sensations as they cross in the central gray matter on their way to the column of Gowers. This is at least a good working hypothesis.

Around the margin of the connective-tissue boundary just described there is usually an abnormal development of bloodvessels which are numerous, distorted, and larger than normal.

Dilatation of the central canal by serous fluid is called hydromyelia, and is ordinarily associated with hydrocephalus.

Symptoms.—The symptoms of syringomyelia consist in *loss of pain sense* and of *temperature sense*, so that the patient may be cut or burnt without feeling pain, although his sense of touch in the affected part is still preserved. In some cases the temperature sense may be preserved, or the sense of heat is lost and that of cold preserved, or *vice versa*. The areas in which the loss of pain sense exists are not symmetrical, but are irregularly distributed over the body. The fact that the lesion usually affects the cervical portion of the cord explains why it is that the areas of analgesia are usually found in the upper extremities. Associated with this impairment of pain sense there develop, as the disease advances, *nutritional changes* in the bones, muscles, and skin, and a progressive paralysis due to the muscle changes.

The outward evidences of trophic disturbance are usually first noticed in connection with some injury which fails to heal and, becoming infected, forces a recognition of its presence upon the patient, not by pain, but by his observation with his eyes that healing does not take place. In some instances, however, nutritional changes occur without any history of injury. *Felons* may develop. When they are accompanied by severe necrosis the condition is usually called "Morvan's disease." In still other cases the *finger-nails become deformed*, or *superficial gangrene* of the skin develops. The *shoulder-*, *elbow-*, and *wrist-joints* become *swollen*, filled with fluid, and absorption of the articulating surfaces takes place, the condition in the upper extremities in this disease being practically identical with that seen in the joints of the lower extremities in certain cases of locomotor ataxia.

The shafts of the long bones often suffer fracture, but these *fractures* are *painless* and only enforce attention because of the incapacity produced. Painless dislocations may be caused by insignificant traumata. *Curvature of the spine* may also occur, due to muscular atrophy, and perhaps to changes in the vertebræ. *Secondary contractures* may take place and produce great deformity of the hands, which, added to the progressive muscular atrophy and the paralysis, impairs the use of the upper extremities very much. In addition to these symptoms several special symptoms, dependent upon the site of the lesion, must be considered. Thus, if the *lower part of the spinal cord* is affected there may be *vesical* or *rectal paralysis*; whereas, if the upper cervical cord is affected there may be *unilateral retraction of the eyeball, narrowing of the palpebral opening*, and a *slow pupillary reaction* because of involvement of the spinal centre of the cervical sympathetic. When anæsthesia is found on the face it is ascribed to implication of the spinal root of the fifth nerve in the cervical region.

Diagnosis.—As already stated, the loss of pain sense with preservation of tactile sense, the trophic changes, and the muscular atrophy all form a picture which reveals syringomyelia. Before all these symptoms develop the presence of a slowly increasing muscular atrophy may mislead the physician into the diagnosis of chronic poliomyelitis or progressive muscular atrophy. Indeed, in its commonest type, syringomyelia presents the picture of progressive muscular atrophy when it is advanced, including the "claw-hand." Less frequently the white columns of the cord are pressed upon by the central mass and symptoms of locomotor ataxia or of lateral sclerosis are found. The true condition is recognized by the dissociated anæsthesia already described. Tumor in the cord usually produces so much pressure that the symptoms of paraplegia are more marked than in syringomyelia, and a tumor is usually associated with severe pain. The dactylitis of syringomyelia bears a resemblance to leprosy, which, however, does not reveal the more general signs of disease in the cord.

Prognosis.—The chance of recovery is of course *nil*, but as the disease progresses very slowly indeed, life may be prolonged for years.

Treatment.—There is no treatment for syringomyelia. The affected extremities should be carefully protected from injury.

HEMORRHAGE INTO THE SPINAL CORD.

Definition and Etiology.—Spontaneous hemorrhage into the spinal cord is a very rare accident, so rare that some writers have denied its existence except when it has arisen from a direct traumatism. Minute hemorrhages, of course, occur in severe forms of acute myelitis. The most common period of life for this accident to occur is between the twentieth and fortieth years, but it may occur in infants. Hemorrhage due to injury may, of course, develop at any time. Gowers cites a case of hemorrhage occurring apparently as the result of repeated sexual intercourse. Occasionally hemorrhage into the cord ensues in cases of asphyxia, as in coal-gas poisoning, and cases have been reported in which hæmophilia produced this lesion. The clot is usually found in these cases chiefly in the gray matter of the cord. Sometimes it is single; in other instances there are multiple clots. If the escape of blood has been copious the blood may perforate the white matter and find its way to the pia.

When the clot is of any size and the cord is examined shortly after the accident, changes resembling those due to hemorrhage into the brain are present. The cord is softened and infiltrated with small round cells and with red and white corpuscles which are seen to be undergoing granular change. The tissues are also stained by blood-coloring matter. If the patient lives for some weeks and then at death the cord is studied, there is found fatty degeneration of the neighboring tissues or a cicatrix of connective tissue which occupies the site of the hemorrhage. Secondary descending and ascending degenerations may ensue as in myelitis.

Symptoms.—The symptoms of hemorrhage into the spinal cord vary, of course, with the level at which the lesion takes place. The general symptoms are *paraplegia* with *loss of sensation* in the paralyzed limbs, and *loss* of *control* of the *bladder* and *rectum*. These symptoms are those of acute myelitis as well. In addition quite *severe pain* may be felt in the spine or be referred to the front of the thorax or to the epigastric region, and even to the legs.

When the hemorrhage is in the *cervical cord* there is *paralysis* of the *arms* as well as the *legs*. The *reflexes* are usually *lost* at first because of shock, but *soon reappear* and are *usually exaggerated* unless the cervical or the lumbar enlargement of the cord is affected, when they are permanently absent in the arms or in the legs. *Spastic contractions* may develop later from descending changes in the crossed pyramidal tracts, as in myelitis. The *muscles* may undergo *degenerative changes* very rapidly because of damage to the cells in the anterior horns of the gray matter at the level of the lesion.

Not rarely, if the hemorrhage has been at all large, a stage of *secondary irritation* and inflammation develops as a result of the extravasation of blood, and this may not only greatly increase the gravity of the symptoms, but destroy life. On the other hand, it not infrequently happens that the hemorrhage in the gray matter may not only destroy this part of the cord, but by pressure abrogate the function of the white matter.

After the acute process is over, the paraplegia is greatly decreased as the pressure is decreased, but complete recovery does not ensue because the cord is permanently damaged, and so atrophy of the muscles, governed by that part of the gray matter which has been damaged, ultimately develops as in acute poliomyelitis. Some degree of spasticity persists through loss of fibres in the pyramidal tracts, and various sensory defects if the dorsal columns have not completely recovered.

Diagnosis.—The diagnosis of hemorrhage in the cord is not to be made until the symptoms are so well defined that there can be little doubt as to their cause. The onset of the symptoms must be sudden, that is, almost instantaneous. If several hours are passed in their development it is probably a case of acute myelitis. Pain is also an important symptom, for, if it is present, it points to hemorrhage.

Prognosis.—This depends upon the severity of the symptoms and upon the site of the lesion. If it is in the cervical or lumbar enlargements, the prognosis is more grave than if it is in the dorsal cord. If bed-sores speedily develop the outlook is correspondingly bad not only because their presence shows grave lesions in the cord, but also because their existence is a menace to the patient's life.

Treatment.—Absolute rest in bed is essential. An ice-bag should be kept over the spine, and small doses of aconite and the bromides used to allay circulatory excitement. The use of ergot in such cases as commended by Gowers does not seem to be based upon a correct conception of the physiological action of this drug. Some time after the hemorrhage the iodides may be used to aid in clearing up the inflammatory exudate.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Definition and Etiology.—A hemorrhage about the spinal cord may be outside the dura mater (extrameningeal or extradural), or inside the dura mater (intrameningeal). If it is between the dura mater and the arachnoid it is called subdural, and if it is between the arachnoid and pia mater it is called subarachnoid.

Rupture of a vessel in the spinal meninges occurs usually in adult life, and more frequently in males than females. Its most common cause is injury to the spine. It has been known to follow violent convulsions and as a sequel to those infections which result in purpura. In newborn infants the blood found between the spinal membranes has its origin in the meninges of the brain, and follows the cord downward. Sometimes, in cases of very severe inflammation of the spinal meninges, small extravasations of blood take place.

In extradural hemorrhage the blood comes from the veins which lie between the dura and the bony canal. The quantity of blood which is poured out varies greatly. In some instances it reaches the full length of the cord. In other instances but a small area is covered by a clot. The most common seat for the hemorrhage is the cervical portion of the cord. The cord may or may not be compressed.

Subdural hemorrhage, that is, the escape of blood between the dura and the arachnoid, also varies greatly in quantity. In subarachnoid hemorrhage the blood comes from the vessels of the pia mater, and the clot may surround the cord for a few inches or extend throughout the whole subarachnoid space. In very rare instances it may actually force its way into the cerebral ventricles.

In all cases of hemorrhage into the spinal membranes, save the extradural type, the cerebrospinal fluid is blood-stained. This may be a valuable diagnostic point, since lumbar puncture may reveal the presence of blood in this fluid.

Symptoms.—No symptoms may be present unless the hemorrhage is extensive enough to cause compression, or unless a secondary meningeal inflammation develops. When the effusion of blood is considerable there is *sudden severe pain* in the *back,* usually about the level of the hemorrhage, which extends into the loins, and it may be to the anterior surface of the body. There may also be some *muscular spasm* in the parts involved by those nerves, the roots of which are pressed upon after leaving the spinal cord. These spasms may be severe enough to produce a *convulsion,* localized or general. Immediately after these symptoms evidences of *loss of power develop* and the symptoms resemble the early stages of acute myelitis, or of hemorrhage into the cord itself, save that it is rare for the paralysis of either sensation or motion to be as complete as it is in those conditions.

If the *hemorrhage* is in the *cervical region,* the *pain* is felt in the *neck* and *arms.* There is *difficulty of swallowing* and of *breathing,* and, it may be, *dilatation of the pupils.* When it is in the *dorsal region* the *pain* is in *the chest and abdomen,* and when in the *lumbar region* it is chiefly felt in the *legs. Consciousness is preserved* unless it be lost through shock. Some hours or days after the hemorrhage a secondary reaction with febrile movement may develop. In fatal cases death usually comes on within a few hours.

From a medicolegal standpoint it is interesting to note that at least one case of meningeal hemorrhage in the spine very closely resembled strychnine poisoning. If the symptoms are severe, death is very likely to occur within a few hours. If the patient survives the first few days, partial recovery from the paralysis may occur.

Prognosis.—The prognosis as to life is worse when the hemorrhage is high up in the meninges than when it is low down.

Treatment.—The treatment of this condition consists in absolute rest, the employment of small doses of the bromides and aconite as nervous and circulatory sedatives, and counterirritation over the back in the shape of dry cups or leeches. The patient should be made to lie on his side or on his face rather than on his back, in order to prevent the accumulation of extravasated blood at the posterior portion of the cord. Where the quantity of blood which is poured out is very large, and the symptoms so severe that death is threatened, it may be advisable to call upon a skilful surgeon to relieve pressure by operation.

COMPRESSION OF THE SPINAL CORD.

Definition and Etiology.—Compression of the spinal cord occurs as the result of disease of the vertebræ, of growths in the vertebræ or the meninges; of growths occurring within the spinal canal, inside or outside of the dura mater; of aneurysm of the aorta, which, by pressure on the vertebræ, cause their absorption; as the result of syphilitic inflammatory processes in the spinal canal, or by the development of a pachymeningitis, which may involve the cervical or lumbar portions of the cord, and which is characterized by a thickening of the parts involved. The result of pressure exercised by any of these causes interferes with the nutrition of the spinal cord and with the transmission of impulses along its tracts, and the symptoms which arise vary in their character and severity with the degree of pressure and the alterations caused by it.

Disease of the vertebræ is most commonly the result of tuberculous infection, particularly in children. As a result of this process, the bones become softened, give way under the pressure which is exerted upon them, and as they do so pressure upon the cord results. In other instances a suppurative process results in the development of so much pus that pressure is produced by it, and not uncommonly a carious process in the vertebræ is associated with an inflammation of the dura mater, with consequent thickening of this membrane, so that pressure is produced. Again, the caseous masses which are formed. or the overgrowth of connective tissue which takes place, may cause pressure. In some instances the dura mater suffers from tuberculous infection, and tubercles are found upon its inner surface, and both the arachnoid and pia mater may be involved. As a result the nutrition of the spinal cord at this point is impaired through interference with the circulation in its bloodvessels and probably also because of the obstruction to the circulation of lymph as well. The cord, in the majority of instances, is involved. If, however, the pressure is severe, there is apt to be an overgrowth of connective tissue whereby a sclerotic process is developed. The axis cylinders become swollen, and fatty globules can be found in the myelin sheaths. If the pressure is severe and is long continued, the cord may be markedly atrophied and the overgrowth of connective tissue be very great. In some instances there may be nothing left of the cord but a band of connective tissue. If the damage done to the cord is of a more severe type, ascending and descending degenerative changes occur.

The symptoms produced by these lesions consist in *pain* in the spine and in the distribution of the nerves supplying the trunk and limbs. Not infrequently pain will be felt in the abdominal wall or in the neighborhood of the sternum because of the irritation of the nerve trunks as they make their exit from the spinal cord, according to the well-known law that pain is frequently referred to the peripheral ends of the nerve affected. Any jarring of the body by a misstep or a sudden movement or even a gentle blow upon the spinal column may cause the patient suffering. The *muscles of the back* are *usually fixed*, in order to protect the spinal column as much as possible. This fixation is partly voluntary and partly involuntary. In some instances a *girdle sensation* is felt in the nerves which make their exit from the area which is diseased.

When the *lesions are high in the cord* there may be *painful sensations* in the arms, and if the lateral tracts are compressed there is an *exaggeration of the reflexes*, with a tendency to *spasticity of the muscles*. In other instances the patient may present all the symptoms of transverse myelitis and develop *bed-sores*. The rapidity with which these symptoms develop in different cases varies very much, depending entirely upon the activity of the pathological process in the spinal column. In some instances years are consumed in the development of the advanced stage of the malady. In others paralysis of the lower extremities may be produced in a few months.

The *diagnosis* of these cases is not difficult if the physician will carefully examine the spine.

The *prognosis* is, of course, not very favorable, but it is a noteworthy fact that in those stages in which the process in the spinal column becomes arrested a very marked degree of recovery may take place. On the other hand, it sometimes happens that spinal disease in infancy results in later life in the development of lateral sclerosis or other diseases of the spinal cord. Much depends in the way of prognosis upon what the surgeon is able to do for the spinal disease. The nervous symptoms are to be considered purely secondary, and every effort made to modify the pathological process in the spine.

The *treatment* of compression of the spinal cord due to disease of the vertebra is entirely in the hands of the orthopedic surgeon, who, by means of proper apparatus, can often do much good. The medicinal plan of treatment consists in the use of cod-liver oil, iron, and arsenic, the following of a perfectly healthy mode of life, and the use of good food. Pain is to be relieved, if necessary, by opiates, and the nervous twitchings by sedatives, like bromide and chloral. In some cases hydrotherapeutic measures are advantageous.

Malignant growths of the vertebræ, such as carcinoma and sarcoma, are rare. They are usually rapid in their growth and produce symptoms of spinal compression as soon as they invade the spinal canal. These malignant growths soon penetrate the dura, the arachnoid, and the pia, and speedily infiltrate the spinal cord itself, although the dura mater is usually capable of protecting the spinal cord from direct infection when the disease is tuberculous.

When a tumor of the spinal cord develops it is in the great majority of instances due to sarcoma. Tumor of the spinal cord is, however, exceedingly rare. Schlesinger found only 147 spinal tumors in 35,000 autopsies, and Starr states that the ratio of tumors of the spinal cord to tumors of the brain is 1 to 13.

Here, again, the symptoms consist, as a rule, in intense neuralgic pain of a shooting or stabbing character caused by pressure upon the nerves as they leave the spinal canal. These pains are more severe than those produced by any other form of spinal disease, and they are felt in different portions of the body, according to the portion of the spinal cord which is involved. If the lower cervical portion of the cord is affected, the pain may be felt in one or both hands and forearms. If the growth is in the *upper cervical region*, they are felt in the shoulder or neck; if it occurs as low as the *sixth dorsal segment* the *pain is felt* in the chest, *near the nipple*; in the tenth dorsal segment it is felt in the *abdomen* and *groin*. When a tumor compresses the cord in its entire thickness degenerations ensue, descending in the lateral columns, ascending in the dorsal and other sensory columns, just as they arise in cases of marked caries of the vertebræ. Under these conditions the symptoms are those of a transverse myelitis. The level at which the tumor is growing can largely be determined by localizing the symptoms, and the fact that tumor is present may be pointed to by the presence of growths elsewhere in the body. If the tumor is of the malignant type its growth is usually exceedingy rapid.

Spinal symptoms due to new-growth differ from those due to caries of the spine by the fact that the stillness of the muscles of the back and tendons on jarring the spine is not so marked in growths as it is in tuberculous disease. In the latter case, also, there may be found a primary tuberculous focus.

When compression is due to tumor the treatment is operative. In many instances it is possible to give the patient some relief by this means. Starr has collected 58 cases of tumor of the spinal cord in which an operation was attempted. In all his cases the tumor was found, and in 16 of them the patients recovered If the growth is malignant the possibility of doing much good by operation is, of course, remote. Pain may, however, be temporarily relieved by the removal of the pressure.

The compression of the spinal cord produced by gummatous growths or by syphilitic exudations about the spinal cord present symptoms which also depend upon the area which is involved, particularly upon the level of the lesion. The condition may arise either in acquired or in hereditary syphilis, and the diagnosis is made by the history of the patient and the presence of pressure symptoms. The treatment is antisyphilitic.

When an *aneurysm* of the aorta grows in such a manner that it erodes the vertebræ, it may produce symptoms of compression of the spinal cord. Thus, paraplegia may be developed, or severe pain may be felt in those parts of the body which are supplied by the nerve trunks which have their origin in that portion of the spinal cord which is affected. If the physical signs of the presence of aneurysm are demonstrable the diagnosis is not difficult, but if

the growth is in a backward direction it may pres
indicate its presence. An examination of the patie
reveal signs of vertebral disease, but a bruit or
and a history of syphilis and of trauma, if add
the bloodvessels are sclerotic, will aid in discove
symptoms.

A sixth cause of compression of the spinal cord
pachymeningitis, the *pachymeningitis cervicalis hy*
This disease consists in a thickening of the dura ma
the spinal cord and the spinal nerves as they pass thro
upon. As a result the spinal cord suffers from menin
mater becomes adherent to the pia mater. Someti
asations occur under the dura, and there is usually
nective tissue about the bloodvessels, both in and a

The *symptoms* of this form of meningomyelitis
already described as occurring in cases in which th
other causes in the cervical region, but they have ce
may aid in the diagnosis of the condition. There is
head and neck, with a certain degree of *stiffness* an
The pain radiates down into the hands and arms,
severe and neuralgic in type. Patches of *anæsthesi*
present and localized muscular spasms may occur, f
and the development of reactions of degeneration
lasted long enough to interfere with the transmissi
from the cord to the muscles affected. Finally, i
great enough to seriously impair the nutrition of th
develop symptoms of spastic paraplegia due to d
changes in the lateral tracts. If the muscles sup
median nerves are chiefly affected, the disease is p
of the enlargement; but if those muscles supplied by
lose power, the upper part of the cervical enlarge
of power in the triceps, anconeus, supinator longus
longior, and the brachialis anticus, therefore indica
segment; whereas, a loss of power in the flexor c
profundus digitorum (ulnar nerve) and all the musc
the forearm and thumb (median nerve) indicates
segment. In some cases myosis from paralysis of t
may be present.

Treatment of cervical pachymeningitis promises
posed from the character of the lesions, probably
so often due to syphilis. Active counterirritation of
by the electrocautery and the free use of the protio
nating with large doses of iodide of potassium, sh
to. Pain is to be relieved by the use of acetanili
these drugs fail to give relief they must be combined

SPINAL MENINGITIS.

Definition and Etiology.—A condition of inflammation of the membranes covering the spinal cord is practically always secondary to some lesion at another part of the body. In some instances the specific micro-organism of croupous pneumonia, of enteric fever, of acute articular rheumatism, or septicæmia finds its way to these parts and causes the pathological process. In other instances tuberculosis is the cause, whether it be primarily present in distant parts of the body or in the vertebral column. In some instances an injury affords a means of entrance to the body for micro-organisms, which attack the spinal meninges, particularly if the vital resistance has been lowered by an accident. Spinal meningitis may also arise from cerebral meningitis by direct extension of an infection.

Pathology and Morbid Anatomy.—Following the stage of acute hyperæmia present in all acute inflammatory processes, there is an excess of serous fluid poured out between the dura mater and the pia, which fluid may, at autopsy, be found to be purulent. Patches of fibrinous exudate are found on the surface of the pia mater, the bloodvessels of which are engorged with blood and often suffer from small hemorrhagic extravasations. The spinal cord, the pia and the dura mater are often adherent. After the inflammatory process has been present for some time, the pia mater becomes much thickened by the development of connective tissue. The secondary changes which ensue consist in an inflammatory process which affects the superficial parts of the spinal cord and causes degenerative changes in the spinal nerve roots, the axis cylinders of which become swollen. Fatty globules appear in the myelin sheath. The changes in the spinal cord are most marked in the posterior and lateral columns, and in subacute or chronic cases these areas are affected by an overgrowth of connective tissue, which ultimately produces sclerotic patches.

Symptoms.—The onset of acute spinal meningitis develops, as do most acute inflammations of serous membranes, with *pain*, *chill*, *fever*, and *general wretchedness*. The *pain* is felt in the *back* and *limbs*, and is greatly increased by movements. There is also a state of *hyperæsthesia* of all the spinal nerves, so that touching the patient may cause great suffering. It is soon noticed that the *patient is stiff* and more or less fixed by muscular rigidity, which is, in part, due to the pain produced by movement and to the irritation of the nerves as they pass from the spinal cord. The stiffness of the muscles of the back and of the neck is the most marked. At this time "Kernig's sign" is developed, which consists in an inability of the physician to straighten the patient's leg at the knee after the thigh has been flexed to a right angle with the trunk. This state is due to spasm of the flexor muscles induced by the irritation at the point of exit of the nerve trunks. There is often soon developed an *increase in the reflexes*, chiefly in the legs, and this in turn is succeeded by *paralysis* and *final loss of reflexes* if the process is severe and prolonged. Along with these symptoms there speedily develops a *paralysis* of the *bladder* and *rectum*, so that there is *retention* or *incontinence of urine* and *incontinence of feces*. There may also be *paralytic incontinence*.

Because of the lesions produced in the nerve roots and in the spinal nerves as they pierce the meninges, *trophic changes in the skin* may develop, as shown by localized areas of pallor and congestion and the speedy development of *bed-sores.*

If the disease is severe and the inflammatory process spreads until the upper portions of the cord are involved, death may ensue by reason of the inflammation reaching the level of the medulla and causing fatal disturbance of the function of respiration or of the heart. In such cases *Cheyne-Stokes breathing* and irregularity of the pulse may be the symptoms of impending dissolution. Not rarely the development of paralysis of the cranial nerves with *convulsions* and *coma* precede death. Death may come within a few days of onset or after several weeks. In the severe cases which recover the patient often permanently suffers from localized palsies, anæsthesias, and atrophic lesions in the skin and muscles.

Diagnosis.—Aside from the character of the symptoms just described, which points strongly to meningitis, we may resort to lumbar puncture for the purpose of making the diagnosis more certain. A strong, hollow needle attached to a syringe, so that it may be easily handled, or a small trocar and cannula are passed into the spinal canal between the third and fourth lumbar vertebræ, on a line drawn between the crests of the ilia. The direction of the needle should be slightly to one side and upward. (For further details as to this method see Cerebrospinal Fever.) As soon as it enters the spinal canal the cerebrospinal fluid will escape, drop by drop, or with a squirt, if the pressure is great. This fluid should be examined for bacteria to determine the nature of the infection. If the fluid contains disintegrated blood, the cause of the affection is a pachymeningitis or an injury. If fresh blood is present, the blood is probably due to the puncture. If the fluid is clear there is probably no true meningitis present. In cases of tuberculosis of the meninges it is usually quite cloudy. If it contains pus a purulent meningitis is present. If inflammation of the meninges is present no sugar will be found in the fluid. A study of the leukocytes in the cerebrospinal fluid may throw light upon the case. They are much increased in number in acute inflammatory processes. In chronic conditions the mononuclear cells are particularly increased.

Prognosis.—Tuberculous meningitis is, of course, a state giving a hopeless prognosis. Septic cases are also grave. Those types due to pneumonia and typhoid fever sometimes recover. (See Pneumonia and Typhoid Fever.)

Treatment.—The treatment of spinal meningitis consists in absolute rest, the patient being placed upon a soft bed. In some instances if there is any sign of bed-sores it is essential that an air-bed or water-bed should be used. The application of blisters or the actual cautery has been recommended, but in view of the possibility of bed-sores developing, it is questionable whether their use is safe. The same objection holds in regard to such forms of counterirritation as cupping and leeching. Twitchings or cramps of the muscles are to be relieved by the administration of sedatives to the spinal cord, such as bromide and chloral, and if the pain is very severe morphine must be used. At one time it was believed that full doses of calomel combined with opium were exceedingly valuable in the treatment of acute inflammation of all serous membranes, particularly those covering the brain and spinal cord. At the present time this method of treatment has almost entirely ceased, but in certain instances it would seem advisable to have recourse to it. The object is to give enough mercurial to exercise its so-called antiphlogistic influence, and to use the opium not only for the relief of pain, but for the purpose of preventing the calomel from purging the patient. The mercurial may be pushed until slight tenderness of the gums is manifested.

Chronic Spinal Meningitis. **Etiology.**—Chronic spinal meningitis is said to occasionally have its origin in an acute inflammation of the meninges of the spinal cord. In all probability, however, such an origin is exceedingly rare, and in the majority of instances it is the result of syphilitic infection, whereby there is a thickening of the dura and the formation of an abnormal quantity of serum and connective tissue under it. As the result of the chronic inflammatory process in the membrane, a somewhat similar one is set up in the spinal cord near its surface, producing a meningomyelitis, which is in its nature closely allied to the acute form of meningitis just considered. There is always present a thickening of the bloodvessels, a small-cell infiltration about their walls, and, if the process is severe, an obliterating endarteritis. Sometimes gummatous masses are formed. In most of these cases there is also present cerebral meningitis as well.

Symptoms.—The symptoms of chronic spinal meningitis consist in *stiffness of the back* and *extremities*, with *pains* and *cramps*. There are also *disturbances in sensibility*, some portions of the skin being hyperæsthetic, others anæsthetic. *Motor power* is also *impaired*, and if the inflammation is in the lower portion of the spinal cord there may be interference with the function of the bladder or rectum.

Diagnosis.—Chronic spinal meningitis is to be recognized by the presence of the symptoms just described and by the use of lumbar puncture, which, if meningitis is present, will show an increased quantity of cerebrospinal fluid, which is usually under pressure, and which will, therefore, escape from the needle with a spurt. Care must be taken that ordinary lumbago with spasm of the muscles of the back and fixation and pain is not confused with this condition. Myelitis is to be separated by the absence of severe pain and of cramps in the extremities, and by the presence of paraplegia.

Treatment.—The treatment of chronic meningitis consists, as must be evident from its cause, namely, late syphilis, in the free use of protiodide of mercury and the iodide of potassium, given until a full physiological effect is produced. In other words, it is not a question of grains administered, but effects obtained. These cases are usually much benefited by going to the various hot springs, because, in addition to the use of mercurials by the mouth, they permit the simultaneous use of hot baths and mercurial inunctions. Everything should be done to keep the general condition of the patient at the highest possible level approaching that of health.

ACUTE ASCENDING PARALYSIS (LANDRY'S PARALYSIS).

Definition.—This is an acute ascending paralysis beginning in the lower extremities and rapidly passing upward until it involves the muscles of the trunk and upper extremities, finally causing death by failure of respiration. The condition is a very rare one. It must be clearly separated from those forms of acute ascending paralysis due to an acute ascending myelitis, or to a hemorrhage into the spinal membranes, and from ascending peripheral neuritis. It is a symptom-complex which results from a lesion of the lower segment of the motor pathway, either in the cord and bulb or in the peripheral nerves. It is sometimes called acute progressive paralysis, or Landry's paralysis, having been first described by Landry in 1859. Rare cases have been recorded in which the disease has begun in the arms and passed to the legs.

Etiology.—The exact etiological causes of this malady are not understood. The disease occurs more frequently in males than in females, and its most common period of occurrence is between twenty and forty years of age. In all probability every case is due to an infection of the peripheral nerves and spinal cord, for it sometimes follows an acute illness due to a micro-organism, as, for example, influenza, smallpox, erysipelas, typhoid fever, and pelvic peritonitis. The excessive use of alcohol has seemed in some cases to be a powerful predisposing cause, and cases have been recorded in which, in the presence of a history of syphilis, the malady has been arrested by the use of specific remedies.

Pathology and Morbid Anatomy.—The lesions found at autopsy in a case of Landry's paralysis are by no means constant in all cases. In some instances the chief lesions have been found in the spinal cord, in others in the peripheral nerves and nerve roots. In the spinal cord the lesions described in some cases have been practically identical with those of acute disseminated myelitis, and in others they have been identical with those met with in severe peripheral neuritis. In every case, however, it is evident that the peripheral motor neurones are the portions of the nervous system most affected.

Symptoms.—In some cases of acute ascending paralysis the onset of the paralytic symptoms is preceded for a few hours by a sense of *general wretchedness*, with *tingling* or *pain* in the *limbs* or *back*. This is followed by a rapidly increasing *weakness* in the *lower limbs*, which may amount to a complete loss of power in from a few hours to several days. The muscles of the lower part of the trunk are next involved, and finally the muscles of the arms and of the upper thorax fall victims to the rapidly spreading malady. The *respiration* becomes *difficult*, the *speech indistinct*, and *dysphagia* may be present. *Sensation* in the paralyzed parts may be *impaired*, but it is not lost. The *reflexes are decreased*, and perhaps lost, but they may be restored later on and ultimately become excessive. Muscular atrophy does not develop even when the patient survives for weeks, and the sphincters usually, but not always, retain their power. Bed-sores do not develop. The mind nearly always remains clear, and the temperature is usually not elevated. Very rare cases have been reported in which the paralysis has been of the acute descending type, the arms being the parts first affected. In such cases fatal bulbar paralysis may occur before the lower parts of the body are affected.

Diagnosis.—Acute ascending paralysis is to be separated from acute poliomyelitis by the absence of rapid atrophy. From the paralysis due to an acute hemorrhage into the spinal meninges it is separated by the absence of pain and of spasm. From an acute ascending myelitis it is separated by the fact that there is no loss of sensation, that the sphincters are unaffected, and that the paralysis progresses more rapidly.

Prognosis.—The prognosis depends upon the state of the respiratory centre and the lungs, and upon the condition of the centres governing cardiac action. If these parts are involved, death, of course, speedily ends the case. Death may come in a few hours or days, or not for several weeks. Cerebral and bulbar symptoms are always grave. A fatal ending usually occurs, but cases sometimes improve and recovery may occur.

Treatment.—The treatment of a patient suffering from Landry's paralysis should be almost identical with that advised in cases of acute myelitis. A warm bath may be given to draw the blood to the surface, and moderate counterirritation should be applied over the vertebræ. It is absolutely essential that perfect rest be obtained, and that the patient shall lie upon the side rather than upon his back, since the dorsal position may increase the tendency to congestion of the cord. As the disease is probably dependent upon infection and toxæmia, the skin should be kept active by mild diaphoretics, and the kidneys should be stimulated to activity by the use of the vegetable salts of potash or other mild diuretics. Some writers recommend the administration of salicylate of sodium, although they do not seem to be able to explain how it can do good. Gowers speaks highly of the use of ergotin, and mentions the case of a man of fifty-seven who developed symptoms of Landry's paralysis after exposure to cold and wet. To this patient ergotin was given every hour till 20 grains had been taken, when the symptoms became markedly improved and the patient speedily recovered, so that by the end of a week he was well. One can not help feeling that in all probability the ergotin had little to do with this remarkable recovery, as it is hard to see how this drug could be useful in combating an infection which was so severe in its nervous effects. When there is a history of syphilis the protiodide of mercury should be administered freely.

CAISSON DISEASE.

Caisson disease is a condition met with in persons who have been exposed to high atmospheric pressures for a number of hours, and is particularly prone to develop if severe toil has been maintained during the exposure. The disease is usually met with in artisans, or laborers, who are engaged in the building of piers or foundations many feet under water, where it is necessary to have a pressure of several atmospheres in order to keep the caisson dry. In some instances the pressure is as great as ninety pounds to the square inch. The atmosphere in the caisson often has a high proportion of humidity, and the temperature may also be quite high. The symptoms develop when the workman leaves the caisson and is exposed to normal atmospheric pressure. In mild cases nothing more than a feeling of dizziness and vertigo develop, associated, it may be, with neuralgic pains in the head. In severe cases the neuralgic pains become excruciating, so that the patient feels as if his muscles were being stripped from his bones, and this pain is followed by a loss of both motion and sensation in the lower limbs, although the patient still complains of the pain. Often nausea and vomiting are present, accompanied by violent epigastric paroxysms of pain. Occasionally, there is loss of power in the sphincters. In some instances pain is absent, but paralysis is present, paralysis being the more constant symptom. In still more severe cases coma develops, in which case death invariably results. The prognosis is generally favorable unless the symptoms of paralysis and pain are unusually severe. The mildest cases rarely last over twelve hours, and sometimes only three or four; but in severe cases, recovery may not take place for days or weeks.

The pathology of this curious condition is not well understood. (A discussion of many of the views concerning it will be found in the author's Fiske Fund Prize Essay for 1886.[1]) It would seem probable that the symptoms are largely dependent upon disorder of the circulation in the central nervous system. Air emboli were found in the small arteries of the posterolateral tracts of the spinal cord, also secondary softening was observed. Those who have had the most experience with the disease in this country are Jaminet, Bauer, and Woodward, who made many observations during the building of the St. Louis Bridge, and A. H. Smith, of New York, who studied it during the building of the Brooklyn Bridge.

Treatment.—The treatment is both prophylactic and palliative. The prophylaxis consists in having superimposed air chambers, each one having a different pressure so that the workmen may pass by degrees from the high-pressure caisson to the pressure of atmospheric air. It is also advisable to have the workmen brought to the surface with as little muscular effort as possible. In the building of the St. Louis Bridge, thirteen of the men who were employed in the building of the east pier, which was sunk 127 feet below high-water mark, died; but in building the east abutment, which was sunk five feet deeper, only one man died. In the latter case the workmen were lifted to the surface by an elevator instead of having to climb a ladder.

When the symptoms come on, morphine should be given hypodermically in adequate dose. A. H. Smith considered that ergot is of value. Hot compresses should be wrung out and applied to the feet and spine. Jaminet recommends a draught of a strong alcoholic stimulant, with ginger.

The whole object of the physician should be to re-establish and equalize the circulation. If the pulse is full and the heart laboring, venesection should be freely employed. Sometimes relief can be obtained by returning the patient to the caisson. In some instances a small caisson for the resuscitation of workmen has been built on the surface, with advantageous results.

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[1] New and Altered Forms of Disease due to the Advance of Civilization in the Last Half Century.

DISEASES IN WHICH THE CHIEF MANIFESTATIONS ARE IN THE NERVES.

NEURITIS.

Definition.—Neuritis is an inflammation of a nerve. When the inflammatory process chiefly involves the perineurium it is called "perineuritis;" if the tissues surrounding the nerve bundles and between the nerve fibres are affected, it is an "interstitial neuritis," and if the nerve fibres themselves are primarily affected, it is said to be a "parenchymatous neuritis." The latter condition is usually a subacute or chronic process and is characterized by degenerative changes in the nerve fibres. The distinction, however, between these different forms is theoretical rather than clinical, because it is not possible to draw a definite line between them in most cases.

Etiology.—The causes of neuritis are very numerous. Any injury to a nerve trunk, as by a blow, stretching, or a wound, may give rise to the inflammatory process, as may also tumors, which by pressure cause irritation. Sometimes the use of a tool or a crutch may, by constant pressure on a nerve, produce neuritis. Various infectious diseases and the abuse of alcohol may produce it, as may also many of the metallic poisons. In those cases, however, in which the malady arises as the result of a poison, the neuritis is usually a multiple neuritis and does not affect one nerve alone. (See Multiple Neuritis.) Gout, and lithæmic states, also cause it.

Pathology and Morbid Anatomy.—The nerve, which is acutely inflamed, is found on examination to be red and swollen and lacking its usual lustre; the bloodvessels supplying it are hyperæmic or congested. If the process has been present for some time the nerve may be marked by swellings due to overgrowth of connective tissue and its endoneurium may be infiltrated by small cells. At this time one of three processes develops. Either the results of the acute inflammatory process undergo resolution or the inflammation becomes so severe that the nerve is destroyed, or if the process is more moderate, but continued, there is an overgrowth of connective tissue and gradual atrophy and loss of function. Microscopically, the inflamed nerve presents additional changes which serve to separate the parenchymatous form of the disease from that in which the perineurium and interstitial tissues are chiefly affected. In the parenchymatous type the myelin is opaque and swollen and soon undergoes segmentation with granular material between the segments. The axis cylinder may be continuous or broken into segments corresponding to the breaks in the myelin. Finally, the myelin and cylinder entirely disappear and only the nerve sheath containing a little granular matter is left. This last state may also arise as a result of the interstitial form of neuritis, but in this form the nerve is, in the early stage of the inflammation, more swollen and congested and the sheath filled with serum or purulent exudate.

Symptoms.—The symptoms of neuritis vary over a wide range in severity. When the inflammatory process is very mild so that the normal function is but slightly perverted, as from moderate pressure, a *tingling sensation* is felt or, in its place, a sense of *numbness* is experienced. This is called *paræsthesia* and can scarcely be said to be due to any real change in the nutrition of the nerve.

When the change in the nerve is more severe the symptoms are more definite. *Tingling* or *pricking sensations* may be present not only at the site of the lesion, but at the peripheral part of the nerve. If the damage is the result of pressure there is rarely any pain, and motor paralysis, more or less complete, is present instead. If, on the other hand, the lesion is associated with any wound, and an infection of the nerve has taken place, then *pain* is usually present and is often severe. Pressure upon the inflamed nerve trunk by the finger-tips also increases the pain not only at the point of pressure, but at the end of the nerve as well. After the process in the nerve is so advanced that its function is greatly impaired, trophic changes occur in the muscles and skin, the former *wasting* and the *skin becoming glossy*. The *muscles cease to respond to faradic electricity* and later fail to respond to galvanic electricity, the reactions of degeneration being first developed. Injuries to parts supplied by the nerve affected may result in sloughing, but sores rarely develop unless an injury is suffered.

When recovery begins the electrical contractility to galvanic stimulation first returns in part and later the power of voluntary movement.

Diagnosis.—The diagnosis of neuritis involving a single nerve, or several nerves in nearly related parts, is not difficult, for the pain is limited to the area of the nerve, as is also the anæsthesia or hyperæsthesia and loss of power. The condition is also pointed to by the history of injury or of some diathetic state which produces the affection. Pressure on the nerve trunk will elicit pain.

Prognosis.—The outlook for recovery in most cases of neuritis is quite good because of the extraordinary power of regeneration possessed by nerves. Even if the damage to the nerve has been so severe that its function is abolished by the division of all its conducting fibres, the function can be restored by the surgeon, who, by excising the destroyed portion and joining the distal and proximal ends together, may re-establish the pathway for both sensory and motor impulses. So rapid is the regeneration that if a nerve is severed by accident, and immediately sewed together, power may return in two weeks. When the damage has been done by pressure or inflammation the recovery rarely ensues in less than six weeks, and even eight months or a year may be consumed in the regenerative process. When recovery fails to occur and it is believed that only a small part of the nerve is diseased surgical procedures are necessary, but if there is reason to believe that permanent damage to a large part of a nerve has taken place, then the prognosis is hopeless. Sometimes the mere exposure of the nerve and the breaking up of adhesions or exudates that cause pressure is sufficient to produce recovery.

Treatment.—The treatment of neuritis may be divided into two parts, that devoted to the relief of pain and that to the abatement of the inflammation and to the regeneration of normal function. For the relief of pain the part affected may be wrapped in lint, which is heavily smeared with equal parts of an ointment of ichthyol and lanolin, outside of which is placed some oil-silk to retain moisture. In some instances a hot poultice of flaxseed or hot compresses may be used in the earlier stages to diminish the activity of the inflammatory process. If the pain is so severe that sleep is interfered with, the various coal-tar products may be employed, of which the most valuable are phenacetin in the dose of 5 grains four or five times a day, if need be. Acetanilid may be given in similar dose, and antipyrin in slightly larger dose. In other cases better results accrue if to these products of the coal-tar group are added small doses of codeine or morphine. If the pain does not yield to these remedies, hypodermic injections of morphine may be necessary for a short time, but their continued use is dangerous, as the patient only too readily develops the morphine habit. If the neuritis affects the arm or some portion of the body which does not by its disability force the patient to lie in bed, it is essential that the part involved shall be placed at rest. Thus, if the arm is affected it should be carried in a sling, and it may be necessary to protect it by a splint.

For the restoration of function no therapeutic measure should be instituted beyond those already named until the acute stage of the inflammatory process has ceased. When it is evident that the acute process is no longer present, strychnine or nux vomica may be given internally in full doses. These may also be combined with phosphorus and small quantities of quinine. The area of the skin which is supplied by the affected nerve should also be stimulated by the application of faradic electricity, care being taken that the current employed is not so strong as to damage the part. It is a good rule never to use a current so strong as to produce suffering. This method of treatment not only tends to rapidly restore sensation in the paralyzed part, but also to bring back faradic contractility in the muscle, and so ultimately restore motor power. If, however, the parts fail to respond, then galvanic electricity must be used, and the current interrupted so as to produce a stimulant effect. In some instances the muscle seems to respond better to the negative than to the positive pole, much depending of course upon the stage of degeneration which is present. Care should be taken that the applications of electricity are not prolonged for more than a few minutes at a time, and that they are not made oftener than once a day. Additional measures for improving the nutrition of the part are massage and manipulation. None of these measures should, however, be employed if there is tenderness in the nerve trunk or if they produce exhaustion in the parts affected. Indeed, it is possible in some instances to produce injuries of the nerve by too active manipulation. It must be remembered that the electricity, the massage, and the Swedish movements only do good by increasing the circulation and modifying the nutritional processes in the parts affected. If anæsthesia of an extremity exposes it to injury, by reason of the patient being unconscious of the presence of heat or cold, or of objects which are capable of doing damage, the part should be carefully protected by a splint or soft dressing.

Special Forms of Neuritis.

Cervicobrachial Neuritis.—Cervicobrachial neuritis follows injuries to the neck and shoulder, and is usually produced by falls or severe blows. Symptoms may also arise as the result of disease of the vertebræ or from an aneurysm. In other cases the acute infectious diseases or gouty or rheumatic conditions, associated with exposure to cold, seem to be responsible for the condition. The symptoms depend to a large extent upon the portion of the cervicobrachial plexus which is affected. If the *four upper cervical nerves* are involved, severe *pain* in the neighborhood of the *occiput* is felt, and the head is held in a fixed position because movement increases the suffering. When the *fifth or sixth cervical nerves* are involved, the *pain* is in the *neck* and the upper portion of the shoulder and axilla, and it may be felt down the back of the arm. Whereas, when the lower cervical nerves are affected, including the branches of the first dorsal, the pain is clavicular and axillary in the area of its distribution and extends down the front of the arm and forearm into the fingers. There is also *loss of power* in all the muscles which are supplied by the nerves making up the cervicobrachial plexus when their fibres are involved. Because of the fact that the cervical sympathetic nerve receives fibres from this part of the spinal cord it sometimes happens that ocular symptoms accompany manifestations of the neuritis in the lower portions of the cervicobrachial plexus, with the result that there may be retraction of the eyeball and narrowing of the palpebral fissure, with a mild degree of myosis and some pallor of the side of the face affected. The skin on this side is dry and does not become flushed on exercise. Cases of neuritis of the cervicobrachial plexus show marked evidences of pain when pressure is made over the plexus or when the arm is moved away from the body.

In some instances of cervicobrachial neuritis of very sudden onset actual hemorrhage may occur into the sheath of the nerves, the so-called "apoplectic neuritis." The same numbness and tingling as has been described under neuritis in general, followed by loss of sensation and of motor power, occurs in cervicobrachial neuritis. Trophic changes in the skin and muscles take place, and the reactions of degeneration develop.

A form of cervicobrachial neuritis which affects children is sometimes called obstetrical paralysis, or birth palsy, and is due to damage of the cervicobrachial plexus in parturition.

Prognosis.—The prognosis in cases of cervicobrachial neuritis, like that of neuritis in general, is good provided the injury has not been so severe as to sever the nerve fibres, and provided the condition has not lasted too long.

Treatment.—The treatment consists in absolute rest, the employment of hot compresses if the condition is seen early in its course, and later on the constant use of counterirritation. Rubbing the parts with some stimulating liniment like that of chloroform or ammonia is of value. Electricity, massage, and Swedish movements are to be resorted to after all evidence of acute inflammation is passed. The rest of the treatment is identical with that given for neuritis.

Obstetrical or Birth Palsy.—A paralysis of brachial nerves is a frequent occurrence in difficult and protracted labors, especially in cases of breech presentation. Pressure with the finger or tenaculum introduced into the axilla in order to facilitate delivery will injure the nerves and produce paralysis. In birth palsy the following muscles are affected: deltoid, biceps, supinator longus, and infraspinatus. This leads to inward rotation of the arm, extension of the forearm, and pronation of the hand. The paralysis is soon followed by atrophy of the muscles. The prognosis of birth palsy is, generally speaking, favorable, except when reaction of degeneration exists. As to treatment, massage and electricity are the only means, and should commence as early as possible.

Multiple Neuritis. **Definition and Etiology.**—Multiple neuritis, sometimes called " polyneuritis" or " peripheral neuritis," is a condition in which a large number of the peripheral nerves of the body suffer from subacute or chronic inflammation as a result of the action of some toxic agent. These toxic agents may be derived from external or internal sources. The external agents are alcohol, lead, arsenic, copper, mercury, anilin, carbon monoxide, and carbon bisulphide. The internal agents are the poisons developed in the various acute infectious fevers, as typhoid fever, smallpox, scarlet fever, influenza, erysipelas, pneumonia, diphtheria, dysentery, and other infectious maladies. Occasionally, too, a multiple neuritis develops as a complication of septic infection, either that following a wound or occurring during the puerperal period. Cases have also been recorded in which the toxic substance apparently has arisen from decomposition changes in the intestines. In some instances syphilis, tuberculosis, diabetes mellitus, and malarial fever have seemed to be provoking agents, but in all probability these affections act indirectly by rendering the nerve trunks susceptible to the action of the poison. A special form of multiple neuritis is that which is known as beriberi or " kakke." (See Beriberi.) In several instances small epidemics of multiple neuritis have been described. Multiple neuritis occurs most frequently between the twentieth and the fiftieth year of age, and is very rare in children, unless it is due to diphtheria. Whatever may be the cause of an attack of multiple neuritis, the pathological changes which are found in the affected nerves do not differ greatly from those already described as occurring in ordinary neuritis of a more limited extent.

Symptoms.—The symptoms of multiple neuritis, be the cause what it may, are fairly constant, although slight variations in the character of the symptoms occur according to the peculiar influence exercised by the poison. As *alcoholic neuritis* is the type most frequently met with, a description of this disease may be used for all forms of multiple neuritis. At the beginning of the malady there may be some slight elevation of temperature, but in many cases this does not occur. The patient first complains of *tingling* or *numbness* in the feet and fingers. In other cases *dull pain* may be experienced. Rarely this pain may be severe. These disturbances of sensation are usually increased by moving the affected limb and by deep or superficial pressure over the nerve trunks, and especially by deep pressure upon the muscle bellies of the forearms and of the calves.

Following these symptoms weakness develops, and it may become so severe that the patient is unable to move his hands or feet, and *foot-drop* or *wrist-drop* may develop. After the paralysis has lasted for some little time, some wasting of the muscles of the affected parts takes place. The *reflexes* are *diminished* or altogether arrested.

A peculiarity of the paralysis of peripheral neuritis is that very often it does not involve all the nerves of a limb. Thus, it not infrequently happens that the peroneal nerves suffer chiefly. In other instances the tibialis posticus is chiefly affected; and it is only when the condition is unusually severe that a complete paraplegia is present. In the arms the musculospiral nerve is most commonly affected. It is a noteworthy fact that the paralysis is usually symmetrical. In some instances the symptoms are more sensory than motor, but this is rarely the case. Sensory symptoms are, however, very constant in alcoholic neuritis, and in most of the other forms. They may, however, be absent or be very slight, as in neuritis due to lead, and in such forms of infectious disease as diphtheria and influenza. These forms of neuritis are often spoken of as "motor neuritis" to indicate that the sensory functions escape.

In addition to the numbness and tingling already mentioned, patches of anæsthesia and hyperæsthesia are often found existing near one another or even coinciding. So, too, there may be a hypersensitiveness to pain and a loss of the sense of touch, or *vice versa.*

Not infrequently the affection develops a train of symptoms which are so exactly like those met with in locomotor ataxia that even the most skilful neurologist may have difficulty in differentiating the two diseases. In other words, a so-called "pseudotabes" due to multiple neuritis is present. This resemblance depends upon the fact that the fibres of "muscle sense" which are affected in their spinal course (posterior columns) in tabes are implicated in such cases of multiple neuritis at their origins in the joints and muscle fasciæ. The presence of the Argyll-Robertson pupil in true ataxia, however, usually determines that the case is not one of peripheral neuritis. As an illustration of how closely multiple neuritis may resemble locomotor ataxia, cases have been reported in which perforating ulcer of the foot occurred.

Aside from the trophic changes already spoken of as occurring in the muscles, local disorders of blood supply and secretion are often present. There may be areas of skin which suffer from excessive sweating. In other cases localized patches of œdema are found, and rarely the joints become swollen, so that the case resembles acute articular rheumatism. Actual breaking down of the skin as the result of trophic changes, however, rarely occurs. The bladder and rectum are usually unaffected, and this aids materially in separating the paraplegia of severe neuritis from that due to myelitis. Occasionally, however, this valuable aid to differentiation fails us, and retention or incontinence of urine or feces is present.

Associated with these evidences of impairment in function in the peripheral nerves it is not infrequent for disturbances to occur in connection with intellection. Confusion of thought and impairment of memory are frequently present, occasionally in a peculiar form characterized by fabrication or "pseudoreminiscence," the patient relating imaginary recent experiences. This mental condition, combined with multiple neuritis, is sometimes called "Korssakoff's disease."

The cranial nerves also share in the malady. Indeed, in some instances they suffer most. Nystagmus, or squint, may be present, but the optic nerve is not often affected, although occasionally it may suffer very slight atrophy. The paralysis of the cranial nerves may be symmetrical. Thus, Oppenheim has reported cases of double facial palsy due to this cause.

Tachycardia and interference with the function of the diaphragm may be manifested from the infection involving the pneumogastric and phrenic nerves. Mannaberg asserts that the multiple neuritis may be confined entirely to the cranial nerves.

When the multiple neuritis is due to *lead*, it is a noteworthy fact that the inflammatory process is not, as a rule, very widely distributed, and that the sensory nerve fibres usually escape. In association with the symptoms of neuritis, already described, there may be a history of lead colic, which will aid in determining the cause of the paralysis. The presence of a blue line on the gums is also pathognomonic. Anæmia is often marked. Aside from the fact that sensation is usually not involved, the multiple neuritis caused by lead is noteworthy, in that it chiefly, and it may be exclusively, affects the extensor muscles of the hand and fingers. Indeed, the paralysis may be so localized in mild cases that only the extensor communis digitorum may be involved, so that the ring and little finger cannot be extended. When the paralysis is very marked, double drop-wrist is present. Another peculiarity of lead palsy is that the supinators, especially the supinator longus, and the triceps, escape. The deltoid, however, may be partially paralyzed, and the abductor pollicis longus and the interossei may be palsied. Occasionally, however, the supinators are affected, as is also the biceps. Muscular atrophy is nearly always marked in lead paralysis, and the reaction of degeneration usually speedily develops. Muscular tremor may also be present. The noteworthy fact that sensation is not disturbed in most cases may be well reiterated. Drop-foot is rarely seen in cases of lead paralysis. Paralysis of the cranial nerves due to lead is exceedingly uncommon. (See Lead Poisoning.)

When the paralysis is due to *arsenic*, it is not infrequently associated with gastrointestinal disturbances, and, unlike that due to lead, it is usually associated with marked disturbances of sensation in the affected parts. Wasting of the muscles supplied by the affected nerves usually develops quite early. Not only are the extensors affected, as they are in lead poisoning, but the flexors are also involved. Another point of difference between arsenical paralysis and that due to lead lies in the fact that the lower extremities are quite as frequently affected as the upper extremities, so that quadriplegia, that is, a paralysis of all four extremities, is present. Reactions of degeneration speedily develop. The pulse is apt to be rapid. Disturbances of the psychic functions are said to occur, which is rare in lead poisoning, unless encephalopathia saturnina is present. Symptoms of ataxia are usually marked. The reflexes are lost, and these two factors may make the case more closely resemble true locomotor ataxia than any other form of multiple neuritis. Nutritional changes in the skin are quite frequent in arsenical neuritis. In some instances herpetic eruptions develop. In others the skin becomes glossy, and there may be falling out of the hair. It is exceedingly rare for the cranial nerves to be involved.

Of all the forms of multiple neuritis due to toxic substances having their origin in the body, that due to the *poison of diphtheria* is most frequently met with. As diphtheria is essentially a disease of childhood, it is evident that diphtheritic multiple neuritis must be more commonly met with in young persons. The peculiarity of this form of neuritis is that it most frequently affects the muscles of the soft palate, changing the character of the speech and rendering swallowing difficult. This paralysis is both motor and sensory, and is often accompanied by wasting. Sometimes the external ocular muscles are paralyzed. In other instances the internal ocular muscles suffer chiefly, and accommodation may be paralyzed as the result of oculomotor involvement. The pupillary reflex is, however, usually preserved. In other instances the paralysis produced by diphtheria is almost universal. I have seen more than one instance in which the child was not only paralyzed in all its extremities, but was unable to exercise any control over the movements of its head, and could only swallow when put in such a position that the liquids could readily pass down the gullet. Such cases are usually characterized not only by loss of motor power, but by loss of sensation as well. The bladder and rectum usually escape the general paralysis, but they may be involved. It is a noteworthy fact that diphtheritic paralysis is not a concomitant symptom, but a sequel to an attack of diphtheria, and the full severity of the symptoms may not be present for several weeks after the diphtheria has ceased. In some instances the diaphragm is paralyzed, and if the nerve supply of the heart becomes affected sudden death may occur. (See Diphtheria.)

Diagnosis.—The presence of numbness and tingling followed by more or less impairment of motion and sensation in certain nerve trunks, with complete or partial escape of other nerve trunks, of course, points to multiple neuritis as the cause of the malady, particularly if the history of the patient reveals the fact that he or she has been exposed to one of the provoking causes already named. In some instances more than one of these causes has been effective, and, therefore, the precise factor in determining the neuritis cannot be relied upon. Thus, I have known more than one instance in which the administration of very large quantities of alcohol as a stimulant during typhoid fever has produced a multiple neuritis. which was attributed to a typhoid toxin, when in reality the alcohol was the active agent. In those cases in which the paralysis comes on very rapidly and is severe, the differentiation must be made between this condition and Landry's paralysis (which see), and this is the more important because Eichhorst has described a *neuritis acutissima progressiva.* A so-called apoplectiform type has been described by other observers. The presence of the Argyll-Robertson pupil, optic nerve atrophy, and the history of exposure to a poison may be the only means by which we can differentiate between true locomotor ataxia and multiple neuritis.

Prognosis.—The prognosis is favorable in nearly every case, unless the patient has been exposed to the evil influences of lead or arsenic or alcohol for so long a time that the nerves cannot undergo regenerative change. In cases of profound alcoholic intoxication sudden death may take place when the pneumogastric nerve becomes involved. In nearly all instances recovery is exceedingly slow The first symptom of improvement is a diminution in the pain and a decrease in tenderness of the nerves on palpation. In other instances the power of motion returns before the sensory functions are restored to their normal condition, and inability to get about may be caused by the intense hypersensitiveness of the feet. Even if the patient does not recover for eighteen months or two years, the condition is by no means hopeless. Care should be taken, however, that complete recovery should not be promised in cases which have been exposed to the poisons for very long periods of time. Not infrequently great disappointment is caused by periods in which no improvement takes place, or, indeed, in which a relapse seems to be threatened.

The prognosis in multiple neuritis due to lead is good so far as the preservation of life is concerned. It is bad in direct proportion to the duration of the condition and of the exposure to the poison. The same facts hold true in regard to the peripheral neuritis due to arsenic.

Occasionally secondary contractures occur as the result of the contraction of non-paralyzed muscles, whereby deformities are produced.

After diphtheria, even in those cases in which the paralysis is most severe, the prognosis is not necessarily very grave. The immediate danger is that some nervous mechanism connected with a vital function may be involved. If this does not occur, partial or complete recovery of motion or sensation nearly always takes place, although twelve months may pass before recovery occurs. Usually, however, two or three months is sufficient.

Treatment.—The treatment of multiple neuritis gives better results than that devoted to the relief of any other form of paralysis. If the cause of the malady is one of the metallic poisons already named, the patient must be removed from further exposure to the poison. Thus, workers in lead and arsenic must cease following such occupations. For the purpose of aiding in the elimination of any of the poisons which may remain in the body, moderate doses, 20 to 30 grains, of iodide of potassium may be given twice or thrice a day. If the patient uses alcohol to excess, this agent must, of course, be withdrawn. While the nerves are hypersensitive to pressure and while pain is present, strychnine and faradic electricity should not be applied to them, since they tend to increase irritation; but when there is anæsthesia and loss of power, full doses of strychnine and phosphorus are often useful, and the rapidly interrupted faradic current may be used to stimulate the affected nerve fibres. Muscles which are suffering from loss of power may be exercised by the use of the slowly interrupted faradic current. Massage may also be employed, but it is of vital importance that no form of exercise shall be used to the point of exhaustion of the affected parts. In other words, only healthy exercise designed to improve the nutrition of the parts affected should be resorted to.

If it is thought that the neuritis is due to toxic materials arising inside the body, these should be removed, if possible. The administration of laxatives or purges is usually needful. If anæmia is present, particularly if it is associated with septic conditions, such as are met with in sepsis and puerperal fever, iron and arsenic are useful. If the patient is rheumatic or of gouty tendency, hot baths, or a visit to any of the well-known hot springs may be resorted to, and the various salicylates or iodides should be administered in sufficiently full doses to produce mild physiological effects. For the purpose of aiding in the elimination of toxic materials, pure water should be drunk freely to flush the kidneys, and Turkish baths may be taken to produce sweating. Pain is to be relieved by the use of such remedies as phenacetin or acetanilid, and by hot applications to those areas which suffer most. Sometimes the application of splints to provide perfect rest for the painful part is useful.

In those forms of multiple neuritis which depend upon infection, such as diphtheria, smallpox, or typhoid fever, the heart should be carefully examined, and if any evidences of tachycardia, bradycardia, or arhythmia are present, the patient should be warned against sitting up in bed, and should be protected from all causes which may throw an increased strain upon the circulation. This is particularly important in diphtheritic multiple neuritis. Contractures should be prevented by massage and Swedish movements and remedied, if they occur, by tenotomy.

DISEASES OF THE CRANIAL NERVES.

The Olfactory Nerve.—Disease of the olfactory nerve, of course, interferes with the special sense of smell, and if this sense is entirely lost the condition is called anosmia. Partial or complete loss of this sense results from lesions of the peripheral ending of the nerve in the nasal mucous membrane and from pathological states of the tissues beneath it, such as morbid growths or disease of the ethmoid bones. Similar loss of function results from meningitis, from injury of the bones forming the base of the skull, or morbid growths affecting these bones. Tumors of the brain may destroy the olfactory nerves or the olfactory bulbs. When complete loss of the sense of smell occurs and no local lesion in the nasal bones or mucous membranes is present, it is usually an evidence of a tumor or abscess in the anterior cranial fossa.

The Optic Nerve.—The optic tract of either side arises by two roots from structures in the midbrain called the primary optic centres. These structures are the external geniculate body, the posterior part (pulvinar) of the optic thalamus, and the anterior quadrigeminal body.

It is important to remember that the fibres from the optic tract undergo partial decussation in the chiasm. The outer fibres do not decussate and they connect the outer half of the retina with the primary optic centres of the same side. The inner fibres, on the other hand, all cross to the opposite side, and they connect the inner half of the retina with the nuclei on the opposite side. It is evident, therefore, that the right optic tract contains fibres which carry impulses from the right halves of both retinæ to the right side of the brain, and that the left optic tract contains fibres which convey impulses from the left halves of both retinæ to the left side of the brain. It is essential to remember these facts in order to understand the condition known as hemianopsia, which will be described shortly.

Optic neuritis, sometimes called papillitis, is an inflammatory condition which is manifest in the intraocular end of the nerve, and it may be due to several causes. In the great majority of cases it is due to brain tumor. Choked disk is an œdematous state. The degree of neuritis has no direct relationship to the size of the tumor, nor to the area of the brain which it affects, although a tumor of the corpora quadrigemina seems to cause the condition more commonly than do growths elsewhere. Tumor of the parieto-occipital region and of the cerebellum also produces papillitis in a large proportion of cases in which these growths occur, while a tumor of the frontal lobes of the cerebrum very rarely causes it. The condition is not materially affected as to frequency or severity by the character of the growth. Meningitis in any of its forms may cause papillitis, but tuberculous meningitis does so more commonly than any other form. Rarer causes are cerebral softening, inflammation, and atrophy, or any cause, such as aneurysm or hydrocephalus, which produces an increase in intracranial pressure. Very rarely disseminated sclerosis, general paresis, or myelitis may cause papillitis, as may the various acute infectious diseases, or the excessive use of alcohol, or lead poisoning.

Symptoms.—There are often no symptoms whatever which point to optic neuritis, at least in so far as the patient complains of impairment of vision. The diagnosis rests solely upon the use of the ophthalmoscope and upon a study of the fields of vision. The ophthalmoscope reveals an *indefinite outline of the head of the nerve*, with redness, followed by swelling of the papilla, which becomes grayish in hue. Finally, the *disk protrudes*, its *outlines* become *lost* and *whitish patches* may be seen upon its surface. The *retinal arteries* are *contracted* and the *veins congested* and *tortuous*. At the point of exit and entrance of the vessels this part may seem devoid of vessels, because they are hidden in the infiltrated mass. *Small*, narrow, *flame-like hemorrhages* may be seen along the vessel walls. The *field of vision* is *concentrically contracted*, and the *perception of red and green* is lost before the other color senses are destroyed. *Hemianopsia* is present if the lesion is so situated as to cause this symptom.

Another *form of optic neuritis*, called retrobulbar neuritis, exists in which the inflammatory process develops in the optic nerve in the orbit. In the acute form the symptoms consist in dimness of vision which always occurs in the centre of the field, and which may end in complete blindness in from one to eight days. With the ophthalmoscope, when the disease is well developed, the edges of the disk are seen to be indistinct, its surface hyperæmic, and its main bloodvessels shrunken. The cause of the acute form is usually some one of the acute infections, such as influenza, scarlet fever, or one of the diathetic diseases, such as rheumatism, gout, and sometimes syphilis.

The treatment of retrobulbar neuritis consists in the production of profuse sweating by pilocarpine, the use of large doses of the salicylates if gout or rheumatism is present, or the employment of mercury and the iodides if syphilis is suspected. Counterirritation on the temple is also advisable.

The *chronic form of retrobulbar neuritis* is usually a toxic condition produced, in the majority of instances, by tobacco, alcohol, arsenic, lead, or poisons made by infectious diseases. Its symptoms consist in diminution of vision and in color scotomata. The prognosis when the cause is tobacco and alcohol is good, if the patient will give up these drugs and if he does so in the early stages of the disease; otherwise the prognosis is bad.

The treatment consists in the elimination of the causes as far as possible, in the use of massive doses of strychnine, and the employment of the iodides and free sweating.

Treatment.—This depends upon the cause. If it is due to brain tumor or abscess, operative treatment is required, unless a gummatous growth is present, when mercury and the iodides are needful. Trephining of the skull to relieve pressure may be resorted to as a palliative measure in cases where a growth cannot be removed.

OPTIC ATROPHY.—Atrophy of the optic nerve, as its name implies, is a condition in which a degenerative process affects its fibres. It is divided into five forms: the primary, secondary, consecutive, retinitic, and choroiditic atrophy. The last two forms are really of the consecutive class.

Etiology.—Primary atrophy of the optic nerve has been thought to be due to impaired nutrition, sexual excesses, and to such diseases as chronic malarial infection, diabetes, syphilis, and to the overaction of certain drugs. The most important causes of primary optic atrophy are diseases of the spinal cord, notably locomotor ataxia. It is also seen in cases of general paresis and disseminated sclerosis. In many instances the optic atrophy may be one of the early symptoms of ataxia. It has also been met with in cases of lateral sclerosis, chronic myelitis, and bulbar palsy.

Secondary atrophy arises from causes which produce pressure upon the optic tract and the optic fibres, as, for example, the growth of a tumor or an aneurysm, or meningitis. So, too, injuries to the head sometimes produce atrophy. Consecutive atrophy follows the various forms of optic neuritis.

Pathology.—The axones lose their medullary sheaths and are converted into fine fibrils, between which are interspersed numerous fatty granules. When the condition is far advanced, the nerve elements entirely disappear and there is a marked increase in connective-tissue formation.

Symptoms.—The subjective symptom complained of by the patient is *diminution in the acuity of vision.* The other symptoms are developed by the use of the ophthalmoscope. When this instrument is used it is found that the optic disk is gray or greenish-gray, or actually white in color, although there may be patches of red throughout it. The *centre of the disk* is *depressed* in direct proportion to the degree of atrophy which has taken place. The *margin of the disk* is *distinct*, and in some cases, when the condition of the optic nerve is due to disease of the spinal cord, there is broadening of the normal scleral ring. The *bloodvessels* are *narrowed*, but in some cases only the arteries seem to be affected, the veins escaping. An examination of the central *vision* shows that it is *markedly impaired*, or *absolute blindness* may be present. The *field of vision* is *greatly narrowed* and there may be a *central scotoma* or *hemianopsia*. The color fields are markedly diminished, the green being most affected; after it the red, and then the blue and yellow.

Sometimes the field for red is first affected. The pupil usually manifests some degree of paralytic dilatation, and when the nerve is completely atrophied the pupil is dilated and the iris motionless. In secondary atrophy the disk is apt to be whiter than in the primary forms, when it is usually gray. In that form of optic nerve atrophy called retinitic and choroiditic atrophy, the disk is often slightly yellowish in hue, but its borders are not distinct.

Diagnosis.—The mere discovery that the optic disk is grayer than normal and that its margins are sharply defined, does not justify the diagnosis of optic atrophy. If, however, any of the diseases so far named are also present, such a diagnosis is usually correct.

Prognosis.—The prognosis as to complete recovery is bad. On the other hand, it must be remembered that the atrophic process is often a slow one which may last for years. Indeed, the prognosis in secondary cases depends largely upon the rapidity with which the underlying disease is advancing.

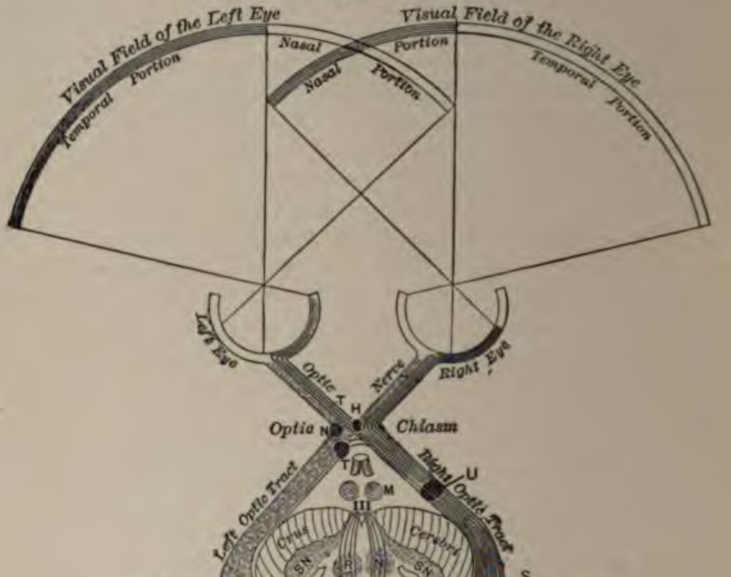
Treatment.—The treatment consists in the administration of full doses of mercury and the iodides if there is any suspicion that a recent or ancient syphilitic infection has been present. Strychnine in large doses combined with nitroglycerin is also useful.

Of the *functional disorders* of the optic nerve the most important are blindness due to uræmia, that due to diabetes, malaria, profound anæmia, and the abuse of drugs. When uræmia is the cause the presence of the symptoms of that condition, in association with dimness of vision or blindness, make the diagnosis clear. The so-called albuminuric retinitis may be present, but the ophthalmoscope may, however, reveal no morbid changes. If the patient survives, vision usually returns.

When the dimness of vision is due to diabetes, the prognosis is unfavorable because the disease is incurable. That form which is due to malarial infection has associated with it other symptoms of this disease. Recovery usually takes place if proper treatment is administered. So, too, in cases of dimness of vision, due to hemorrhage and profound anæmia, the prognosis is good, unless the anæmia is one of the so-called essential anæmias which always go from bad to worse. The treatment in such a case consists, of course, in the use of drugs designed to combat anæmia.

HEMIANOPSIA.—Hemianopsia, or blindness of one-half of the visual field, occurs in three forms: that known as bitemporal hemianopsia, binasal hemianopsia, and homonymous hemianopsia, each variety being named not from that part of the retina which is blind, but from the visual field which is affected. In the first there is loss of vision in both temporal fields, in the second in the nasal half of each eye, and in the third form the same side of each eye is lacking in function—that is, for example, the outer half of the left eye and the inner half of the right eye. When the left half of each retina is inactive, the condition is called right homonymous bilateral hemianopsia, and when the right half is functionless it is designated left homonymous bilateral hemianopsia. Homonymous hemianopsia is the most common. Binasal hemianopsia is very rare. Bitemporal hemianopsia is produced by a lesion, such as a tumor or an aneurysm, which presses upon the middle of the chiasm. Homonymous lateral hemianopsia is produced by a lesion of one optic path at any point back of the chiasm, either in the neighborhood of the calcarine fissure (occipital lobe), in the optic radiations, including the point where they pass just back of the internal capsule, in the primary optic

FIG. 128



The visual tract. The result of a lesion anywhere between the chiasm and the cuneus is to produce homonymous hemianopsia. H. Lesion at chiasm causing bilateral temporal hemianopsia. N. Lesion at chiasm causing unilateral nasal hemianopsia. T. Lesion at chiasm causing unilateral temporal hemianopsia. SN. Substantia nigra of crus. L. Lemniscus in crus. RN. Red nucleus. III. Third nerves. P, Q, R, S, U. Lesions in the occipital lobe and in front of it, producing left homonymous lateral hemianopsia.

centres, or in the optic tract. (See Fig. 128.) It is important to remember that the lesion in cases of hemianopsia is on the opposite side to that of the dark field.[1] (See Fig. 129.) De Schweinitz has condensed the following rules as to the significance of various forms of hemianopsia from a series prepared by Dr. Seguin:

(*a*) The lesion in hemianopsia is on the opposite side of the dark fields.

(*b*) If the preserved fields are accompanied by concentric contraction, the smaller half-field will be in the eye opposite to the lesion; contraction of the preserved half-field is most common with lesions of the cortex, but also may occur in lesions of the tractus.

FIG. 129

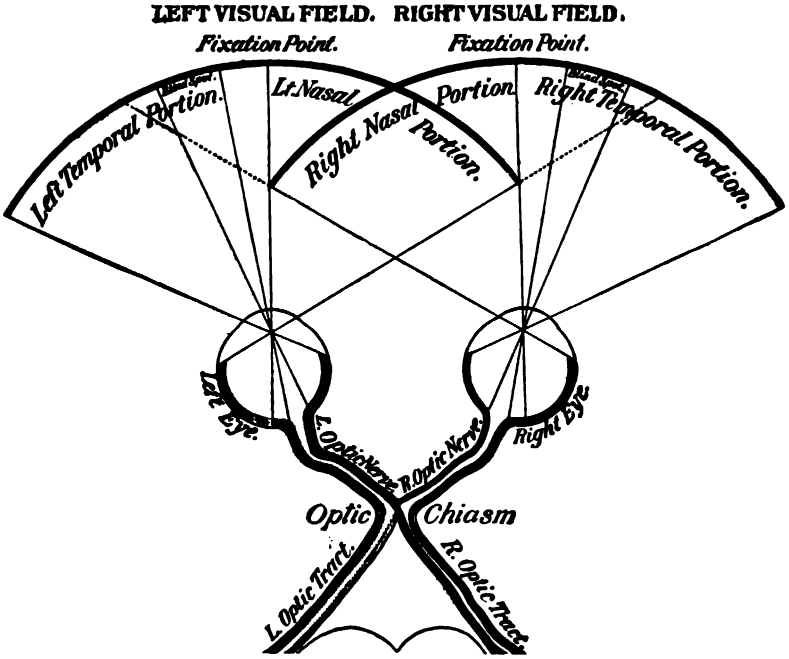


Diagram illustrating why it is that the lesion is on the opposite side to the dark field. (Oliver.)

(*c*) If the hemianopsia is relative, the lesion is probably in the cortex; but cortical lesions are not excluded by absolute hemianopsia.

(*d*) A lesion confined to the cuneus, or to it and the gray matter immediately surrounding it, on the mesial surface of the occipital lobe, produces homonymous lateral hemianopsia without motor or sensory symptoms, at least without these as a direct consequence of the lesion, although they may appear as indirect or, as they are sometimes called, distant symptoms. Slight motor symptoms such as deviation of one eye inward may, however, be added to the visual symptoms of a lesion in the occipital lobe (Mills).

(*e*) A lesion producing typical hemiplegia, aphasia, if the right side is paralyzed, little or no anæsthesia, and lateral hemianopsia, is probably due to disease in the area supplied by the middle cerebral artery.

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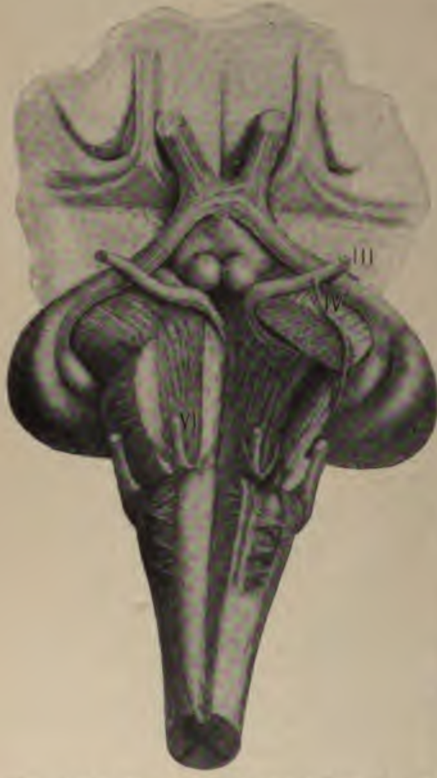
[1] For a more complete study of the significance of this difficult subject see the author's Practical Diagnosis, fifth edition.

(*f*) A lesion causing hemiplegia, hemianæsthesia, and lateral hemianopsia is probably situated in the posterior portion of the internal capsule.

(*g*) A lesion causing hemianæsthesia, ataxic movements of one-half of the body, no distinct hemiplegia, and lateral hemianopsia, could be situated in the posterior lateral part of the optic thalamus.

(*h*) A lesion causing the symptoms of disease of the base of the brain, associated at the same time with changes in the pupil, changes in the nerve head, and lateral hemianopsia, could be situated in one optic tract or in the primary optic centres on one side.

FIG. 130



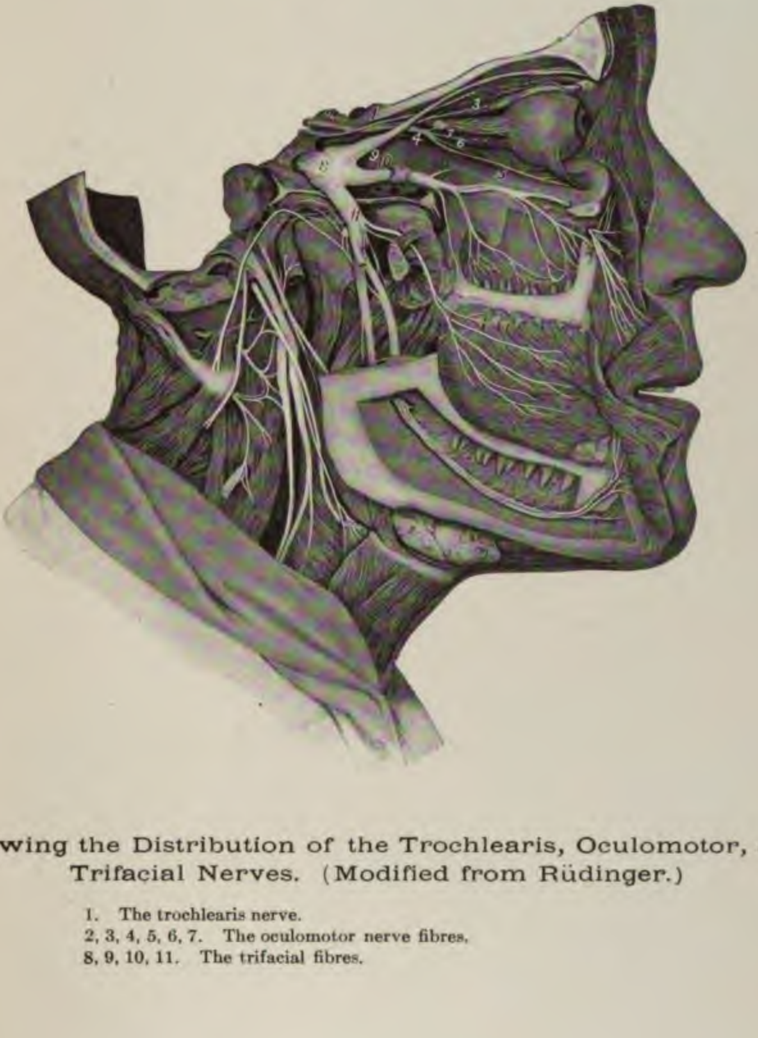
Showing the nearness of origin of the oculomotor (3), pathetic (4), and abducens (6). The roots of these nerves are shown by an incision which has divided the pons. III. The third nerve, arising from several roots. IV. The fourth nerve. VI. The sixth nerve, arising from three roots. (Modified from Arnold.)

(*i*) Incomplete hemianopsia, assuming usually a quadrant-shaped defect, may be present on account of a lesion confined to the lower half of the cuneus. It may also occur with less definite limitations in lesions of the subcortical substance of the occipital lobe, and then may be associated with other symptoms, as hemiplegia and hemianæsthesia. Finally, it may occur from a lesion of the tract, but then will be accompanied by other symptoms indicating basal disease, or from a lesion of the external geniculate body.

(*j*) A hemianopsia in which there is preservation of the light sense, but loss of either the color sense or the form sense, indicates that the lesion is in the visual centre of the cortex.

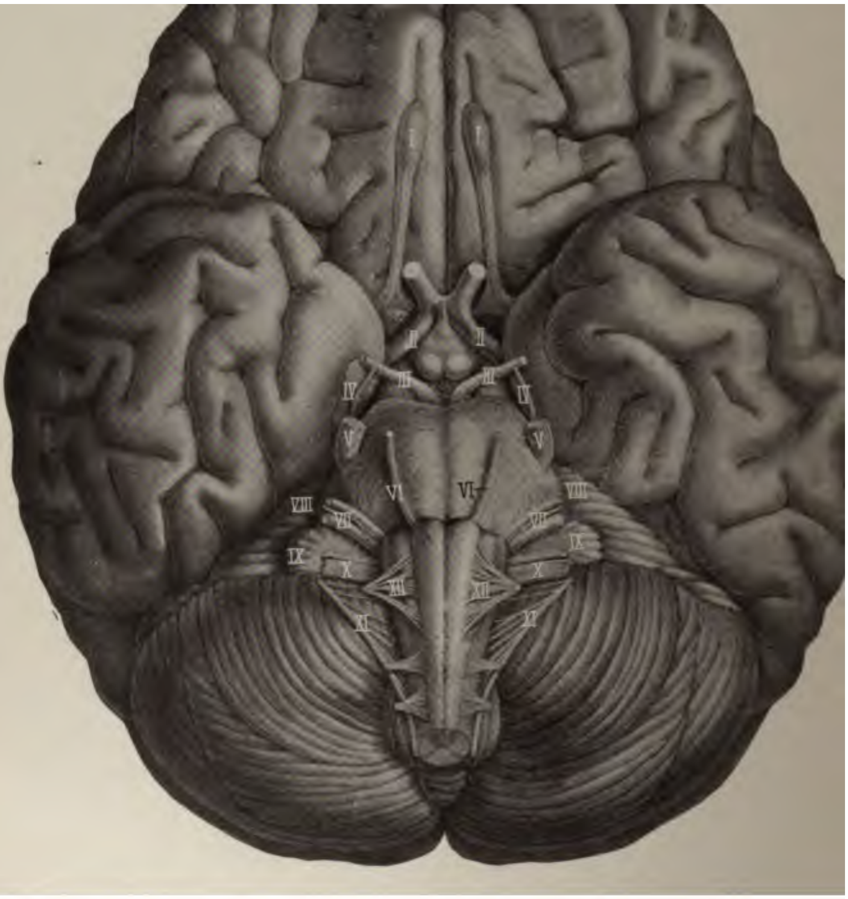
The Third or Oculomotor Nerve.—The third nerve has its origin from groups of cells in the floor of the aqueduct of Sylvius. It then passes through the tegmentum of the crus cerebri, and makes its exit in a bundle on the inner side of the crus. (Fig. 130; see also Plate IX.) It then passes from the crus to the sphenoidal fissure and so into the orbit, where its fibres divide and go to supply the ciliary muscle, the sphincter of the iris, the superior rectus, internal rectus, inferior rectus and inferior oblique muscle. (See Plate VIII.) It also sends fibres to the levator palpebræ muscle. As it is a motor nerve, paralysis follows its injury. The causes of disturbance in its function are

PLATE VIII.



Showing the Distribution of the Trochlearis, Oculomotor, and Trifacial Nerves. (Modified from Rüdinger.)

1. The trochlearis nerve.
2, 3, 4, 5, 6, 7. The oculomotor nerve fibres.
8, 9, 10, 11. The trifacial fibres.


I
I
II
II
IV
III
III
IV
V
V
VI
VI
VIII
VIII
VII
VII
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X
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XI

numerous. They may exist at the base of the brain, where the nerve leaves the crus, in the sphenoidal fissure, in the orbit, and even in its peripheral filaments in the eye itself, although change in its functional activity in the latter area is usually due to the effect of drugs. Of the causes which produce disturbance of its function at its origin in the crus, we find tuberculous meningitis or that due to some acute infectious disease, abscess of the brain, and hemorrhage. For this reason meningitis of either form in infancy very frequently involves this nerve, and so produces symptoms which call the attention of the physician to the existence of the disease at the base of the brain. In adults, aside from tuberculous meningitis, there may be syphilitic exudation, or the nerve itself may be inflamed, owing to the presence of this same disease. Tumors or abscess at the base of the brain may press upon it. When the nerve is injured in its passage through the sphenoidal fissure, the cause is usually some traumatism which results in fracture of the bone, or very rarely a severe blow which damages the nerve by pressure against the bone. In a case which came to the writer's attention, a severe blow with the hilt of a sword upon the forehead caused paralysis of this nerve, probably in this manner.

In the orbit a tumor may press upon the nerve fibres. Occasionally the nerve loses power through the action of the poison produced by diphtheria or typhoid fever.

Symptoms.—The dominant symptoms of paralysis of the oculomotor nerve are *ptosis, mydriasis,* and consequent *loss of pupillary reaction* to *light* and *accommodation.* As it supplies the internal rectus, external squint may be present. The paralysis of the ocular muscles also results in diplopia. If the patient is directed to look upward, downward, or inward he is unable to do so. The *inability of the pupil to contract* when light is thrown into the eye may be due to a lesion of the nerve before it enters the orbit, or, as already stated, to the action of a drug upon its peripheral filaments. It will be remembered that pupillary contraction, when produced by the entrance of light into the eye, is due to a reflex impulse which passes along the optic nerve to the neighborhood of the corpora quadrigemina, thence to the third nucleus and along the fibres of the third nerve to the ciliary ganglion, from which, by way of the ciliary nerves, it goes to the iris and causes contraction of its circular muscular fibres. A lesion in any portion of this reflex arc interferes with pupillary reaction. In addition to those injuries of the oculomotor nerve already mentioned which cause paralysis, a loss of pupillary reaction may occur in locomotor ataxia, in multiple sclerosis, in general paresis, in bulbar palsy, and in myelitis when that disease involves the fibres of the arc. When these diseases are responsible for the loss of pupillary reaction, the lesion is supposed to exist, in the majority of instances, in fibres which connect the optic tracts in front of the corpora quadrigemina with the oculomotor nuclei. When drugs produce paralytic mydriasis, their action is usually exercised upon the peripheral ends of the nerve.

Diagnosis.—The diagnosis of paralysis of the oculomotor nerve is readily made if the symptoms just described are kept in mind.

Prognosis.—The prognosis depends upon the underlying cause of the paralysis. In diphtheria and typhoid fever recovery usually takes place, and unless the damage produced by an injury is very great, the outlook is favorable. On the other hand, if the cause is tuberculous meningitis, tumor, or abscess, or any one of the progressive nerve diseases just named, recovery is, of course impossible.

The Fourth or Trochlearis Nerve. This nerve supplies the superior oblique muscle of the eye (1, Plate VIII.). Interference with the action of this nerve is not uncommon and is rarely recognized by the general practitioner. The *symptoms* are not developed until the eye is tested by means of placing a colored glass over one eye, when it will be found that the object which is placed before the patient stands in its normal position as seen by the normal eye, but is displayed outward and obliquely when seen by the eye supplied by the impaired pathetic nerve. As the nerve arises from an area almost identical with that of the third nerve, the centric causes of trochlearis paralysis are practically identical with the causes of oculomotor paralysis. It is important to remember that, should paralysis of the fourth nerve be present without involvement of the third or sixth nerve, it probably indicates a growth in the cerebellum or an inflammatory exudate upon the under surface of its middle lobe

The Fifth or Trifacial Nerve.—The trifacial nerve contains motor and sensory fibres, the sensory fibres being by far the more numerous. The motor fibres have their origin in the pons, a little above its middle, receiving also the root descending from the midbrain; they pass out in a bundle separate from the sensory fibres until, outside the cranial cavity, they take part in forming the inferior maxillary division of the nerve, through which they supply the muscles of mastication.

The sensory fibres of the fifth nerve arise from a nucleus at about the middle of the pons, and, in addition, by the spinal root, from a chain of cells descending through the medulla as far as the first cervical segment of the cord; they emerge from the pons in a heavy trunk, which passes to the Gasserian ganglion (8, Plate VIII.) and then, beyond the ganglion, divides into three branches: ophthalmic (9), superior (10), and inferior maxillary (11). In addition to providing sensation to the greater portion of the face, it also supplies the anterior two-thirds of the tongue.

Symptoms.—When the fifth or trifacial nerve is paralyzed in its motor fibres, the patient is *unable* to *contract his masseter muscles* and there is *dropping of the lower jaw.* Unless the paralysis is bilateral, however, it may not be easily discovered, since the muscles on the unaffected side may hold the jaw in position. And, moreover, if the lesion be in the brain the function of mastication is maintained from the other side by bilateral innervation. In some instances of paralysis of the fifth nerve, deafness arises as the result of interference with the function of the tensor tympani muscle, for a small branch from the motor fibres of the fifth nerve passes through the otic ganglion and supplies this muscle. When this muscle is paralyzed, the tympanic membrane is relaxed and this interferes with its function. Motor paralysis of the fifth nerve is rarely met with. Certain poisons like gelsemium may cause dropping of the jaw by paralyzing the muscles of both sides. When the sensory portion of the nerve is affected there is anæsthesia of the skin of the face in the areas supplied by the particular branches affected.

If the area be that of the forehead, the upper eyelid, the conjunctiva, and the nostril, the ophthalmic branch of the fifth nerve is at fault, and the lesion is probably at the sphenoidal fissure or within the orbit, reflex winking of the eye no longer takes place because the conjunctiva is anæsthetic, and for the same reason a flow of tears does not occur upon irritating the conjunctiva, because the lachrymal reflex is abolished.

If the skin of the upper part of the face is anæsthetic, the superior maxillary branch is involved; and if the skin of the temporal region and that of the jaw and the under lip are anæsthetic, the inferior maxillary branch is diseased. When both of these branches are paralyzed there is probably a tumor of the superior maxillary bone; and if the entire area of the three branches is anæsthetic, the Gasserian ganglion may be the part affected, and this will be accompanied by trophic changes in the anæsthetic parts. The most common cause of anæsthesia of the trifacial is, however, neuritis.

Romberg makes the following differential statement:

(*a*) The more the anæsthesia is confined to single filaments of the trigeminus, the more peripheral the seat of the cause will be found to be.

(*b*) If the loss of sensation affects a portion of the facial surface, together with the corresponding faucial membrane, the disease may be assumed to involve the sensory fibres of the fifth pair before they separate to be distributed to their respective destinations; in other words, a main division must be affected before or after its passage through the cranium.

(*c*) When the entire sensory tract of the fifth nerve has lost its power, and there are at the same time derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerve in its immediate vicinity, is the seat of the disease.

(*d*) If the anæsthesia of the fifth nerve is complicated with disturbed functions of adjacent cerebral nerves, it may be assumed that the cause is seated at the base of the brain.

When the fifth nerve is paralyzed the *mucous membrane of the nose and mouth* are also *anæsthetic* and usually *dry*. The *sense of taste is lost* and trophic lesions may develop, although it is questionable as to whether these depend upon affection of the sensory fibres. These lesions consist in *ulceration of the cornea*, *loosening of the teeth*, *atrophy of the gums*, and the *development of herpes zoster*. As the *sensation* in the anterior two-thirds of the *tongue* is impaired, this organ is often damaged by the teeth. The dryness of the mucous membrane of the nose also interferes with the *sense of smell*, and irritating substances may be inhaled through the nostrils without pain, because of the lack of sensation in the nasal mucous membrane. Paralysis of this nerve is, however, very rarely met with.

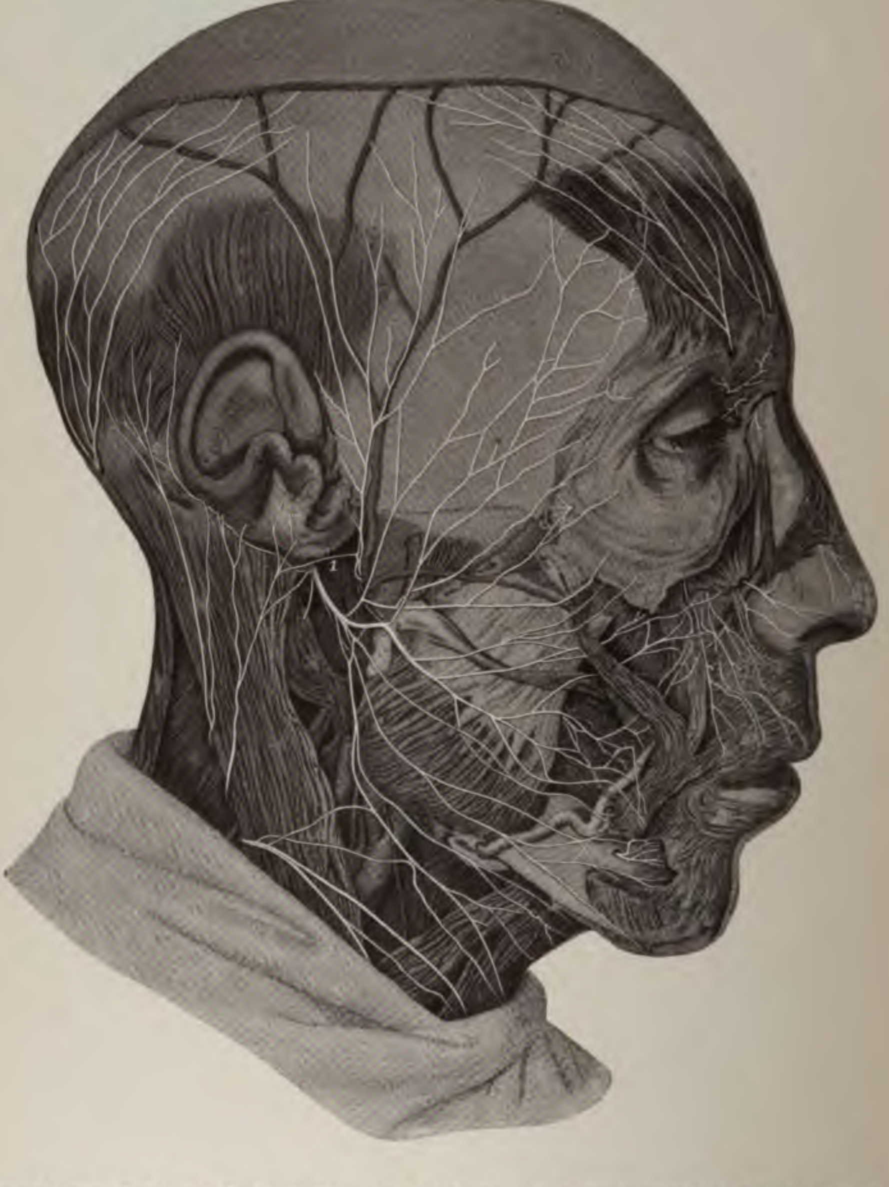
In the great majority of instances in which a physician is called on to treat a lesion of the trifacial nerve, the patient complains of *severe neuralgic pain*, which in most cases arises from the Gasserian ganglion (8, Plate VIII.). When trophic changes are very well marked, they result in *hemiatrophy of the face*. In those cases in which the motor fibres of the fifth nerve are irritated, there may be lockjaw as in true tetanus, and so-called masseter spasm may have a reflex origin because of the presence of dental irritation.

Paralysis of the Sixth Abducens Nerve.—The sixth nerve has its origin from cells in the floor of the fourth ventricle, passes through the pons, and makes its exit in the groove between the pons and the medulla (Plate IX.), whence it passes through the sphenoidal fissure. It is subject to the same lesions at the base of the brain as is the oculomotor nerve, such as tuberculous meningitis and syphilitic exudation, tumor and fracture of the base of the skull. Injury may occur to it in the sphenoidal fissure. The sixth nerve supplies the external rectus and its paralysis thus causes internal squint, the patient being unable to rotate the eye outward.

The exact lesion which produces paralysis of the sixth nerve can only be determined by a study of the associated symptoms. In those cases in which there is facial palsy on the same side as the squint, and paralysis of the arm and leg upon the opposite side, in other words, "crossed hemiplegia," the lesion is in the pons or at the base of the brain in such a position that it produces pressure on the pons on one side and above its lower third.

Disturbances of Motility in the Ocular Muscles Depending on the Third, Fourth, and Sixth Nerves.—The movements of the eyeballs depend, of course, upon the associated action of different muscles supplied by different nerves. When the axes of the eyeballs converge, they do so by the action of the internal recti muscles supplied by the third nerve, and when they diverge they move in these directions by the external recti supplied by the sixth nerve. If, however, there is conjugate deviation, then a much more complicated nervous mechanism is brought into play, for if the axis of each eyeball is turned to the right, for example, this motion is made by contraction of the external rectus of the right eye and the internal rectus of the left eye, each being supplied by different nerves, the right sixth and the left third, and yet it is essential that they shall act in accord. This is accomplished by the presence of association fibres which, by joining together the nuclei of the nerves, enable them to act in unison. If by disease these association fibres are destroyed (in the posterior longitudinal bundle), conjugate deviation of the eyes becomes impossible. When the eyeballs are deviated by reflex action, the pathway of the nervous impulse is through the optic nerve by connecting fibres to the motor nuclei of those nerves governing the ocular movements, which not only join the nuclei of the different nerves of one side, but connect them with the nuclei of the opposite side as well. When they are moved by voluntary action, the impulse leaves the motor centres in the anterior part of the motor area of the cortex, and thence passes down through the anterior part of the knee of the internal capsule, thence through the crus cerebri, and finally crosses in the raphé, passing to the nuclei of the oculomotor nerves and of the fourth and sixth nerves. When the impulse for conjugate deviation arises in the motor cortex, it passes first to the nucleus of the opposite sixth nerve, and thence is sent along the association fibres through the posterior longitudinal bundle to the nucleus of the third nerve on the opposite side, just as it is in reflex deviation. When a nervous explosion takes place in the motor cortex, as in cases of epilepsy, it often happens that there is conjugate deviation of the eyes away from the side on which the lesion exists, and, conversely, if the ocular centres in

PLATE X.



Showing Exit of Facial Nerve (1) from Stylomastoid Foramen and its Distribution to the Muscles of the Face. (Rüdinger.)

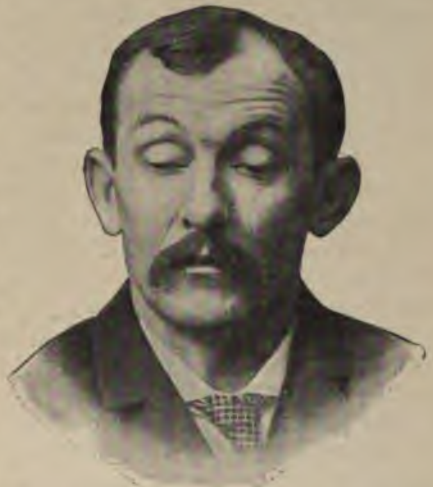
the cortex are destroyed, there is conjugate deviation of the eyes toward the side on which the lesion exists. This has given rise to the statement that in the coma of ordinary apoplexy the patient "looks toward his lesion" at least in those instances in which an apoplexy destroys these centres. (See Apoplexy.)

There are two states which give rise to an erroneous diagnosis in connection with these symptoms, namely, ' rheumatic palsy" of the ocular muscles, which disappears under the free use of the iodides and salicylates, and so-called "recurrent oculomotor paralysis," which is probably the result of congestion and œdema, and which is accompanied by sick stomach, diplopia and fever.

Ophthalmoplegia or Paralysis of the Internal and External Muscles of the Eyeball).—This condition depends not upon disorder of function of any single cranial nerve, but upon interference with the action of the third, fourth, and sixth nerves. As already stated, the third nerve supplies the ciliary muscle, the circular fibres of the iris, the superior rectus, internal rectus, inferior rectus, inferior oblique, and the levator palpebræ. The fourth nerve supplies the superior oblique, and the sixth the external rectus. When morbid changes take place in the nuclei of these nerves the normal co-ordinated movements of the eye are impaired or lost, that is to say, ophthalmoplegia is developed.

Ophthalmoplegia is of two forms: ophthalmoplegia externa, when the paralysis affects the external muscles of the eyeball and the levator palpebræ; and ophthalmoplegia interna, when only the pupillary and ciliary muscles are involved. *Ophthalmoplegia interna* is quite rare, although a modified form of it occurs in that state called the Argyll-Robertson pupil, a condition in which the pupil reacts to accommodation, but not to light. In this condition the lesion exists not in the nuclei of the nerves, for if it did there would be no reaction to accommodation, but in the association fibres, whereby the reflex pathway is destroyed. *Ophthalmoplegia externa*, on the other hand, is by no means uncommon. It is a condition depending upon a centric lesion, and occurs in an acute and chronic form (see below). Because of the fact that the lesion is centric it is usually bilateral, and if all the muscles are paralyzed it is said to be complete external ophthalmoplegia, while, on the other hand, if they are simply impaired in function, or if one nerve escapes while the others are involved, it is spoken of as partial (Fig. 131).

Fig. 131



Patient suffering from chronic ophthalmoplegia externa. The wrinkling of the forehead in the effort to open the eyes is noticeable. The external strabismus can be seen. (Starr.)

Etiology and Pathology.—Ophthalmoplegia is due to a large number of causes, such as tumors, areas of degeneration, or inflammatory exudations, where the nerves take their exit at the base of the brain. (See Plate IX.) The additional causes are small hemorrhagic extravasations, arteritis, thrombosis, or embolism of the small vessels which supply the nuclei of these nerves. In some cases the lesions resemble those of acute poliomyelitis, and belong to the affection called by Wernicke "polioencephalitis superior."

Symptoms.—The symptoms of ophthalmoplegia externa vary, of course, with the nerves which are affected and with the severity of the lesions. When the morbid process is severe, there is not only loss of power in the ocular muscles, but in other parts as well, so that the symptom-complex of bulbar paralysis may be present; or if the tracts to and from the higher areas of the brain are involved, such symptoms as hemianæsthesia, hemiplegia, or hemiataxia may be present. The pathological processes just described are varied not only as to cause and situation, but as to acuteness as well. The acute form is ushered in by a train of symptoms which necessarily arise when areas of the nervous system so important to life are affected. Thus, the patient suffers from vertigo, headache, vomiting, and even coma. Unconsciousness may last for several days and end in death, or, after a period of a week or ten days, consciousness gradually returns and the symptoms connected with the eyes alone remain. These consist in double ptosis and various palsies of the ocular muscles, or total ophthalmoplegia. The chronic form arises when the nervous lesions are gradual in onset, although it may result from the acute type just discussed. Here again the degree of the paralysis depends upon the severity of the lesions. In one case a total palsy may be present, in another a partial palsy, and in still a third the palsy may be progressive, one muscle after another failing. Sometimes one muscle improves as another fails. Ptosis and other forms of ocular palsy may be a part of the transient and recurrent paralysis in myasthenia gravis. The prognosis depends upon the cause. If syphilis is a factor the outlook is favorable as compared to that type which is due to disseminated sclerosis or bulbar palsy. In no case is the outlook anything but grave as to recovery, although about one-half of the mild cases recover.

The treatment also varies with the cause, and yet it may be said that, be the cause what it may, the only drugs which offer any promise of relief in the chronic form are mercury and the iodides. Hot baths may be useful.

In the acute form freedom from any cause of excitement, the application of cold to the head, and the use of aconite to quiet the circulation, if it is excited, may be of some value.

The Seventh or Facial Nerve.—The nucleus of the facial nerve is found in the lower part of the pons. From this nucleus its fibres pass upward and backward to the floor of the fourth ventricle, where they make a sharp turn inward and forward about the nucleus of the sixth nerve, and finally make their exit between the pons and the medulla (Plate IX.) near the eighth nerve. After leaving the pons, the seventh nerve passes into the internal auditory foramen of the petrous portion of the temporal bone, and after passing through the aqueduct of Fallopius emerges from the stylomastoid foramen upon the surface near the lobe of the ear. (See Plate X.)

At this point it is divided into many branches which supply the muscles of the face with motor impulses. (See Plate X.) Upon the fibres of the facial nerve just as it enters the auditory foramen a ganglion occurs, commonly called the geniculate ganglion. This ganglion consists of an aggregation of cell bodies connected with sensory fibres from the chorda tympani nerve, which is a nerve of sensation and is concerned with the special sense of taste. The fibres of this nerve do not, however, remain in contact with those of the facial, but leave it at once, and by way of the Vidian, or superficial petrosal, nerve pass to the superior maxillary branch of the trifacial. In addition to these sensory fibres of the chorda tympani, the facial nerve also has associated with it the nerve of Wrisberg, which is probably sensory in function, and which lies by the side of the facial nerve as its fibres pass from the pons to the auditory foramen, where the ganglion of the chorda tympani just named exists. The fibres of the nerve of Wrisberg then pass to the nucleus of the glossopharyngeal nerve.

Etiology.—Interference with the function of the facial nerve arises from many causes, of which the chief and most frequent are injuries in its course after it leaves the pons. These may be called peripheral lesions, and when the paralysis is peripheral it is called Bell's palsy. Thus, it not infrequently happens that a child is born with facial palsy, which is usually due to injury to the nerve during labor as by the pressure of forceps. So, too, facial paralysis is sometimes seen in children and in adults as a result of a severe blow at the lower part of the ear, or of an attack of mumps in which the inflammation and swelling has been severe. Tumors of the neck and inflammation in the middle ear also may cause facial palsy in this manner.

In adults facial palsy is often due to an inflammation in the stylomastoid foramen as the result of exposure. It is thought by some that this takes place in certain individuals by reason of the fact that this foramen is so small that very slight swelling causes pressure on the nerve and ablation of its function. It is this type of paralysis following exposure to cold which has given rise to the belief among certain ignorant persons that it is possible to be "moon-struck," because a person has slept out-of-doors in the moonlight and has developed facial palsy afterward. The real cause is, of course, the exposure to cold, and not the influence of the moon. That cold cannot always be the cause of this particular form of facial palsy is, however, evident from the fact that the condition is no more frequent in winter than in summer. Perhaps the condition is really one of rheumatic neuritis.

More serious causes of facial palsy are disease processes inside the skull which press upon the nerve before it passes through the aqueduct of Fallopius. These conditions are tumor, inflammatory processes at the base of the brain, most commonly arising from injury, syphilis, or tuberculosis, and occasionally one of the acute infectious diseases. So, too, a fracture of the base of the skull may produce facial paralysis. Facial palsy due to a lesion in the pons is exceedingly rare as a single symptom, as is also facial palsy due to a lesion in the cortex. On the other hand, facial paralysis is usually present in cases of hemiplegia, but in hemiplegia the upper part of the face escapes the paralysis, being innervated from both hemispheres of the brain.

The pathological changes which take place in the facial nerve in cases of facial paralysis depend, of course, upon the situation of the lesion. If the lesion occurs in the nucleus of the nerve, or involves its fibres in such a way that it fails to receive its normal trophic impulses, degenerative changes at once ensue, the neuritis being of the so-called parenchymatous type.

Symptoms.—The symptoms of facial paralysis are very characteristic. The paralysis is nearly always unilateral and often total, in the sense that all the muscles upon one side of the face are impaired in function. It sometimes happens, however, that the muscles of the forehead partly escape. Because of the paralysis of the muscles of one side of the face the patient is unable to wrinkle the brow upon one side, and is not able to close the lids, either as a reflex act, as in winking, or by volition. The corner of the mouth on the paralyzed side is drooped, and if the patient attempts to smile only one-half of his visage is wrinkled. The nasolabial fold is obliterated on the paralyzed side and is usually accentuated on the normal side as a result of the contraction of the muscles which are no longer counterbalanced by opposing muscles. The condition is not painful. If recovery does not promptly ensue the reactions of degeneration speedily develop in the paralyzed muscles, and there may be contractures in them.

Diagnosis.—The manifest paralysis of the muscles of one side of the face, which is particularly noticeable when the patient attempts to smile or frown, renders the diagnosis of facial palsy easy and the symptoms which the patient presents can, moreover, be used very successfully in many cases in determining the site of the lesion. Thus, in some cases of facial paralysis the sense of taste is modified or lost upon the anterior two-thirds of the tongue on the side affected. If this symptom is present, it indicates that the lesion is one which involves the facial nerve between the geniculate ganglion and the point a quarter of an inch above the stylomastoid orifice, where the chorda tympani fibres leave it; or, to put the proposition reversely, if loss of taste does not accompany a facial palsy the lesion is either in the stylomastoid foramen within a quarter of an inch of the orifice or it may involve the nerve before it enters the bone. So, too, if there is unusual sharpness of hearing with some buzzing in the ear, this also is an indication of a lesion near the pons or in the Fallopian canal, since it is due to paralysis of the stapedius muscle which is supplied by the stapedius nerve. If deafness and vertigo are present it is probable that the condition is due to middle-ear disease or to some lesion which also involves the auditory nerve at the base of the brain. A study of the electrical reaction of the paralyzed muscles is also of great value for the purpose of localizing the lesions. Thus, if the lesion exists in the stylomastoid foramen, the muscles of the face are cut off from the trophic impulses which they normally receive from their nuclei in the pons, and as a result the reaction of degeneration speedily develops and may become complete; whereas, on the other hand, if the lesion which causes paralysis is situated in the motor tract above the nucleus of the facial nerve, or, in other words, if it involves the fibres which descend from the motor area of the cortex, the reaction of degeneration does not develop because the muscles still receive trophic impulses. Further than this, in these cases the paralysis is never so complete as in the peripheral type, the patient usually being able to wink and to wrinkle the forehead, the muscles of the forehead frequently escaping. Centric facial paralysis is, however, exceedingly rare unless associated with other symptoms, as already stated. In those rare instances in which the facial paralysis arises from damage to the nucleus of the facial nerve in the pons, there are other symptoms of a pontile lesion producing, in some instances, a crossed hemiplegia, as already described, an associated paralysis of the sixth nerve, or the symptoms of ordinary bulbar paralysis. In these cases, too, reactions of degeneration speedily develop.

Prognosis—The prognosis in cases of facial paralysis varies, of course, with the situation of the lesion and with its severity. The majority of instances get well because they have their origin in an inflammatory process in the stylomastoid foramen. The outlook when the lesions are back of the stylomastoid foramen are not so favorable, and when the nucleus of the nerve or the motor area of the cortex is diseased, the prognosis is, of course, very doubtful as to recovery of power in the muscles of the face.

Treatment.—The treatment of paralysis of the seventh nerve depends somewhat upon the lesion which produces it. For the relief of that form which is due to inflammation in the stylomastoid foramen, it is customary to administer mild alteratives, such as small doses of the iodide of potassium or sodium, in order to hurry the absorption of the inflammatory exudate. It is also advisable to apply a small blister, about the size of a postage stamp, immediately in front of the ear for its counterirritant effect. In those cases in which there is a gouty or rheumatic diathesis, the best results are often obtained by the use of the salicylates in moderately large doses, 10 to 15 grains three or four times a day, or 10 drops of the wine of colchicum root and 10 grains of iodide of strontium may be administered three times a day. The use of electricity for the purpose of maintaining the nutrition of the facial muscles in any case of peripheral facial palsy is not only futile, but may be harmful, for the cause of the muscular wasting is lack of trophic impulse, and as these impulses cannot reach the muscle, it is speedily exhausted if stimulated by the electrical current, when deprived of its ordinary means of recuperation. If middle-ear disease is present, it must, of course, be treated by those measures which are commonly employed by aurists. In cases where the lesion is centric, counterirritation is useless. The only hope is that nature aided by alterative drugs, such as the iodides, may cause an absorption of the results of the local inflammatory process. Where the lesion is severe enough to have destroyed the nerve cells, treatment is also useless.

Facial Spasm.—Facial spasm due to irritation of the facial nerve is a frequent affection. It may be general or localized in one or two muscles. When the orbicularis palpebrarum is affected, the condition is called "blepharospasm." As a rule, the muscles about the mouth are also affected. When this is the case, the condition is called one of "blepharofacial spasm." To this condition the French term "tic convulsif" is sometimes applied. The cause of facial spasm is unknown. It sometimes develops in nervous individuals as a result of a severe nervous shock. In some instances it seems to partake of the nature of a habit spasm, and in these cases not infrequently a lightning-like contraction of the muscles of the face takes place. Sometimes in addition to facial spasm there is also torticollis. Very rarely facial spasm is due to an irritating focus in the motor area for the face in the cortex, and sometimes it is the early or first symptom of an oncoming epileptic seizure. In ordinary facial spasm the muscles are not persistently contracted, but suffer from twitchings which come on in paroxysms, or which occur singly at varying intervals.

The *prognosis* in a case of this kind is not very favorable, although, as a rule, there is no organic lesion to maintain it.

The *treatment* of facial spasm consists in a careful investigation to determine if there is any localized focus which gives rise to reflex irritation, as, for example, disease of the middle ear. If such an area is found, it should of course be removed. In some instances a hyperæsthetic spot in the nasal mucous membrane may be discovered. If no such local area of irritation can be found, there is little left for the physician to do except to administer nervous sedatives, such as the bromides, chloral, and cannabis indica, but these in turn rarely do good in this annoying, harmless, but persistent condition. In young persons who have a tendency to facial spasm as a result of habit, a powerful mental impression may aid in breaking up the habit, particularly if there is a tendency to hysterical manifestations.

The Eighth or Auditory Nerve.—Disease of the auditory nerve may result in *deafness*, *tinnitus*, *vertigo*, and *loss of equilibrium*.

When *deafness* is due to disease of this nerve it commonly arises from some degenerative change, which in turn may be due to the effect of an infectious disease. In other instances the deafness is due to a congenital defect, but in still others it occurs as a part of the course of locomotor ataxia, disseminated sclerosis, or general paralysis of the insane. More rarely it arises from a tumor of the brain or from cerebral syphilis. In deafness due to these causes the so-called cochlear fibres of the auditory nerve are involved. The condition may be differentiated from that form of deafness which is due to disease of the middle ear, the peripheral fibres of the auditory nerve not being affected, and by the fact that the latter class of patients possess the power of perceiving sound transmitted through the bones of the head. Thus, if a tuning fork is placed against the head, or the teeth, the patient can perceive the sounds which it generates if the deafness is due to a peripheral cause, which produces interference with aerial sound conduction in the external or middle ear, but he cannot perceive its sound if the deafness is due to a centric cause, such as a lesion of the cochlea of the internal ear, or of the auditory nerve trunk, or of the auditory pathway to the cortex. Again, in those cases of disease of the auditory nerve of a centric character, the patient does not find it easy to hear in the presence of loud noises, as he often does in cases of deafness due to a peripheral lesion.

The prognosis in deafness due to centric disease of the auditory nerve is very bad.

When *tinnitus* is present, it arises as the result of irritation of the cochlear portion of the auditory nerve which supplies the organ of Corti in the internal ear, or of those fibres which pass from this organ in the nerve trunk itself. The sound may vary from a slight buzzing to a roaring, ringing, or explosive noise which may be so severe as to be insufferable. Suicide is sometimes threatened by persons who are not only persecuted by these noises during the day, but are unable to sleep by night from the same cause. Little can be done for many of these cases. In those instances in which tinnitus arises from middle-ear disease or from anæmia or from the use of drugs, such as quinine and salicylic acid, the condition can often be relieved, as it depends upon an irritation and not upon an actual lesion, as a rule.

The treatment depends upon the cause of the tinnitus. If it is due to an actual lesion of the internal ear the prognosis is bad and treatment is futile. If it is due to gout, anæmia, or similar causes, the prognosis is fairly good.

Vertigo is a condition in which the patient loses the sense of his normal relation to surrounding objects. In some instances he seems to be whirled about in space. In other instances he seems to remain stationary, while other objects are whirled about him. As the patient's conception of his relation to surrounding objects is disturbed, he frequently falls, since this conception has much to do with the motor impulses by which he controls his muscles in connection with the function of muscle sense. The cause of vertigo may be a functional disorder of the branches of the auditory nerve which supply the vestibular portion of the internal ear, as when it occurs in the course of indigestion (autotoxæmia) or under the influence of a drug such as quinine, or it may be due to actual lesions in connection with these nerve fibres such as hemorrhages into the internal ear, or other damage to the semi-circular canals. Sometimes also it seems to be due to reflex irritation produced by disease in the middle ear or in the external meatus. In other instances the presence of a foreign body in the meatus produces vertigo. When vertigo is severe, there may be associated with it great nausea and vomiting, palpitation of the heart, profuse sweating, a sense of approaching syncope, and even collapse. The respirations may be rapid. These symptoms are in part doubtless due to the mental distress or fright from which the patient suffers. There is probably no symptom which causes so much fright and which is so rarely followed by death as severe vertigo. Nothing but the awful apprehension of true angina pectoris approaches the mental distress of the patient who suffers from this condition in its well-developed form.

One form of vertigo arising as the result of disease of the internal ear is called *Ménière's disease*. It is usually severe in its nature. Its onset is sometimes sudden, the patient being seized with prostration, pallor, vomiting, roaring in the ears, and deafness immediately after hearing a loud report which has not, of course, arisen from any extraneous source. This form of vertigo is supposed to be due to a hemorrhage in the semi-circular canals, and resembles in its onset an apoplectic stroke, but it is not characterized by paralysis. In some instances it seems to depend upon arteriocapillary fibrosis. It may or may not be associated with absolute deafness. In the majority of cases the patient suffers from recurrent attacks, but as the disease progresses the attacks last longer and longer, and finally he not uncommonly has constant vertigo.

The prognosis in cases of vertigo depends entirely upon the cause of the disorder. If it is due to an organic disease, unless that disease exists in the middle or external ear and is removable, the outlook is serious. Indeed, the condition may be considered incurable if an actual organic and centric lesion is its cause.

In Ménière's disease the treatment consists in rest in bed, the use of an ice-bag on the head, or blister behind the ear, and the employment of large doses of nervous sedatives, such as the bromides and chloral. Certain practitioners claim to have obtained good results from the administration of large doses of quinine, but it is difficult to see how this drug can do good under these circumstances. Indeed, one would expect it to make the condition much worse.

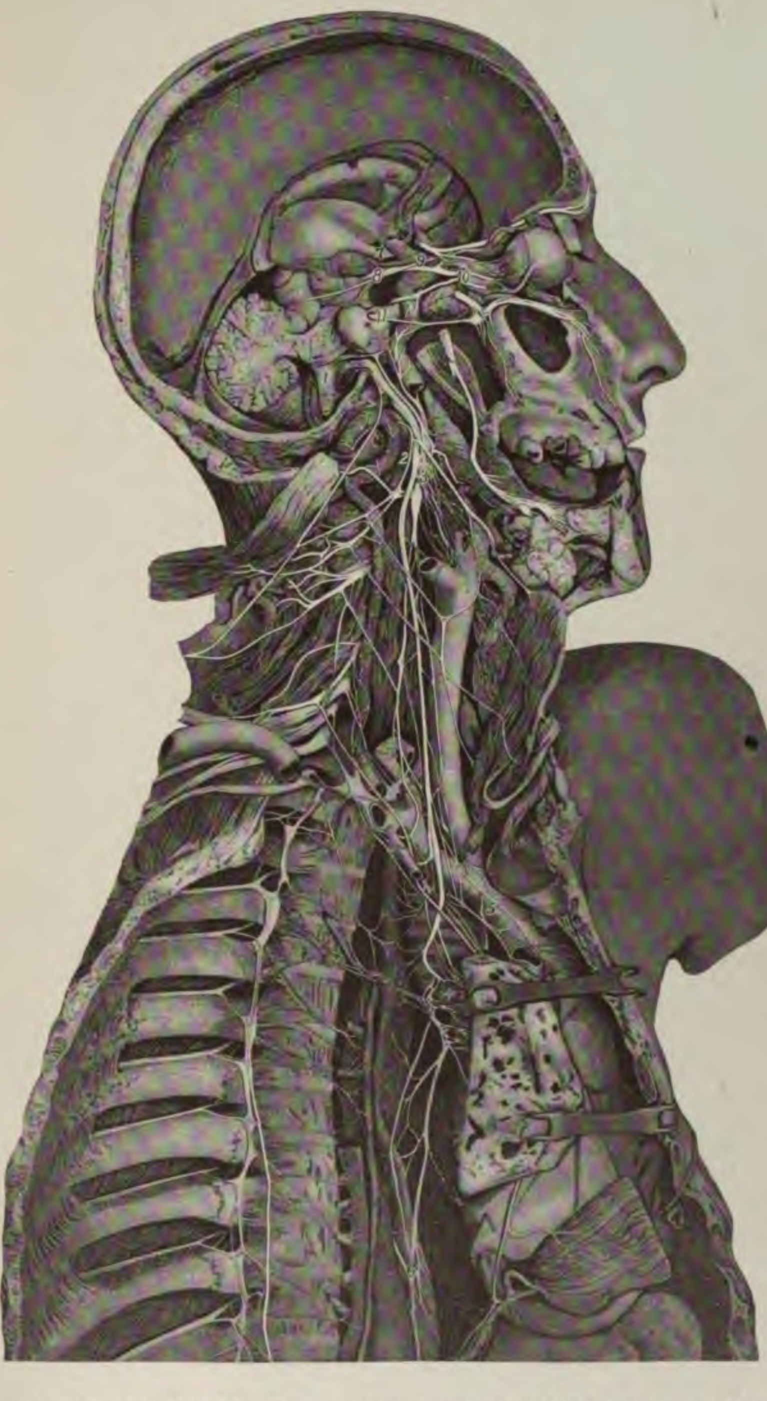
Vertigo which is due to autointoxication arising from an abnormal state of the bowels or kidneys should be treated by the administration of diuretics, cholagogues, purgatives, and free sweatings.

The Ninth or Glossopharyngeal Nerve.—The ninth nerve is the nerve of sensation of the pharynx, the palate, and the middle ear. It is also probably connected with the special sense of taste in the posterior third of the tongue, although it is considered by some physiologists that those fibres which are connected with this function join the glossopharyngeal fibres from the fifth nerve. Some of its sensory fibres enter the medulla oblongata near the olivary body and terminate in the gray matter on the floor of the fourth ventricle, while another set of fibres ends in the substantia gelatinosa. From the latter point some of its fibres ascend into the brain. In addition to its sensory function it also contains motor fibres which spring from cells known as the nucleus ambiguus. These fibres make their exit from the side of the medulla back of the olivary body, and escape from the skull through the jugular foramen. The motor fibres supply the muscles of the larynx, the œsophagus, and pharynx, and are also connected with the function of respiration, deglutition, and phonation.

Paralysis of the glossopharyngeal nerve is exceedingly rare, and therefore we possess but little information, either clinical or pathological, concerning its condition in disease. Should the nerve itself be damaged, the symptoms consist in *loss of sensation in the upper half of the pharynx, loss of the sense of taste* on the posterior half of the tongue, and *difficulty in swallowing* because of paralysis of the pharyngeal muscles and because of the loss of reflex irritability of the mucous membrane of the pharynx. Such a condition sometimes develops during postdiphtheritic paralysis. When lesions of the nuclei of this nerve take place, the symptoms are practically those of bulbar paralysis (which see).

The Tenth or Vagus Nerve.—This nerve, sometimes called the pneumogastric nerve, is composed of both sensory and motor fibres. The sensory fibres pass upward from the various portions of the body which they supply and enter two ganglia, one of which, the upper, is large and oval, and the other is long and irregular in outline. After leaving these ganglia the fibres pass to the medulla, some of them terminating in the gray matter which exists in the floor of the fourth ventricle, thereby forming the sensory centres connected with respiration and the heart. Other of these fibres join the ninth nerve and end in the substantia gelatinosa and from this point new fibres ascend to the brain. Those fibres of the pneumogastric which are

PLATE XI.



The Vagus and Sympathetic Fibres of the Right Side and Their Anastomoses. (Modified from Rüdinger.)

1, origin of vagus; 2, anastomosis with sympathetic; 3, superior laryngeal and pharyngeal plexus; 4, the pulmonary plexus; 5, the inferior cardiac fibres, with sympathetic fibres; 6, the œsophageal plexus. The course of the optic nerve (7), the oculomotor (8), the trochlearis (9), the abducens (10), and the facial *(11) are also shown.*

motor in function take their origin from the cells of the nucleus ambiguus and escape from the side of the medulla, forming the main trunk of the nerve. The distribution of the afferent and efferent fibres of this nerve is well shown in Plate XI.

The tenth nerve has a far larger distribution than any other craniai nerve, supplying the pharynx, the larynx, the heart, the lungs, œsophagus, stomach, and intestines, and even the external ear through an auricular branch. According to some physiologists it is the chief motor supply of the palate. It joins the glossopharyngeal, or ninth nerve, and certain sympathetic nerve fibres, in the formation of the pharyngeal plexus which supplies the pharyngeal muscles.

The superior laryngeal branch supplies the cricothyroid muscle, the thyroepiglottic and aryepiglottic muscles, and the inferior laryngeal branch, sometimes called the recurrent laryngeal, supplies the other laryngeal muscles. By means of the sensory fibres which exist in the superior laryngeal nerve, the mucous membrane of the epiglottis possesses sensation, and by means of those sensory fibres which exist in the recurrent laryngeal the mucous membrane below the vocal cords is supplied with sensory filaments. If the sensory fibres in the superior laryngeal nerve are stimulated, the respirations become slower and deeper, or they may be arrested as the result of a reflex impulse which passes to the centre in the medulla. Closure of the glottis may also be produced in this manner.

The pulmonary branches of the vagus contain motor fibres which supply the unstriated muscles of the bronchi, and sensory fibres for the mucous membrane of the bronchi. They apparently also contain fibres centripetal in character, which when stimulated diminish the inhibitory action of the pneumogastric nerve upon the heart, thereby producing tachycardia. Those branches of the vagus which supply the œsophagus innervate its muscles, on the one hand, and supply its mucous membrane with sensory filaments on the other; while those which pass to the stomach contain not only fibres which govern its muscles, but also other fibres which control its secretion and its blood supply. The same facts hold true of those fibres which pass to the intestines. Last, but by no means least, the pneumogastric sends fibres to the heart, and through these pathways an inhibitory action is exercised which if stimulated may temporarily arrest the heart in diastole.

Not only is the pneumogastric nerve of very great importance because of the multiple functions which it possesses, but it is also of great importance to the clinician because it not infrequently suffers from disease. Though it is rarely the victim of primary neuritis, cases of rheumatic neuritis of both recurrent laryngeal nerves have been reported, and instances in which this nerve has been involved in cases of multiple neuritis due to poison, such as alcohol, for example, are by no means rare. So, too, it sometimes suffers in diphtheritic paralysis, and from neuritis arising from the poisons of various infectious diseases such as typhoid fever, pneumonia, scarlet fever, malaria, and influenza, from tumors and inflammation in the mediastinum, disease of the jugular vein, tuberculosis of the mediastinal glands, and from pressure upon the nerve exercised by reason of dilatation of the left auricle in cases of mitral stenosis. In those instances in which the centres of the pneumogastric nerve are affected by disease, we find that tumors, hemorrhagic extravasations, the lesions of locomotor ataxia, and disseminated sclerosis are the causes. In still other instances the disorder of the function of this nerve develops as the result of bulbar paralysis. Cases are also on record in which the fibres of the nerve outside of the medulla have been pressed upon by tumors, by the exudations due to meningitis, hemorrhages, and by bone disease.

The *symptoms* of disorder of the function of the vagus nerve are, of course, very varied. If the lesion exists at the base of the brain it nearly always happens that there is paralysis of the other cranial nerves, particularly of the ninth, eleventh, and twelfth. In such a case if the fibres on one side alone are affected, there is unilateral paralysis of the fauces, the palate, and the larynx. The *speech is nasal*, and the act of *swallowing* may be *impaired*. There is also interference with the action of the vocal cords. If the recurrent laryngeal branch is affected, there is *laryngeal paralysis*. The vocal cord on that side remains midway between adduction and abduction, and fails to move during phonation. If *both* of the recurrent laryngeal nerves are *paralyzed*, the patient suffers from *aphonia*, *inspiratory stridor*, and *dyspnœa*. When the pulmonary fibres are affected, particularly if the lesions are bilateral, the *respirations* may become *rapid* and *irregular*.

Irritation of the pulmonary fibres, directly or indirectly, may cause spasm of the bronchial muscles, and hyperæmia and congestion of the bronchial mucous membrane (asthma).

If the gastric fibres are involved, there may be *vomiting*, *pain* in the stomach, and *loss of the sense of hunger* and *thirst*. When the cardiac fibres are severely affected, the *pulse rate* may be markedly *accelerated*. If they are irritated, an *exceedingly slow pulse* may be present.

The *treatment* of disorders of the vagus nerve depends largely upon the cause which underlies the disturbance. If there is reason to believe that there is a syphilitic exudate at the base of the brain, or a syphilitic arteritis, the iodides and mercury are, of course, indicated. So, too, in that form of disorder of the vagus which results from lead poisoning, the iodides, hot baths, and purgatives are required. If it is believed that a gummatous growth exists in the thorax which irritates the vagus by pressure, antisyphilitic treatment is necessary. If there is a distinct rheumatic history leading one to believe that the recurrent laryngeal nerves are suffering from rheumatic paralysis, already mentioned, the iodides and the salicylates are advisable. Digitalis may also be useful for the purpose of stimulating the pneumogastric nerve in those cases in which tachycardia is present, and atropine may be used with the object of diminishing irritation in this nerve by depressing its peripheral fibres when the pulse is unduly slow.

Eleventh or Spinal Accessory Nerve.—The eleventh or spinal accessory nerve is composed of two parts, an accessory portion, which goes to the pneumogastric, and a spinal portion. The accessory branch is formed by several fasciculi which spring from the medulla in series with the roots of the vagus. These fasciculi form a trunk, and to this trunk are joined the fibres from the spinal portion. The nerve leaves the cranium with the pneumogastric. In its passage through the jugular foramen it sends fibres to the root ganglion of the vagus, while others pass over the surface of this ganglion into the pharyngeal, superior laryngeal, and recurrent laryngeal nerves. Most of the motor fibres of the pneumogastric are derived from this accessory branch. The spinal portion of the nerve arises by a series of roots which spring from the lateral portion of the spinal cord, even as low as the sixth or seventh cervical nerve roots. They spring from the lateral column near the origin of the posterior nerve roots and form an ascending trunk, which enters the skull and unites with the accessory portion, as already described. Before entering the jugular foramen, however, certain of its fibres leave the accessory portion, make a sharp turn backward near the internal jugular vein, and enter the deep surface of the sternomastoid muscle, which muscle it supplies. Passing through this muscle, it enters under the trapezius, a short distance above the clavicle. Here it anastomoses with fibres from the third and fourth cervical nerves, forming a plexus, which supplies the trapezius muscle.

Symptoms.—Disturbances in the function of the accessory nerve, so far as its spinal part is concerned, result in torticollis, which occurs as congenital wryneck, as wryneck due to injury, and true spasmodic wryneck. The *congenital form* is due to some defect in development or to injury of the sternomastoid muscle at the time of delivery. The right side is affected in the majority of cases. The sternomastoid muscle is not in the spasm, but the head is drawn to one side and rotated to the opposite side as the result of shortening of the muscle upon the side to which the head is drawn. Not rarely there is associated with this atrophy some wasting of the muscles of the face upon this side. *Spasmodic wryneck*, on the other hand, is due to a true spasm of the muscles supplied by the spinal accessory. It is not met with in children, and very rarely in advanced life, but occurs most frequently in middle-aged persons. In a certain proportion of cases the patients are distinctly hysterical, and the spasm follows some nervous shock. In other cases no hysterical stigmata are present, and it is thought that the condition is due to that somewhat indefinite state called "rheumatism." This form of wryneck differs from the congenital variety in that it is usually accompanied by pain. The spasm may not be constant but intermittent. The chin is often protruded and raised. At times the spasm extends to the muscles of the face, and facial twitching may occur. Often the condition becomes one of tonic spasm after having begun as clonic spasm, and if the condition persists for any length of time the affected muscles may undergo hypertrophy, and those on the opposite side may atrophy from disuse.

Those forms of wryneck in adults which are characterized by intermittent or clonic spasms are rarely due to rheumatism, so called, but depend upon some neurosis; whereas, the tonic spasm may be due simply to muscular fixation through pain. In the latter class of cases the prognosis is exceedingly good, the condition usually disappearing under the use of hot applications or counterirritant liniments, and the internal administration of the salicylates and the iodides. Certain persons have advised the intramuscular injection of atropine, in the dose of $\frac{1}{50}$ of a grain, directly into the belly of the afflicted muscle in order that it may depress the peripheral motor nerve endings. While this is efficacious in some cases, it is prone to produce moderate systemic symptoms, and is not to be resorted to unless the condition fails to yield to the plan of treatment already suggested.

The type depending upon a neurosis is much more difficult to treat. It often remains unchanged for many months, and indeed may become a permanent condition. Sometimes a nervous shock, or some accident, may suddenly end the spasm.

Under the unfortunate name of "spurious wryneck," a condition of wryneck develops as a result of caries of the spine, the spasm of the muscle being due to the lesions in the vertebræ, or the distortion is due to the fact that these bones do not properly support the head.

Under the name of "spasmus Nutans," or "nodding spasm," a condition is met with in which the muscles upon both sides of the neck are affected in such a way that there is a nodding movement. It occurs in poorly nourished, neurotic individuals, and closely resembles habit chorea. The symptoms become most marked when attention is called to them, and are usually absent during sleep.

Paralysis of the spinal accessory may be due to injury, disease of the vertebræ, muscular atrophy, or any form of disease of the spinal cord in the cervical region. As the result of the paralysis there may be loss of power to rotate the head upon the vertebral axis. The sternomastoid muscle does not stand out prominently as it does when affected by spasm, and there is difficulty in raising the arm at a right angle to the body. In those comparatively rare cases in which the paralysis is bilateral, the head may appear to be fixed, as if the fixation were due to spasm.

Twelfth or Hypoglossal Nerve.—The twelfth or hypoglossal nerve arises from a group of cells in the floor of the fourth ventricle, at its lowest point. Its fibres emerge from the medulla, and escape from the skull through the anterior condyloid foramen of the atlas, and so pass to the muscles of the tongue. Injury and disease of this nerve rarely take place in its peripheral filaments. Nearly always when it is affected, the lesion is in the bulb or in the brain. Thus, out of 79 cases collected by Ascoli, in only one-third were the peripheral fibres affected. The causes of centric disease of the hypoglossal nerve are an inflammatory process or growth at the base of the brain, or in the medulla oblongata. When the lesion is in the medulla, there is usually bilateral paralysis with wasting of the tongue, but as the nuclei of all the cranial nerves have their origin nearby, it nearly always happens that there are evidences of paralysis of the other cranial nerves present. Inside the bony casement the causes of hypoglossal paralysis are inflammatory exudates, hemorrhages, and disease of the bone. Very rarely the peripheral filaments of the nerve, after they take their exit from the atlas, suffer from neuritis.

In some cases of locomotor ataxia, syringomyelia, and multiple sclerosis, there is hemiatrophy of the tongue with paralysis. The same symptom also occurs when damage is done to the peripheral fibres of the nerve. The tongue lies on the floor of the mouth, and it is impossible for the patient to move the tongue upon the paralyzed side. If the tongue is protruded, it deviates to the paralyzed side, but sometimes while the main body of the tongue may be deviated in this manner, the tip points toward the sound side. Remak points out that while the tongue lies on the floor of the mouth apparently paralyzed, it can be easily pushed about by the finger if the paralysis is organic; whereas, in hysteria, all attempts to move the tongue by the finger-tip cause efforts to resist these movements. The paralysis of the tongue impairs the speech, and also interferes with deglutition and mastication when the paralysis is bilateral.

The prognosis varies with the cause of the lesion. In syphilitic cases recovery sometimes occurs under active mercurial treatment. In the so-called rheumatic cases, it may take place under the influence of the iodides or salicylates. If an actual lesion at the point of origin exists, the prognosis is bad.

DISEASES IN WHICH THE CHIEF MANIFESTATIONS ARE IN THE MUSCLES.

MUSCULAR DYSTROPHIES.

Definition.—Under this term are described several related maladies, characterized by alterations in the trophic state of the muscles, which are met with almost always in early life and which are not due to disease of the nervous system—that is to say, they are primarily muscular in origin. The alterations in the muscles cause loss of power and the paralysis may be thought to be due to a spinal lesion, but this is not the case.

Muscular dystrophy has been divided by Erb into three forms, namely, pseudomuscular hypertrophy, Erb's juvenile dystrophy, and the Landouzy-Déjerine type of dystrophy. Several other types have been described by other writers. (See below.)

Etiology.—The etiology of these dystrophies is not known, but they are all believed to be dependent upon faulty development of the muscles affected. In other words, it would seem as if the vitality of certain muscles is of such a character that they undergo senile changes early in life. When injuries and the acute infectious diseases seem to be causative factors it is probable that they act only indirectly in that they hasten the degenerative changes in the feeble parts.

Pathology and Morbid Anatomy.—The changes in the muscles in cases in which atrophy takes place consist in a wasting and thinning of the muscle fibrils within the sarcolemma. They become shortened and pigmented. In other cases a true degenerative process goes on, the fibrils become swollen, suffer from fatty or albuminoid changes, and show fatty and granular masses within the sarcolemma, until finally the sarcolemma may contain nothing but fatty globules. In a third form of dystrophy there is, in addition to fatty degeneration in the fibrils, a deposit of fat between the sheaths covering the fibrils. With these changes there is also an overgrowth of connective tissue, and as a consequence a muscle which is so large as to appear strong and powerful is in reality feeble or powerless.

Pseudomuscular Hypertrophy.—This form of muscular dystrophy is essentially a disease of early childhood beginning between the second and seventh year. It is characterized by enlargemen calves of the legs, which soon are seen to be prop the rest of the child. It is then noticed that these this weakness causes the patient to walk awkwardly obstacles, to tire easily, and to have difficulty in to the erect posture, so that the patient gets on his does, largely by the aid of the forelimbs. But whil cles of the calves and of the anterior portion of the are enlarged, it will also show that the gluteal mu that the muscles of the back are weakened, with developed anterior curvature of the spine and a p

When the disease is still further advanced alteratic muscles about the shoulder-blades is usually presen supraspinatus, the biceps, and the triceps under appear to be increased in size because they seldom which makes the muscles in the legs seem unusual however, such a fictitious hypertrophy may be p because of loss of power in the rhomboidei, in the and in the serrati. The shoulder-blades occupy position, and because of the wasting of the muscl may be great feebleness in the movements of the u of the forearm and hand, however, usually escape

Contractures appear, which, like contractures i cular atrophy, result in deformities such as club-fo upon the thighs and the thighs upon the pelvis. take place in the arms. It is a fact worthy of note of muscular atrophy, this disease does not show f the muscles, nor are the reactions of degeneration muscles are considerably atrophied. Sensation is are preserved, unless the muscles are so complete unable to contract.

Prognosis.—The prognosis in these cases is Periods of arrest in the advancement of the disea mately the patient is absolutely helpless. Death ne muscular hypertrophy directly, being caused in mc diseases which attack the enfeebled body.

Erb's Juvenile Muscular Dystrophy or Sc This form of dystrophy begins at about the time tween twelve and sixteen; rarely as late as the twen muscles, the trapezii, the latissimus dorsi, the rh toids undergo apparent hypertrophy with progre results in the falling forward of the shoulders, so tl attitude is maintained as if there was a fracture scapulæ are prominent. In some cases the diseas if it does not, the loss of power extends to the m various forms of spinal curvature develop. After t and those of the thigh become enfeebled. They may seeming hypertrophy. When the muscles of the leg may be present. The chief difference, therefore, between pseudomuscular hypertrophy and Erb's juvenile type of muscular dystrophy is the fact that the latter disease develops later in life, that it affects the upper extremities before it affects the lower extremities, and that atrophy is more marked than hypertrophy.

Landouzy-Dejerine Type of Muscular Dystrophy or Facioscapulohumeral Type.—This type of muscular dystrophy may appear in early childhood or in adult life. It is characterized by the peculiar fact that the atrophy develops in the muscles of the face, particularly the orbicularis oris, and extends to the muscles of the cheeks and those of the forehead, with the result that the lips lose power; the mouth cannot be closed, but has a peculiar pouting expression, and speech, at least so far as labial and lingual sounds are concerned, becomes very defective. So, too, the face loses its power of expression from a similar cause, and there is dribbling of saliva because the lower lip sags. Both sides of the face are usually affected. The orbicularis palpebrarum usually escapes, as do also the masseter muscles. Later, the muscles of the shoulders become affected, and finally those of the trunk and legs become involved until the case closely resembles either one of the forms of muscular dystrophy just described, save that the facial symptoms are prominent.

In some cases the symptoms of these three forms of dystrophy overlap one another to such an extent that it is difficult to determine to which type an individual patient belongs.

Treatment.—No form of special treatment can produce advantageous results. The best that can be done is to order for the patient an out-door life, if possible in the country, and in a climate where he can remain for many hours in sunshine and in a place where he can receive excellent food. Gentle massage and Swedish movements may be employed, but care must be taken not to tire the wasting muscles. The most that can be expected from this plan of treatment, however, is the temporary arrest of the malady. No real improvement usually occurs. Efforts to correct deformities produced by contractures are generally useless, since the relief is but temporary.

MUSCULAR ATROPHY OF THE PERONEAL TYPE.

Definition.—This is a form of progressive muscular atrophy which begins in the muscles innervated by the peroneal nerves, and which does not extend higher than the knee. After the symptoms in the legs have developed, a somewhat similar condition may affect the muscles of the hand and forearms. It is sometimes given the name of the "Charcot-Marie-Tooth" form of progressive muscular atrophy, or is called the " progressive neural muscular atrophy of Hoffman," or primary neuritic or neurotic atrophy. It is an uncommon disease, but not so rare as was formerly believed.

Etiology.—There seems to be a distinct hereditary predisposition, since it frequently affects several members of a family, and can be traced through several generations. It usually develops during the first two decades of life.

Pathology and Morbid Anatomy.—The condition erative changes in the muscles, in the nerves, and terior columns of the spinal cord. There is also som atrophy in the anterior horns of the gray matter, and the lateral columns of the cord. Whether the diseas nerves primarily, or whether the lesion begins in th gray matter in the spinal cord, is unknown.

Symptoms.—The symptoms of this form of musc *weakness of the muscles of the foot* and of the *pero* followed by atrophy in the anterior and posterior ti production of *drop-foot*, which makes it impossible f Unlike the two forms of muscular dystrophy just d *tractions* are present in the affected muscles, and t activity and of electric excitability, so that the las of degeneration may finally be present. The various come on as secondary conditions. When the upper e fibrillary contractions can be seen in the muscles of t of power has been present for some time, deformities from contractures. In some cases the progress of th rapid, but in others it is equally slow. In still othe so that all the muscles of the extremities and trunk, face, may undergo atrophy.

Prognosis.—The prognosis as to recovery is hope live for many years unless destroyed by some inte

Treatment.—Aside from hygienic measures, no m arrest the progress of the malady.

FUNCTIONAL NERVOUS DISEASES AND DISPUTED PATHOLOGY.

MYOTONIA CONGENITA.

Definition.—Myotonia congenita, commonly calle is an exceedingly rare affection not dependent u vous system, and characterized by hypertrophy o with the proliferation of their nuclei. These ch power.

Symptoms.—The chief symptom of Thomsen's dis *muscles*, which develops after they have been quiesce muscles comes on when the patient attempts to move as to make walking practically impossible. Because patient to balance opposing muscles in different po may fall. After a time, if the patient persists in his make other movements, the spasm passes off, and the the normal individual so that ordinary movements c ease; but if the muscles are irritated by percussion or another attempt is made to move, they instantly pass into spasm. The affected muscles develop electrical reactions of a peculiar type, namely, a tonic contraction under the galvanic current, which comes on sluggishly and lasts longer than in health. If the electrical application is continued contraction waves pass over the muscles, but there is no marked atrophy or great loss of power except in so far as the spasm interferes with ordinary muscular movement. The disease is so exceedingly rare that less than 40 cases have been reported. A physician named Thomsen, himself a sufferer from the disease, first described it.

PARAMYOCLONUS MULTIPLEX.

Definition.—Paramyoclonus multiplex is a condition of the motor nervous system in which sudden contractions of the muscles take place, the contractions resembling those produced by the use of a slowly interrupted faradic current. It is sometimes called myoclonus, multiple myoclonus, myoclonus epilepticus, spinal epilepsy, and Friedreich's disease, but it is not to be confused in the mind of the student with Friedreich's ataxia, a very different malady. It is a rare affection.

Etiology.—The cause is unknown. In a very few instances it has been thought to be hereditary, but in all probability this is the case only in the sense that the parents or grandparents have suffered from neurotic states. It is not rarely associated with epilepsy, and it may develop when a nervous system naturally unstable is sapped by excessive mental overwork or other stress.

The contractions may simultaneously affect similar muscles in both limbs, or may occur in series involving first one side and then the other, or pass from muscle to muscle. Very rarely only one side is affected. In most instances the face and trunk muscles escape as in Friedreich's original case. The ocular muscles are never affected. The arms are more commonly affected than the legs and the muscles about the arm and shoulder and those of the thigh are more frequently and severely affected than those of the forearm or leg. The muscles of the hands and those of the feet escape. The severity of the contractions varies greatly. In some instances they are so moderate as to be noticeable only when the patient is stripped of his clothing. In others they are severe enough to throw him off his feet. The motions or attitudes of the patient vary, of course, with the muscles affected and the degree of their contractions. The effect of voluntary movements upon the contractions is also varied. In some instances, as in Friedreich's first case, a voluntary movement inhibits or diminishes the contractions, but in other cases voluntary movement seems to exaggerate it. Mental excitement increases them. They cease during sleep and are usually less severe when the patient is standing than when he is sitting or lying down. If the affected muscles are irritated by tapping them a spasm is induced. The deep reflexes may be increased or diminished, but electrical irritability is not altered nor do any trophic changes occur. Some superficial vasomotor palsy may be present in the extremities.

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Diagnosis.—Paramyoclonus multiplex is separ
fact that the movements of chorea resemble thos
tain groups of muscles act together and do not c
an electrical shock. Voluntary movements are p
myoclonus and to increase those of chorea. Cho
affection, while myoclonus is usually bilateral. T
are so commonly affected in chorea, are rarely
under discussion. Electrical chorea, or Dubini's
paramyoclonus multiplex by the fact that it is
progressive muscular atrophy, and paralysis, and
part of the muscles to faradic electricity. Fro
myoclonus is separated by the presence in hysteri
of that state, such as disorder of the color fields a
or anæsthesia. In those cases of hysteria in which
the differential diagnosis may be impossible.

Paramyoclonus multiplex may be separated fr
convulsifs," described by French neurologists, by t
tion the movements are more like gestures and
is present.

Prognosis.—The prognosis as to complete and p
good. Rarely death ensuesin a few months. Mor
persists in varying severity for years.

Treatment.—The treatment consists in measures
ment of the general health by out-door life, good f
causes of nervous irritation and exhaustion. Re
phorus, iron, and similar roborants are useful to th
tive. Occasionally a carefully regulated course o
well-equipped and well-managed sanatorium is ser

PARALYSIS AGITANS.

Definition.—Paralysis agitans, sometimes calle
"Parkinson's disease," is a condition in which dif
especially the forearms and hands, are affected
When the disease is well advanced, the patient le
peculiar attitude, and may suffer from festination.

Etiology.—The precise cause of paralysis agitan
been thought to follow severe nervous shock and in
vous system. In other instances it has followed
Thus, in one case under the writer's care, the t
corporation, after many years of hard work, develo
of paralysis agitans. He was quite certain that t
those muscles which were employed in the signing o
a day.

Paralysis agitans develops most frequently betw
fifty-five. Gowers analyzed 80 cases and found t
to be fifty-two years, and Wollenberg found that 10

at ages varying from fifty to fifty-five. The disease also not infrequently develops during the fifth and seventh decades of life. A few cases occurring in early adult life and in childhood have been reported. Hadden, Gowers, Berger, and others have seen the disease in individuals whose ages ranged from twenty to thirty, and Weil and Rouvillois have reported a case occurring in a child of ten. Lannois also mentions a case occurring in a child aged twelve.

Men are more frequently affected than women. Thus, of 67 cases collected from the reports of St. Thomas' and St. Bartholomew's Hospitals, London, 47 occurred in men and 20 in women. In 78 American cases Dana found the proportion of men to women to be as 5 to 3.

The neuropathic constitution may be considered as a predisposing cause, but direct inheritance of the disease is rare. A few instances of apparent direct transmission from parent to offspring have been reported, and Berger cites one in which the disease appeared in three successive generations. Borgherini reported 7 cases occurring in a family of 9 brothers and sisters. Three children of these individuals developed paralysis agitans between their fortieth and fiftieth years.

Pathology—Nothing is known of the pathology of this affection which throws any light on the cause of the symptoms.

Symptoms.—The symptoms of paralysis agitans are *tremor, muscular rigidity,* a *retardation of ordinary voluntary movements,* and a *change* in the *gait.* The tremors are rhythmical in character, amount to four or five per second, and move the part in various directions. Thus, when the hand is affected, the fingers may be flexed and extended, abducted and adducted. The most common movement is a rubbing of the thumb against the index finger, in much the same way that a pill might be made by such a rolling movement. In some instances, particularly if the patient becomes excited, the amplitude of the movements becomes greatly increased, so that the hand or the head shakes as it does in a severe rigor. Unlike the intention tremors of certain forms of organic nervous disease, the tremor of paralysis agitans is passive. Be the position of the body and arms what it may, the trembling continues, and while certain attitudes may diminish the amplitude of the tremor, it is always present except when some definite and active movement is attempted, when the tremor diminishes or even ceases. Thus, if the fist is clenched, or the patient shakes hands with a friend, the movement may stop momentarily. On the other hand, nicely adjusted muscular movements such as are involved in writing do not stop the tremor, and for this reason handwriting is usually impossible when the disease is well developed.

The tremor not only involves the head and arms but extends to the legs as well, and it may affect the muscles of the thigh. Rarely the muscles of the jaw are affected. The tremor continues only during the waking hours, and usually ceases in sleep. Not rarely paralysis agitans is associated with insomnia because the twitching movements keep the patient awake, or the aching in the affected muscles makes the patient so uncomfortable that sleep is postponed until the patient is exhausted. As the patient stands in front of the physician, the chin is usually pushed forward and the body bent forward. The arms and the elbows are slightly flexed.

After the disease is well developed, chronic musc
symptom which is even more constant than tremor.
parts that we have spoken of, but also the muscles of
even those of the face. As the patient walks, his gait us
and the attitude is that of a person who is attempti
It is unfortunate that the term "paralysis agitans"
stages of this disease, for paralysis, in the sense of gr
occurs in its very last stages, and not infrequently
from the malady for years without developing para
skin covering the parts affected is not infrequently u
of profuse perspiration. There are no disturbances
mind is unaffected save by the mental depression wh
annoyance of the disease.

Diagnosis.—Paralysis agitans is to be separated f
by its onset late in life, by the fineness of the trem
is present when no voluntary movement is made, a
rigidity. An examination of the eyes fails to reveal
changes in the optic nerve which are characteristic of
paretic dementia the tremor is not rhythmical, occ
makes a movement and not when at rest, and there
tremor the mental disturbance, the scanning spee
paralysis.

Sometimes in old persons a senile tremor develops
the head, and is increased rather than decreased by
in some instances senile tremor bears a close res
agitans. Indeed, it has been suggested that the latte
a premature nervous senility.

Prognosis.—Paralysis agitans does not materially
quently lasts for twenty years. Recovery practicall
porary remissions in the severity of the symptoms m

Treatment.—As in many diseases which apparentl
tional derangement, treatment is not followed by ve
The patient should be forbidden to subject himself
worry, which materially increase the severity of the
should be done to reinstate his nervous balance by
and freedom from care. Massage and electricity, as
In some cases they do harm by increasing the exha
muscles.

A very large number of drugs have been employ
results, but none have the confidence of those mem
who have had large experience. Of all the reme
recommended, hyoscine seems to have received th
praise. It should be given in the dose of $\frac{1}{100}$ of
thrice a day; the size of the dose and the frequenc
being governed by the severity of the tremor and b
the patient to drugs of this character. Duboisine
similar doses. Where there is much aching of the
compresses give relief, and if the patient is in a con

tation, the bromides and chloral are advantageous. The employment of such powerful nervous and vascular sedatives as veratrum viride and gelsemium must be resorted to with great caution. In doses large enough to quiet the tremor they are prone to produce too much circulatory depression.

CHOREA MINOR.

Definition.—Chorea minor, or acute chorea, sometimes called "Sydenham's chorea," or "St. Vitus' dance," is a nervous disease characterized by irregular, purposeless movements, sometimes limited to certain muscles, but at others involving all the muscles of the limbs, face, and trunk. It affects, in the great majority of instances, children between the fifth and fifteenth years of life.

Etiology.—That sex acts as a predisposing cause of chorea is shown by the fact that girls are affected three times as often as boys, and in the period of life from the fifteenth to the twenty-fifth year males escape almost entirely. The age which predisposes to its development, or is most susceptible, is from the fifth to the fifteenth year. After the fifteenth year it steadily decreases in frequency until the twenty-fifth year is reached, after which it is very rarely met with. The disease may, however, occur at all ages. Nervous, high-strung children suffer from it more frequently than those of a more phlegmatic temperament, particularly if, in addition, they are anæmic, and have a family history indicating that they are prone to attacks of acute rheumatism.

Of the exciting causes may be named sudden shock or acute mental excitement, but in all such cases these causes are indirect, that is, they serve to disturb the nervous equilibrium, already unstable from other causes. In some cases the disease seems to be acquired by association with a choreic child, and in this way a large number of children in homes and asylums may become affected. Whether many of these cases are true chorea or merely imitations of it, or due to hysteria, is difficult to determine.

That there is a very close relationship between acute articular rheumatism and true chorea is certain. Even those physicians who deny that the rheumatism produces chorea are forced to admit that the occurrence of chorea after attacks of acute rheumatic infection is remarkably frequent. Not rarely chorea complicates the development and progress of the acute endocarditis produced by this infection. Whether the poison of rheumatism affects the nerve cells of the brain, or whether the disease is due to changes in the finer capillaries supplying the brain, or to minute emboli, is unknown. Certain clinicians have endeavored to show that chorea is due to a specific infection, and Pianese isolated a diplococcus which was capable of producing an experimental chorea. There is no proof, however, that such a specific agent exists.

Under the name of *chorea gravidarum*, a form of the disease is met with in pregnant women, usually only in primiparæ. In these cases the gravid state seems to develop a condition of lack of nervous equilibrium, for the condition ceases, as a rule, with the termination of pregnancy. Occasionally

in old age a senile chorea develops, but it is a
chorea of childhood.

Frequency.—Chorea is much more frequently m
in the United States. Morris Lewis has shown t
frequency is March.

Pathology and Morbid Anatomy.—Cases of chore
quently, and therefore we have not as much infor
mortem findings in this disease as is desirable. I
in which death occurs during an attack of chorea,
endocarditis, or chronic endocarditis with an acu
rarely some degeneration of the myocardium.
instances the results of examining the brain have
positive results which have been obtained are com
be most variable. In some instances an inten
present; in others minute hemorrhages; in still
small areas of inflammation and softening; while
signs of meningitis, or thrombosis of the smalle
the brain. So, too, the changes which have been
have been too varied to lead one to believe that th
connected with the disease. Some observers have
the result rather than the cause of the condition.
sometimes develop in persons who have suffered
lesion, but there is no reason to believe that this fo
minor have any close anatomical relationship.

Symptoms.—The onset of chorea may be either
those cases in which it is gradual, it is first noted
and seems unable to keep still. Not rarely it is *a*
and falls over, or bumps into articles of furnitur
developed the child is continually restless, the ar
in every possible direction. Sometimes the musc
worked as if the child was uncomfortable by rea
The body is rotated from one side to the other, an
one shoulder then to the other, then elevated, the
movements are marked, *walking is interfered* wit
for the patient to stand. Unlike most involuntar
ments of chorea are not confined to one group
attack different groups alternately. Neither is the
of a tremor. It is *like a voluntary movement,* but i
plete. Not infrequently the child *laughs* and *cries*
The movements affect the upper extremities more
ties. The tongue is sometimes involved, and for th
be disturbed. The child also frequently gives ver
smacking sounds, due to the action of the muscle
and tongue. These sounds may also be increased b
Occasionally, chorea may be limited to one limb,
chorea, or when it is confined to one side of the bod
These motions distinctly interfere with ordina
and it becomes almost impossible for the child to

slowly; if it is to be successfully carried out, it must be done with great rapidity. Any cause which produces mental excitement in the patient greatly increases the severity of the jerking. Some clinicians have recorded an exceedingly severe form of chorea in which the patient has such violent muscular movements that he bites his tongue, can not eat, and is thrown about from side to side as if he were in a violent convulsion. The twitchings of chorea may or may not stop with sleep. In those cases in which the movements continue at night, the disease is usually severe, and it is in this type of cases that death sometimes occurs from exhaustion.

The mental state of the patient is one of irritability and peevishness. In adults there may be hallucinations, and even a violent delirium. Some have thought that those cases in which insanity develops, and to which the name *chorea insaniens* is applied, do not belong to ordinary chorea minor. These mental disturbances not rarely complicate the chorea of pregnancy.

Except for the exhaustion of the general system which is produced by the movements, there is no impairment of strength, nor is the electric reaction of the muscles altered. In some cases of a severe type, leading to a fatal issue, hyperpyrexia has been noted, which is probably due to endocarditis. Many years ago I reported a case of monochorea in which the temperature of the affected part was raised. In some instances weakness or even marked paralysis occurs in one or more of the affected limbs, and to this type of cases is applied the term "paralytic chorea."

Complications.—As already stated, chorea is a disease which is associated with a lack of nervous stability. It is manifest, therefore, that it may often be complicated by symptoms of hysteria. Indeed, it may be difficult to determine whether the patient is hysterical or choreic. That endocarditis frequently precedes, or accompanies, or complicates chorea, has also been stated, but every case of chorea that presents a cardiac murmur is not necessarily suffering from endocarditis, since the murmur is not infrequently due to anæmia, or to relaxation of the fibres surrounding the mitral orifice.

Diagnosis.—Ordinary cases of chorea in childhood are easily diagnosed, particularly if the history of the patient is borne in mind. Between the ages of fifteen or twenty-five care must be taken that it is not confused with hysteria. Sometimes, too, choreiform movements develop in those parts which are affected by infantile cerebral palsy, but in such cases paralysis is present and muscular rigidity is noticeable, while the movements are really different (athetosis).

Duration and Prognosis.—Chorea minor usually lasts from two to three months, and sometimes extends over a year. Mild cases may continue for only a few weeks. The prognosis as to recovery is good, the mortality being about 3 per cent., if all cases are included, death being due to complications rather than to the disease itself. Relapses are not infrequent in chorea. Unfavorable symptoms are rapid loss of flesh, fever, and delirium. The prognosis is worse as to duration in adults than in children. In the chorea of pregnancy the prognosis is very much more grave than any other form of the disease, the mortality varying from 20 to 25 per cent. Senile chorea is often a permanent affection and is rarely fatal.

Treatment.—From what has been said in regard to
of the patient who suffers from chorea, it is evident th
quiet are absolutely essential. The child should n
excitement or mental stress of school-work, neither
to punishment or to criticism because of its moveme
the fact that it is suffering from choreic movements sh
the physician is convinced that the case is one of hyst
Iron and arsenic are to be given if anæmia is present,
useful if there is a rheumatic history. If the movem
to exhaust the child, it should be kept in bed and sle
by the use of hypnotics. These drugs, however, m
lest they produce general depression, and it should
that a hot pack will often put a choreic child to sle
permanently arrest the choreic movements. I have
least two instances by the hot pack.

Although there is no specific remedy for chorea, ars
the position of a specific. How it acts is not kno
administer it is in the form of Fowler's solution, starti
times a day for a child of ten, and increasing it a
puffiness about the eyes and nose or gastrointestinal i
the full physiological effect of the drug is present, whe
or cut down to one-half the quantity. If this is
neuritis may develop.

Next to arsenic in value is cimicifuga, given in the
the fluid extract twice or thrice a day. Antipyrin an
been employed with success. Bromides should be
drugs have failed.

If the chorea of pregnancy becomes severe it may
labor.

Other Forms of Chorea.

Huntington's Disease.—Under the name of " h
Huntington's disease, an affection is met with which m
tiated from chorea minor. It is a rare condition which
is hereditary, although it does not always affect co
sometimes passing from the grandparent to the child,
the parent is usually excessively neurotic. Both sexes
The disease begins between the thirtieth and fortiet
instances, and no exciting cause can usually be disco

Its early symptoms consist in *twitchings of the m*
upper extremities, which gradually increase in seve
which they involve, until the entire muscular system
patient, under these circumstances, carries on a series
ticulations, but it is a noteworthy fact that he, or
movements at least for a period long enough to
movement which it is desired to make. When the
and legs are involved the body is tossed hither an

movements. Sensation is not involved. *Paralysis of a hemiplegic type* develops very rarely. The mind gradually fails, the *mental failure* being preceded by depression and irritability.

Huntington's chorea is an incurable disease. It usually ends in the patient becoming bedridden and dying from some intercurrent malady. It may, however, last for many years. Cases are on record in which the patient has lived thirty years after the disease began to manifest itself. The progressive character of the malady, the period of life at which it develops, the progressive dementia, and the history of heredity all aid in separating it from chorea minor.

The morbid anatomy is not understood. In some cases the lesions of the brain have resembled those of paretic dementia.

The treatment consists in a healthy out-door life, and the use of nervous sedatives, and tonics, with the object of maintaining the patient's general health. Cases apparently identical with Huntington's chorea, but occurring singly, are spoken of as chronic chorea.

Dubini's Disease.—Under the name of "electrical chorea," or "Dubini's disease," a form of chorea characterized by severe muscular contractions resembling those produced by electricity has been described by Dubini as affecting peasants in Northern Italy. Occasionally, the movements may be epileptiform in character. Paralysis soon develops; pain is suffered in the head, neck, and back, and death results, as a rule, from exhaustion. A few cases have been reported as having recovered.

Another form of electrical chorea seen in children has been described by Bergeron. Such cases usually recover. I showed such a case before the Neurological Society of Philadelphia many years ago. The patient, a boy, a little past puberty, suffered from violent contractions which were electrical in character.

HYSTERIA.

Definition.—Hysteria is a chronic functional disturbance of the nervous system in which the motor nervous system may manifest its disorder by convulsions, palsies, or contractures, the psychical nervous apparatus by emotional disturbances, and the sensory apparatus by lost, diminished, or increased sensibility. It is manifest, therefore, that the disease involves both the central and peripheral portions of the nervous system, but there can be no doubt that the dominating condition is a psychosis. Hysteria undoubtedly depends upon a condition of disturbed nervous equilibrium.

Etiology.—There can be no doubt that this affection is, to some extent, hereditary; that is, a parent or parents who possess an unstable nervous system naturally transmit a similar condition to their offspring, and in a very large proportion of cases it will be found that the patient is a child of parents who have at various times manifested neurotic or hysterical disorders. Age has a distinct influence upon the disease. Its most frequent period of occurrence is from fifteen to twenty-five years of age in women, although in males it usually appears at from twenty to thirty. Occasionally, however, children suffer from it, particularly before puberty, and sometimes much earlier than this. The condition is met with far more frequently in females than in males, but statistics vary from 40 to 1 to 4 to 1 or even 2 to 1, according to different writers. It is much more common in the very poor and in the rich than in the middle classes, and in the Latin races than in the Anglo-Saxon race. In America it is most frequently met with in the poorer class of Jews, who are often underfed, poorly housed, much confined, and under great nervous excitation and stress. It is much more common in France and Italy than in Germany and England.

If it be true that the underlying cause of hysteria is a lack of nervous control or balance, it is evident that a number of conditions may be considered as direct causes of the malady. In other words, any condition which upsets the nervous balance may provoke the disease. It is, therefore, frequently found that some great grief or intense joy has been productive of the first manifestation of the disease, or, again, that some injury or fright has acted in a similar manner. Great worry in business, or over a love affair, may produce a similar result. None of these causes would profoundly affect a healthy nervous system. All of them are sufficient to disturb the balance of a nervous organization already abnormal.

Pathology.—As already stated, hysteria is a purely functional disease, and the central and peripheral nervous systems show no alterations which can be considred as responsible for the malady.

Symptoms.—Hysterical individuals usually present evidences of *nervous irritability* which may manifest itself in great excitement, in *violent anger*, in *undue anxiety*, or in *great mental depression*. All of these manifestations may follow one another with extraordinary rapidity. The patient also manifests distinct *lack of self-control*, both in regard to her emotions and her impulses. At times she may seem utterly incapable of accomplishing anything which ought to be done. At another time she can develop an amount of energy and persistence which is surprising, provided that she conceives it to be her duty or her wish to accomplish such an end. The power of thought is in no way impaired, but judgment is warped and uncertain. Not infrequently the patient has perverse ideas which may seem to amount to delusions, but which do not remain constant as in cases of insanity.

In some instances the first symptoms of the malady are manifested by a hypersensitiveness, so that the girl cries easily, and perhaps laughs more readily and for a longer time than is necessary in the appreciation of a remark which is amusing. Restless sleeping also may be present. As the condition develops, attacks of headache and vomiting may come on, and she may suffer from somnambulism.

When the condition becomes still more severe, so that it amounts to that state which is sometimes called "hysteria major," the disturbances of sensation and motion become intense. In addition to attacks of crying and laughter, the patient may pass into a trance or into a condition of catalepsy. Or, again, the patient may suddenly fall and be seized by a convulsion which is distinctly epileptiform in character. Often, however, the convulsion is more largely tonic than clonic, the hands and fingers being flexed and the forearm flexed on the arm, while the legs and feet are extended and the eyes closed. If the eyelids are lifted, the eyeballs are often found to be fixed in convergence or undergo irregular movements. The pupils are dilated. The surface of the body is more or less anæsthetic. As a rule, the patient does not froth at the mouth as much as in true epilepsy, nor does she bite her tongue as is done in epilepsy. So, too, she rarely hurts herself when in the convulsive seizure. The attack may last from a few minutes to several hours, or even longer than this, and may vary in its intensity from semi-consciousness with slight twitchings of the muscles to apparent total unconsciousness and severe convulsive seizure. The expression of the face is often quite characteristic. In some instances it is remarkably peaceful after the convulsion passes by. In others it is ecstatic or terror-stricken. In very young patients curious guttural and other sounds may be made, and the patient may bark like a dog or mew like a cat.

It is noteworthy that many of these patients are conscious of what is going on around them during the attack, although at the time they may manifest no evidence of this. Not rarely a sharply spoken word of command may bring the attack to a close, or the threat of applying some instrument which is capable of causing pain may do likewise. When the patient returns to consciousness, it may be found that there is loss of power in an arm or leg, or upon one side of the body, with or without loss of sensation. Frequently the so-called hysterogenic zones may be discovered, pressure upon which causes pain and may provoke an hysterical attack, or if pressure on these parts is used during the attack it may arrest it.

The sensory symptoms of hysteria, in distinction from those just described in connection with a critical period, consist in *anæsthesia* in all its forms, particularly analgesia of the cutaneous and mucous surfaces and disturbances in the special senses. The most common form of *cutaneous anæsthesia* is *hemianæsthesia* involving exactly one-half the body. After this, the most common type is segmental anæsthesia, in which an arm, or leg, or part of the face is anæsthetic, the margin of the anæsthetic area being sharply defined, while the disturbance of sensation does not correspond to the distribution of any one nerve trunk. Much more rarely patches of anæsthesia occur in different portions of the body. In these anæsthetic areas the senses of touch and of heat and cold are usually preserved to some slight extent. Occasionally, the affected part has a subnormal temperature. When the anæsthesia is limited to one side it affects the left far more frequently than the right half of the body. If the anæsthesia is of the hemianæsthetic, or segmental, type there is usually more or less complete loss of motor power in the same limb.

The *disturbances of special sense* consist in an anæsthetic condition of the retina whereby the visual field is greatly narrowed, particularly for certain colors and often for those colors which normally have the widest field, and the color sense is disturbed or reversed. These disturbances may or may not complicate those just named. When hemianæsthesia is present, the eye upon the affected side is sometimes partly or even totally blind, not as in organic hemianopic hemianæsthesia. So, too, the acuity of the auditory nerve may be diminished, particularly upon that side of the body which is

most affected. The sense of taste may also be per\
smell may be lost.

Neuralgic pains are not common in hysteria excep
grave anæmia and other common causes of neuralgi
of the body, however, hyperæsthesia of the skin m
hysterogenic zones described. These zones are m
females in the groin, whence the name "ovarian"
the mammary glands, and in males on the scrotum
but they may be found in any part. Paralysis of
is sometimes met with. Usually the internal rectus
times the external rectus. Speech may be impair
adductors of the vocal cords. The development of
"hysterical aphonia," the patient being speechless, o
in a whisper. The onset of this condition is usually

The *paralysis of motion* already referred to often p
of time, and in association with this paralysis it n
other muscles, often those which are antagonistic
paralyzed, suffer from contractures. At first these
abnormally irritable and the contractures may be fle
as long as the paralysis of motion and sensation, an
the contractures which are sometimes met with in cere
may also affect the muscles of an arm, of the face,
tremors may resemble those of paralysis agitans or
terized by tremor, particularly if the patient has bee
a case. The amount of atrophy or wasting which
part is usually very slight, and depends almost entir

Of the internal viscera it may be said that the fu
impaired in most instances, unless perchance these
chief manifestations of the disease. A very commo
present in the majority of cases is the sensation as if
rose into the pharynx. This is called "globus." In
attacks of vomiting develop. In still others, the "rifti
of gas, or of air which has been swallowed, takes
much rumbling in the abdomen. In still other insta
it is sometimes called, "merycismus," occurs; that
gitates food, which has been swallowed, into the mout
At other times intestinal disorders are present. I hav
tumor of the intestine, in a patient who had constantl
was emaciated to the last degree, give rise to the
growth was present. At times these patients have a
ing chalk or other materials not commonly swallowe
times very limpid and free. At other times it is sc
After an acute attack of hysteria it is usually limpid.

Of the circulatory disorders attacks of *tachycar*
uncommon. Sometimes the patient will complain
neighborhood of the heart. This pseudoangina is ch
tion of distention of the heart in distinction from th
which is usually described as if the heart were bei

The peripheral vascular system may also be disturbed. Abnormal flushing or blushing or local anæmia and pallor may be present. At times even œdema may develop. In other instances the part becomes so pallid or slate-like in color that it resembles Raynaud's disease, but true gangrene does not develop. Very rarely, indeed, a sharp febrile movement may take place. Sometimes hysterical patients suffer from attacks of hiccough or of sneezing, or of rapid breathing, cough, or difficulty in swallowing.

Under the name of "hysteroepilepsy," a form of hysteria characterized by violent convulsions closely resembling those of true epilepsy is described by many foreign authors. It is rare in this country. In some instances the convulsive seizure is not epileptoid, but cataleptoid or tonic. The patient may lie in bed with the arms extended, as in ecstasy, or with the hands tightly clinched, as in terror or anger, or the state may resemble simple stupor or even coma. At other times a psychical disturbance is manifested so that the patient develops a delirium. Often several of these processes are combined. At first the patient experiences an aura as in epilepsy. This is followed by the epileptoid form of convulsion, and this again by the contortions and emotional attitudes just described. An emotional confusion is not rare; hallucinations and delusions may occur and confinement in an asylum may be necessary. Finally a stage of delirium may develop.

Diagnosis.—Under some circumstances there is no more difficult diagnosis than the differentiation of hysteria from organic nervous disease, particularly if the patient has had an opportunity of studying the symptoms presented by patients with organic nervous lesions. The important points in differentiation are the contraction of the visual fields and the alterations in the color fields, the fact that the areas of anæsthesia are not confined to any given distribution of sensory nerves, the presence of hysterogenic or hyperæsthetic spots, the fact that wasting does not develop to any degree in the paralyzed muscles, the absence of the reaction of degeneration, the maintenance or persistence of the deep reflexes, and the peculiar emotional state. Again, the anæsthetic area may be moved from the first place affected by the mere placing of a coin or a magnet over the affected part. Hysterical contractures also usually disappear in sleep or when the patient is under the influence of an anæsthetic. In the epileptoid form of attack the tongue is never bitten nor the limbs injured.

Prognosis.—The prognosis as to life is good. The attacks usually diminish in frequency and severity with advancing years, but the question of prognosis is also governed by the degree of nervous instability in the patient and in her parents. When the hereditary influence is bad and the surroundings of the patient are unfavorable the malady may last a lifetime. In those who are well-to-do and who can afford to take the treatment required by such cases, the outlook is better than in those who are continually exposed to bad surroundings, with nervous stress and strain.

Treatment.—The treatment consists in the removal of the patient from those causes which tend to produce nervous irritability and stress. If the home surroundings of the patient are such as to increase nervous irritation, the patient must be removed from those surroundings. Such a patient should always be taken from school and given lessons under a private instruc-

tor. If the symptoms are severe the Weir-Mitche
If they are not severe enough for this, an out-do
amount of healthy exercise carried to the point of fat
tion, is needful. If anæmia is present it must be ov
Insomnia and peripheral nervous irritation should
cold packs and the various forms of hydrotherapy. S
but it must be remembered that the administration
patients frequently produces a drug habit. Local
to being treated by hydrotherapy, will also be ben
rapidly interrupted faradic current administered b
wet sponge, or, if the anæsthesia is marked, throu
The physician must exercise a dominant influence o
must be put under the charge of a trained nurse of s
the one hand, will not be irritated by peculiaritie
courage them. This mental side of the treat
overlooked.

The treatment of the hysterical attack itself consi
of nitrite of amyl. There are few cases of true hys
once relaxed if convulsed, and the attack stopped
the one hand, is perfectly safe, and, on the other,
mental impression that its use is appreciated by th
amyl cannot be employed, ether may be used and p
form is usually too agreeable to the patient to be ad
the use of the dry electric brush or even of the act
be touched at various points on either side of the
through the powerful mental impression it produces

EPILEPSY.

Definition.—Epilepsy is a disease characterized by
ness, which, in the well-developed form of the mala
followed by convulsions. The convulsions at the
usually tetanic or tonic in type, but almost imm
Indeed, so typical of epilepsy are clonic convulsio
of this class are called "epileptiform." Epilepsy se
convulsive conditions associated with unconsciousne
a chronic malady, whereas epileptiform convulsi
causes occur but a few times in the lifetime of the indi
in puerperal eclampsia or uræmic poisoning. Hyst
ever, closely resemble it.

Etiology.—The etiology of epilepsy is unknown,
proportion of cases injuries to the brain substance
internal causes undoubtedly predispose to or produc
instances it has been thought that the condition is he
tainly true in the sense that epileptic parents often
By far the largest number of cases collected by a
the author is aware, are those of Gowers, who anal

cases of epilepsy, finding that an inherited tendency was indicated by the presence of insanity or epilepsy in ancestors or collateral relations in rather more than one-third of the cases (35 per cent.), and rather less frequently in males than in females, for there was this history in 33 per cent. of the males and 37 per cent. of the females. There was a family history of epilepsy in two-thirds of the inherited cases, of insanity in one-third, and of both disorders in one-tenth of the cases. In the 56 cases recorded by Sieveking heredity was the cause in 11. Reynolds, in his collection of cases, found the proportion to be 31 per cent. Hasse has collected 1000 cases, and has found heredity the cause in no less numbers than the others. If we take the average result of the conclusions reached by the clinicians just named, who give exact figures, we find that we have to deal with 4300 cases of epilepsy, of which a little over 26 per cent. were due to heredity. Whether epilepsy can be induced in a child by hereditary influences arising from chronic alcoholism or chronic lead poisoning is open to debate. Certain neurologists are firmly convinced that these factors are active.

In other instances, apparently healthy children develop epilepsy after suffering from some of the infectious diseases such as scarlet fever. In these cases the infection has either produced some definite lesion in the brain or has so impaired the normal growth of certain cells in the cerebral cortex that their natural balance is destroyed, with the result that periodic explosions of nervous energy take place.

Syphilis acts as a cause of epilepsy in two ways. When the parent is syphilitic, the child may suffer from hereditary syphilis or from a parasyphilitic disease of the nervous system, with imperfect cerebral development. In other cases acquired syphilis produces epilepsy in adults. Indeed, more than one writer has expressed the belief that an epilepsy beginning after the twenty-fifth year is syphilitic in origin. This is rather an exaggerated statement, but it is nevertheless true that more than three-fourths of all cases of epilepsy begin before the twentieth year. About half of them begin in the second decade of life. Quite a large proportion begin between the seventh and tenth years.

In some instances the epilepsy dates from the reception of some severe injury to the head. Cases that have a traumatic origin, those which are due to syphilitic gumma or other form of brain tumor, usually belong to that type which is called "Jacksonian" or "localized" epilepsy, although they may ultimately develop all the characteristics of the so-called idiopathic form.

For many years it was considered that a host of conditions tended to produce epilepsy by reflex irritation. Such causes as foreign bodies in the ear or in the nose, intestinal worms, and uterine disorders have been considered as causative factors, but in all such cases it cannot be denied that these agents act indirectly in the sense that they provoke an irritation which reflexly unsets the nerve balance or equilibrium of an unstable motor area of the brain. In other words, in any case of epilepsy it is to be understood that the underlying factor is a lack of stability or nervous balance.

The influence of sex is not very great, but males are affected somewhat more frequently than females. Althaus has examined an enormous

amount of statistics to obtain results bearing on this
in 54,442 cases there were 28,960 males and 25,482

Posthemiplegic epilepsy due to cerebral injury ma
there can be no doubt that it far more commonly o
adults. In at least two-thirds of the cases the onset
of age, and in nearly one-half it is during the first tw
uncommon for the paralysis to occur in infancy an
at puberty. This prolonged interval is rare in adult
seizures usually begin in less than one year.

The frequency with which epilepsy comes on a
childhood has been very exhaustively studied.
20 children out of 97 suffered from it. In the
Gaudard 11 children had hemiepilepsy, and 66 chi
collected by Wallenburg were epileptic after hemiple
of cases collected by Osler 15 children out of 23 we

Pathology.—The pathology of epilepsy is not kno
autopsy many cases of epilepsy show atrophic or dege
cerebrum, but this holds true of only a certain prop
instances of so-called idiopathic epilepsy in which
syphilis, or of injury, or of damage to the brain throu
vessels. In this idiopathic form the most careful m
scopic examinations of hundreds of cases have failed
which can be considered as responsible for the malad
scopic lesions which have been described by certain
out doubt present, but in these cases the question a
not the result rather than the cause of the affection.

That epilepsy is a result of an explosive discharge
the motor areas of the cortex is proved by the fact t
can be produced in man and in the lower animals b
and that growths and injuries which irritate them pro
The somewhat ancient theory that the convulsive di
of lesions in the medulla and the pons is no longer a

Symptoms.—One of the first and most marked sym
attack of epilepsy is a peculiar sensation felt in som
generally below the head, which gradually rises up o
rapidly or slowly, like an oncoming cloud, until, the hea
the patient is immediately convulsed and unconsciou
is seen to be in the very acme of the nervous storm. S
arrival of this *aura* in the cervical region the person
scream, so characteristic that it has been called the
probably due not so much to a voluntary impulse as
of the air from the thorax by the convulsive contra
muscles, as well as those of the thorax, and its rapi
glottis narrowed by rigid spasm of the muscles g
Synchronously with this cry the *muscles of the whole*
attack, *become strongly contracted* until they are in a
having momentarily relaxed, pass into *alternating r*
tions, which throw the patient now to this side, now

During the tonic spasm the muscles of the face often produce marked distortions of the features, in some cases bringing about the so-called *risus sardonicus;* the head may be drawn to one side, and under these circumstances the eyes are generally turned in the same direction; the jaws are locked one against the other, and the lower jaw may also be drawn away from the median line of the face in the same direction as the eyeballs. Sometimes the whole body is rotated. In 970 cases analyzed by the writer, complete rotation to the right is mentioned as being present in 49 persons, and to the left in 52 cases. There is, therefore, no difference worthy of note in these numbers.

The arms are strongly flexed at the elbows, while the hand is still more strongly flexed at the wrist; the fingers are also so bent into the palm of the hand that not unfrequently the skin in this region is found indented by the nails. The arms, legs, and body are drawn and jerked in the direction of the most powerful muscles, and, as a consequence of this, opisthotonos, during the tonic stage, is by no means uncommon. Exceptions to this rule do, however, frequently occur, and when present show that the paroxysm is exerting its chief influence on the weaker muscles, while the stronger ones are affected at least to a less degree. As a general rule, too, the muscles of one side suffer more than those of the other. Unfortunately, in the cases collected by me, in only 158 instances were any remarks on this point made. In these 158 the right side was most affected in 77 cases, and the left side in 81 cases. It is evident, therefore, that both sides suffer about equally. The legs may be firmly flexed on the abdomen, while the fingers are rigidly extended.

The change in the color of the face is very marked and almost typical of the disease, being at first pale, then flushed, the flushing deepening often into a livid purple, owing to the asphyxia produced by the convulsive contraction of the thorax. In some cases the eyelids are widely drawn apart so that the eyes, owing to their fixation, have a staring appearance; in others they are so tightly closed that the fingers of the onlooker can scarcely force the lids apart. The staring, but blank, expression of the eyes is also increased by the slow dilatation of the pupils which always accompanies the asphyxia.

The duration of the tonic contractions rarely exceeds two minutes, and in most cases is limited to but a few seconds. It is followed by the clonic spasms, already described, which are ushered in by more or less violent tossings, but whose onset is forewarned by peculiar vibratory thrills, which run through all the affected muscles. The eyelids tremble, the body changes its position never so slightly, and then, as if the vibrations gained greater and greater power with each moment, the fibrillary tremors give way to muscular contractions. The expression of the face, which in the preceding stage was set and firm, is now constantly changed by the movements of the facial muscles; the jaws, no longer locked together, are gnashed and crunched one upon the other; the tongue is alternately protruded and drawn back, and, as a consequence, is often caught between the teeth and bitten and lacerated. The excessive movements of the muscles of mastication force the increased quantities of liquid secreted by the salivary glands from the mouth in the form of froth, which is often stained with blood by reason of the injuries to

the tongue. The constancy of the convulsive mov
and less marked; well-developed remissions occur
body, until the movements cease entirely; but it sh
in mind that the prolongation of the remissions does
in the severity of the intervening spasm, the final
more violent than the first.

The intense discoloration of the face begins to
remissions, by their length, permit the blood to
pearance being temporarily arrested by each parox
having ceased, the patient lies before us relaxed, un
and passes into a deep sleep or coma, which lasts
and from which he cannot be aroused except ve
great difficulty.

One of the most interesting and important of
so-called aura. Difference of opinion has arisen
occurrence, some authors stating it to be very rar
constantly. There can be little doubt that in man
present as in others it is absent, and it would app
the patient has something to do with the occurr
attack; at least, if we may judge by the statemen
each nation. Thus, in America, Wood states that
very large proportion of the cases of true epilepsy
with him. In England, Gowers stated it to occur
cases, and Bristowe states it to be not uncommon.
the aura appears to be present in more than half
another, as it is also in Germany, according to
collected by the writer it was found that the aura w
362 cases and absent in 138 cases.

The *aura*, or *warning*, while possessing genera
to all cases, is by no means identical in each indiv
number of cases, where it is present, have it in
not there, then it is often in the stomach; and it
persons suffering from epilepsy who have as an a
sensation all over the body. In much more rar
situated in the organs of special sense, and are evi
of blindness or deafness. It is worthy of note, h
aura may differ in every case in origin, seat, and li
ably constant in the same individual, rarely, if ever,
they may vary in degree. A careful analysis of
cases by hundreds of observers shows that the a
with is that beginning in the hand; next, that beg
next most common, that arising in some of the visc
which arise in the face and tongue. The rarest f
arises in the sides of the trunk.

Not only may the seat of the aura be varied,
even more aberrant. Undoubtedly the most comm
scribable sensation of a vapor or cloud, already s
number of cases the sensations are described as

perhaps as partaking of the feeling that the part is in active movement when in reality it is still quiet. Others speak of it as a sensation of cold, others of heating and burning, and still others of trembling and indescribable distress. In certain cases the sensation is confined to the spot where it is first noticed, and fails to travel upward or toward the central nervous system.

Status epilepticus is a condition in which convulsion follows convulsion so rapidly that consciousness is not regained. In some instances the patient dies within a few hours as a result of exhaustion or asphyxia. As the case goes on the convulsions are replaced entirely by coma, or, in rare cases, violent attacks of mania may develop. In this state the body rapidly emaciates, bed-sores develop, and death ensues from exhaustion.

An extraordinary number of fits may occur in a brief space of time without causing death, or even very great exhaustion. A very good example of this fact is that of a case reported by Newington, which is as follows: On the twentieth day of the month, at 5 A.M., the fits began in the woman under his care. By 5 P.M. the same day she had had 274 fits, and by 5 A.M. on the 21st she had 384 more, or 622 fits in twenty-four hours. This makes a rate of one nearly every minute. By 5 A.M. on the 22d she had 400 more; by 5 A.M. on the 23d, 525; by 5 A.M. on the 24th, 355, and from 5 A.M. on this day to 5 A.M. on the 25th she had 214 fits. Altogether she had 2156 fits in five days, and yet survived, being fed by the rectum.

Motor paralysis may succeed epileptic paroxysms, and this is particularly the case in those instances where the convulsive movements are largely unilateral in character.

A very important question, connected not only with the prognosis of epilepsy, but also with its relation to medical jurisprudence, lies in the influence which the disease may exercise on the mental condition of the sufferer. Russell Reynolds has arrived at the following conclusions in regard to the effects of the disease on the intellect:

1. That epilepsy does not necessarily involve any mental change.
2. That great mental impairment exists in some cases, but this is the exception rather than the rule.
3. That females suffer (in mental vigor) more frequently than males, and also more severely.
4. That the commonest failure is loss of memory, and that this, if regarded in all degrees, is more frequent than integrity of that faculty.
5. That apprehension is more frequently preserved than lost.
6. That ulterior mental changes are rare.
7. That depression of spirits is common in males, rare in females, but that excitability of temper is found in both sexes.

Complications.—Naturally enough, a very common variety of complication is some traumatism, severe or mild, which is suffered as the result of the fall accompanying the fit, whereby the head is struck against some hard or sharp object. The severity of the injury may be anything from fracture to a slight abrasion or bruise. When such an accident happens it should not be forgotten that the coma of the fit may be dangerously deepened by the concussion, and also that the coma may mislead the physician so that it is regarded as the natural sequence of the attack rath injury. Fractures of the clavicle are very common various dislocations may ensue. The presence of a is a very much more serious matter than would ap even if the fits are not very frequent they are almo solution of continuity, or even to convert a simple in by the jerkings of the muscles. Splints are, of co limb may be wrapped in a pillow. Careful watchin must always be insisted upon, since under these fall is avoided on the advent of a new attack.

In other cases apoplexy may occur, due to the cerebral bloodvessels during the fit, and if the co is prolonged or peculiar, this fact should be called t of the pupils, the stertorous respiration, the fact th protruded straight from the mouth, all point to a cer of temperature, the coma, and, last of all, the hemi of both states, and cannot be used for differential d

Diagnosis.—Undoubtedly, the most similar conv have is that due to hysteria, and the diagnosis of as difficult in some cases as it is essential and nece cure. The other conditions, with which it might b alcoholic epilepsy, tetanus, and syncope. In the a arranged all these disorders, which briefly and succi points between them, although of necessity it is account of the lack of space.

The very irregularity of true epilepsy makes i give clear and well-defined outlines of it agains ticularly when we remember that epilepsy and hy hand.

By far the most important differential point bet just named, when not complicated with still another of the movements. As already pointed out, in epi at variance with those of daily life, while in hysteri typical of ordinary muscular contractions, or, in purposive in character; and frequently there is prol of the muscles, giving rise to the assumption of positi less resemblance to normal attitudes. In hysteria impaired sometimes, but never so completely as in most commonly the individual knows all that goes o she may give no sign of consciousness by words or l she may afterward be able to narrate all that has occ however, a condition known as automatic conscio during the paroxysm, the patient understands all t everything on the return to quietness.

The fact that the patient is a female cannot be evidence of hysteria in the least, but if the fit occu taken as fairly positive evidence of epilepsy; and y remembered that males may suffer from hysteroid

TABLE OF DIFFERENTIAL DIAGNOSIS OF EPILEPSY FROM HYSTERIA, ETC.

Signs.	Epilepsy.	Hysteria.	Uræmia.	Petit mal.	Alcoholic epilepsy.	Tetanus.	Syncope.
Apparent cause.	None.	Emotion.	None.	None.	None.	None.	Mental shock.
Aura or prodromata.	Generally present, but short.	Globus hystericus, palpitation, choking.	Headache, vomiting, and dyspepsia.	Faintness and dimness of vision.	Tremors.	Nervousness.	Not so well defined as in epilepsy.
Onset.	Sudden.	Often gradual.	Often gradual.	Sudden.	Sudden or gradual.	Gradual, begins in jaw.	Sudden or gradual.
Scream.	At onset and sudden.	During attack.	Frequently none.	Frequently none.	May or may not be present.	None.	None.
Convulsion.	First tonic, then clonic.	Rigidity more pronounced with more aching.	Rigidity generally absent.	No rigidity.	Movements more clonic than tonic.	Always tonic.	None.
Biting.	Tongue.	Tongue, lips, and hands.	Tongue.	None.	Rarely.	None.	None.
Micturition.	Frequent.	Never.	Never.	Rarely, except when bladder is affected.	Rarely.	Sometimes	Never.
Defecation	Occasionally.	Never.	Never.	Never.	Rarely.	Rarely.	Never.
Talking.	Never.	Frequent.	Muttering.	Never.	Never.	Never.	None.
Duration.	A few minutes.	Generally many minutes.	From a minute to hours.	Momentary.	May be prolonged.	Hours.	Indefinite time.
Consciousness.	Lost.	Generally preserved.	Lost.	Not lost always, but clouded.	Lost.	Preserved.	Lost.
Termination.	Spontaneous.	May be induced by shock.	Spontaneous	Spontaneous	Spontaneous	Spontaneous.	Gradual, with no somnolence.

The movements of the hysterical patient after the tonic condition has passed away are as clonic as those of epilepsy, but still possess some purposive characteristics, and are not so bizarre as are those of the true disease. Thus, the head, arms, and legs are struck with evident endeavor against the floor or surrounding furniture. Another point, which, when it occurs, is very distinctive, is the onset, toward the close of a hysterical convulsion of a second stage of tonic spasm such as occurred at the beginning. It will be remembered that this does not occur in epilepsy; although it must be borne in mind that in cases of the "status epilepticus" the rapid onset of another attack may show a second tonic stage. This can be separated, however, by the fact that it is followed by clonic movements, whereas the secondary tonic stage of hysteria is usually followed by relaxation and temporary recovery.

Finally, too, in hysteria, some peculiar emotional position is often assumed,

as of the crucifix, or of intense grief, or, perhaps, in
corresponding movements of the trunk. If the pat
a smile may float across the face, while the eyes,
pain, or entreaty, may seem to be gazing at some
some very well developed cases the expression of
look of pain, with painful movements, or an a
entreaty, with sensual and venereal desire evide
commonly areas of anæsthesia and hyperæsthesi
and are of all degrees of intensity and limitation
erally shows their presence after attacks of convulsi
from one attack to the other, or develop spontaneo
these areas are unilateral, and may extend over
line of demarcation of the anæsthesia or hypera
area, being clearly and abruptly defined, generally
front and back of the trunk. It will be called to
are absent in true epilepsy. Hallucinations are
the fit in hysteria than in epilepsy, and sometime
the attacks.

A very useful differential point, strongly insiste
Bourneville, is that in true epilepsy there is gene
rise of temperature during an attack, while in hyst
ture remains normal or only slightly raised. Not r
attacks of epilepsy, and very serious injuries are
by these persons to carry out their designs. The
are: the condition of the pupils, which, in the simul
normally; nor can the corneal reflexes be held bac
rarely changed, and the thumbs are rarely flexed
has pointed out that in malingerers the bystand
out the thumbs, and that they remain so; whereas
become flexed again.

Suggestions as to movements are sometimes f
and the movements generally lack the bizarre chara
If tobacco smoke or ammonia be held to the no
ally is forced to disclose his true condition. The
there is no rise of temperature is a differential point

Prognosis.—The physician can always assure the
so far as the disease itself in its ordinary form is
danger of death, since, as a general rule, unless th
death rarely occurs, unless indirectly by the fall o
or well, or when in some position where a steady he
Accidental asphyxia, due to the burying of the fa
or to the impaction of food in the larynx, may occ
is uncommon.

The question which the friends will always ask
of ultimate recovery, or, at the least, will there be
improvement? Unfortunately, the reply ought not
able, even for ultimate improvement, for the experi
practitioner has been that cures rarely occur.

Jacksonian Epilepsy.—By the term Jacksonian epilepsy we mean an affection which separates itself from true or ordinary idiopathic epilepsy by several peculiarities. By far the most important of the peculiar signs is the character of the onset, which always begins, in the typical Jacksonian disease, in some peripheral portion of the body, and most frequently in the muscles of the thumb or hand, so that for the moment the movements are localized and may remain localized at the point of origin, or immediately diffuse themselves over muscle after muscle until all the arm, leg, or other groups of muscles are involved. It is of the greatest importance, however, that the reader should keep the aura of an attack separated in his mind from the onset, remembering that the term onset is here used by the writer to designate the beginning of the period following the aura, if there be one. Jacksonian epilepsy may be of almost any degree of severity, for in rare cases but one muscle may suffer throughout an entire attack, or in others the entire body may be finally convulsed. There may or may not be loss of consciousness, its presence or absence being dependent upon the severity of the attack. In those instances in which only a few localized muscles are involved, consciousness is more commonly preserved than lost.

Petit Mal or Minor Epilepsy.—Petit mal differs in no way in its essential characters from epilepsy of a much more highly developed form, but in its minor characteristics it is sufficiently at variance with *haut mal*, or *grand mal*, to separate it in the minds of clinicians. In its most common form petit mal consists of a momentary loss of consciousness, accompanied by pallor, or, more rarely, flushing of the face. The man who is subject to the disease suddenly stops what he is doing for a moment or two, and then takes up his work or subject as soon as he recovers, and at the point where he ceased, being often unconscious of the break in his conversation or labor. Reynolds has divided this minor form of the affection into two divisions. In the first he places those who are attacked and have no evident spasm, and in the second group are those who have evident spasm. The seizures are characteristically fugacious, and if any spasm is present it is nearly, always of the tonic variety. Sometimes the disorder of motility lies chiefly in an inhibition of an act about to be performed. The fork in a man's hand at a dinner-table may be raised half-way to the mouth, then held in mid-air for a moment, and then, as the attack passes away, continue on its journey to the mouth; or, a woman playing the piano may suddenly pause with her fingers raised from the keys, miss the time of three or four bars, and then go on exactly where she left off, as if no interruption had occurred.

Treatment.—By far the most valuable drug in use to-day for the relief of epilepsy is bromide of strontium. In many cases the remedy undoubtedly gives relief when it is pushed in a suitable manner, and, in the majority of instances, the seizures are so decreased both in violence and frequency that its use may be said to be indicated in nearly every case of the disease. In a very small minority, however, it signally fails.

A very important point to be borne in mind is that the drug often seems to have produced a complete cure, and this results in carelessness in the

regularity of administration. The patient should b
that every day passed without a fit is a step forward,
him many steps backward. He should also be m
moderation for at least three years after all fits hav
after that time, for the slightest sign of their retur
each day should be gradually decreased, not sudder

The iodide of potassium is entirely useless in epil
syphilis, when it is of the greatest service. Undei
mides and all other drugs should be set aside wh
utmost. As is well known, syphilitics usually bear
and the writer knows of one instance where no les
taken every twenty-four hours, with rapid improvei

When the convulsions are due to a gumma the
however, too slow in its action, and should be repla
to break down the growth without delay, lest a se
asphyxia or some similar accident.

In every case the physician should make carefu
ence of an aura, and, if it is present, he should oı
provided with pearls of amyl nitrite, one of which
contents inhaled the moment the warning of an a
By this means attacks can often be abortive.

ECLAMPSIA.

The term "eclampsia" is applied to convulsion
pregnant women, or women who have just been de

Infantile Eclampsia.—In *infantile eclampsia* the
in character and seem to depend upon a condition
the nervous system, which is still further disturbe
Thus, it is commonly supposed that gastric and in
produce infantile eclampsia, and certainly the pr
in the stomach and intestines may act in this
physicians believe that the first dentition, by reasoı
gums, may result in such a seizure. A host of ot
irritation have also been held responsible. Not rar
is rickets. It is often stated, in text-books on m
infectious fevers are frequently initiated by a co
matter of fact this rarely occurs in an ordinary chi
manner.

The convulsive attack varies in severity from
fingers and the drawing of the thumb into the palm
clonic or tonic convulsion closely resembling epile
In many instances the child has a single attack and
several attacks occur within a few days. In still
one or more attacks of convulsions seems to develo
in these instances the child may become a confir
cases, however, it is probable that the condition

direct cause, simply induces a nervous explosion on the part of a brain, which has an impaired stability.

Diagnosis.—The condition must be separated from the convulsions produced by organic cerebral disease (which see).

Prognosis.—The prognosis in infantile eclampsia is good for single attacks, and becomes grave in direct proportion to their severity and repetition. Such attacks occurring in feeble, poorly nourished children are more grave than in those who are better able to withstand an illness.

Treatment.—This consists in removing the cause of local irritation, if it can be found. If it exists in the stomach or the bowels, it should be removed by an emetic or a purge. If the gums are inflamed they should be lanced. If rickets is the cause it must be cured if possible.

The treatment of the attack itself consists in the administration by the mouth, if swallowing is possible, of 5 or 10 grains of bromide with from 2 to 4 grains of chloral, or by the use of 20 grains of sodium bromide with 5 grains of choral in starch-water, by the rectum. If laryngeal spasm is marked, and is a dangerous symptom an inhalation of nitrite of amyl may be used, or chloroform may be employed if the heart is not weak.

Puerperal Eclampsia usually occurs in young primiparæ. The convulsions are tonic and clonic. The pathology of the condition is not understood. Without doubt the condition is toxic. In some instances it is probably due to perverted functional activity, or actual disease, of the kidneys. In other instances it seems to be dependent upon perverted metabolism. Not infrequently, in association with the albuminuria of pregnancy, there is *albuminuric retinitis,* and even blindness with general anasarca. That the presence of *fœtus in utero* exercises some influence is shown by the fact that not infrequently the convulsions cease as soon as the uterus is emptied.

Puerperal eclampsia is an exceedingly dangerous condition. The mortality varies from 20 to 30 per cent., or even more than this. In a certain proportion of cases it can be prevented, and for this reason the physician should repeatedly examine the urine of the pregnant woman for several months before the termination of pregnancy to determine that the kidneys are carrying out their eliminative function properly.

Treatment.—The uterus must be emptied, the poisons must be eliminated, and the nervous system must be quieted. For the best method of emptying the uterus, the reader is referred to books upon obstetrics. If arterial tension is high and there is much cyanosis, the patient should be freely bled and the intravenous injection of normal saline solution employed, unless there is a tendency to pulmonary œdema, when the intravenous injections should not be used. Copious irrigation of the large bowel or the injection of an ounce of magnesium sulphate dissolved in a half-pint of water and 2 ounces of glycerin are also useful. If the convulsions are severe in these cases many physicians treat the condition by the use of large doses of 20 to 30 minims of the tincture, or even of the fluid extract, of veratrum viride, giving it in some cases hypodermically. This drug lowers arterial tension, quiets the spinal cord, and produces sweating. Pilocarpine is never to be employed, as it almost invariably causes pulmonary œdema.

TETANY.

Tetany is a condition in which intermittent unilateral or bilateral tonic and painful spasm affects certain muscle groups, usually of the upper limbs, although occasionally it involves the legs as well. It is sometimes called "tetanilla," or "idiopathic muscular spasm." The disease is exceedingly rare in America, but comparatively common in certain European countries, notably Sweden and Austria. It is probable that tetany is merely a symptom of several different conditions. One type of it occurs in epidemic form in Austria, particularly during the months of March and April, affecting chiefly youths between fifteen and twenty-five years of age. These persons usually belong to the lower walks of life. Occasionally it develops in women, particularly at the time of pregnancy or during nursing. A very few cases have been reported in children below puberty and in persons of advanced years.

Etiology.—In the opinion of many persons tetany is due to autointoxication or to intoxication due to some infectious agent. Thus, it has been frequently reported as occurring in persons who are suffering from chronic gastrointestinal disorders, particularly cases of gastric dilatation, and it has occurred in such cases after the gastric contents have been removed by lavage. It seems to be more frequent in persons who follow certain occupations than in others. Thus, out of 314 male patients mentioned by Frankl-Hochwart no less than 141 were shoemakers and 41 were tailors. This has caused certain persons to believe that certain types of tetany were of the nature of an occupation neurosis. Tetany sometimes develops in those who have suffered partial or total extirpation of the thyroid gland. It is also met with in children who are suffering from rickets. In some cases tetany is a manifestation of hysteria.

Pathology and Morbid Anatomy.—As the condition is a functional one, and as few of these cases come to autopsy, we know little concerning their morbid anatomy. In a few instances autopsy has revealed hyperæmia and minute hemorrhages in the anterior cornua of the spinal cord, but it is very doubtful if these are characteristic of the malady.

Symptoms.—The prodromal symptoms of tetany are usually those indicative of a toxæmia. The patient first suffers from some *aching* or *pain in the extremities*, and may have *headache* and *dizziness*, and feel heavy and stupid. As already stated, the disorder usually affects one or both arms and involves in particular the muscles of the forearms and hand, causing the palm of the *hand to be flexed upon the wrist* while the fingers are extended. Sometimes the *forearm is flexed* at the elbow. In other instances the phalanges are flexed and the distal phalanges extended. When the lower extremities are affected, the feet and toes show somewhat similar contractures. The *toes may overlap one another* and be forcibly flexed, and the foot may be bent at the ankle in the position of clubfoot. Occasionally, in very severe cases, some of the muscles of the trunk and those of the neck and throat may be involved, and even the ocular muscles may contract, so that a form of nystagmus is present. It is a noteworthy fact, first enunciated by Trousseau, that pressure exercised upon the affected limb will generate an attack, provided that the nerve trunks or the bloodvessels are affected by the pressure. This is known as "Trousseau's symptom." The pressure must be continued from thirty seconds to five minutes to produce an effect. While the presence of Trousseau's symptom is pathognomonic of tetany, its absence does not disprove the presence of the disease.

Under the name of "Trousseau's sign" tapping of the nerves of the arms, or legs, when surrounded by an elastic band, may induce the spasm. Spasm may also be induced if the facial nerve is irritated in this manner (Chvostek's sign). Under the name of "Erb's sign" lies the fact that the motor nerves manifest a marked increase in electrical irritability, particularly with the galvanic current. Hoffmann has pointed out that the superficial sensory nerves are also exceedingly sensitive, and that moderate pressure upon them, which ordinarily would not be felt, may cause a severe pain similar to that produced by striking the ulnar nerve at the elbow ("Hoffmann's sign"). If the irritation of the motor nerve is repeatedly produced, a marked increase in the excitability of the tributary muscles follows. Occasionally, nervous lesions appear in the skin such as urticaria or herpes, pigmentation, and loss of the hair and nails. An attack of tetany may last from a few minutes to several days. It may be so moderate that it can be overcome by the will of the patient, or so severe that the limb is entirely beyond control. If an attempt is made to reduce the spasm by force it causes great pain, and if the contractions of the muscles are marked and cramp-like the pain is also severe. The attack passes off gradually and is often followed by impaired sensation and loss of power in the affected parts. There is no loss of consciousness in the great majority of cases.

Diagnosis.—The development of comparatively localized tonic spasms in association with the other symptoms already described renders the diagnosis of tetany quite easy. The disease must be separated from Jacksonian epilepsy and hysteria. This can be done by the development of Trousseau's, Chvostek's, and the other signs just named, by the absence in tetany of the various stigmata, including the reversal of the color fields, found in hysteria. It is differentiated from Jacksonian epilepsy by the prolonged character of the attack and the fact that it can be produced at the will of the physician. Hysterical contractures sometimes assume the form of tetany, and cases of apparently true tetany may have hysterical features.

Prognosis.—The prognosis as to life is good unless the provoking cause is in itself serious, as, for example, when the thyroid gland has been removed. In other words, in no instance does tetany itself threaten vitality, although the underlying cause of the tetany may. Most cases recover. Some suffer from only one attack. In others the symptoms disappear after many attacks as soon as the cause is removed.

Treatment.—This deals largely with the removal of the exciting cause. If gastric dilatation is present and if its nature is such that it can be benefited by lavage or operation, these measures must be instituted. In some instances where there is reason to believe that the condition results from autointoxication, mild saline purgatives, diuretics, and moderate doses of calomel or blue mass are advisable, and, in addition, hot packs may be given to aid in the elimination of poisons by the skin and to act as nervous sedatives. When disease of the thyroid gland is present, the administration of thyroid extract is indicated. If there is present a general condition of debility, anæmia, iron, arsenic, and similar tonics combined with an out-door life and avoidance of nerve irritation are essential.

LATAH.

Latah is a state very closely allied to the saltatory spasm described by Bamberger, and the patients described by Beard as "jumpers." The chief symptom of latah is involuntary and uncontrollable mimicry by the patient of everything she sees or hears. There is also frequent coprolalia or the spasmodic ejaculation of filthy words. The disease is common among the Malay races, and its geographical distribution corresponds with the countries inhabited by these people. It occurs commonly enough among the Filipinos and is known by the Tagalogs as "mali-mali." It is seen in Ceylon and Burmah, and the disease known in Siberia as "myriachit" is probably identical with it. Kraepelin allies latah with hysteria.

The subjects of latah are almost invariably women in early adult life. Men rarely, if ever, suffer from the disease. There is a distinct hereditary tendency, but the cases show no evidence either of hysteria or epilepsy. It is a very common spectacle indeed, in Malay villages, to see one of these unfortunate women pursued by a crowd of tormenting boys. They dance in front of her, going through all sorts of grotesque and obscene movements, and the unfortunate victim, apparently struggling to the utmost to resist the impulse, exactly imitates all their actions to her own great rage and mortification. Besides such examples of complete echochinesia, or mimicry of motion, there frequently is echolalia, or mimicry of speech. When startled or frightened these patients utter irrelevant words or incoherent noises and make involuntary movements. Consciousness is never lost during these attacks. This latter type closely resembles the "jumping Frenchmen" of Maine and Canada, who jump violently and suddenly with a loud cry when startled or when under strong emotion. Jumpers, and latah patients as well, will frequently obey any sharp, sudden command given them. Undoubtedly, this represents some form of psychic suggestion acting on a weak and unstable will. The Malay is notoriously unstable in his mental makeup, and the patients are markedly neurotic. They are pusillanimous and easily startled. As a rule, both the superficial and deep reflexes are increased. Many of these patients suffer later from serious mental disorders. Among the Philippine natives "mali-mali" patients are believed to be particularly prone to the outbreaks of maniacal furor known as amok.

AMOK (RUNNING AMOK).

This term is used, in Malayan countries, to designate cases of maniacal furor in which a native rushes out in the streets of his village with kris or barong, cutting down every one in his path, until he himself is dispatched or commits suicide. It is a question whether these outbreaks should be considered as evidences of a specific disease. Preceding the attack the patient is in a stupid, morose, or melancholic condition for several days. During this period there frequently is amnesia, and during the attack itself complete amnesia is the rule. The exciting causes of the outbreak are usually psychical; grievance over some real or fancied wrong, over financial losses, marital difficulties, fear of disgrace or punishment, and the sight or smell of blood. The disease almost always attacks young adult males. Various causes have been advanced for this condition. Alcoholism may be excluded, as the Malay, although not a total abstainer, is very frugal in the use of liquors. So, too, opium smoking cannot be considered the cause of the disease. Bevan Lewis believes it to be a psychical epilepsy, and, indeed, transitory furor very much resembling the attacks of amok are frequently seen in epileptics. Earlier travellers and writers ascribed these attacks to religious mania. Schuebe discredits this idea on the ground that the Koran does not justify the killing of unbelievers, and he quotes Ellis to the effect that amok-running occurred among Malays before they were converted to Mohammedanism. The Malayan races chiefly subject to amok are the Bugis, Illanums, and the Sulus, or Joloanos, in the Southern Philippines. In many instances among this last tribe the motive is undoubtedly religious. During the service of the United States army in the Philippines a most melancholy case occurred in an officer, corresponding exactly to the typical amok cases. This man, an excellent soldier, and a man of exemplary personal habits, after a few days of brooding and melancholy, suddenly appeared on the veranda of his quarters with a rifle and began to shoot into his company formed up in close proximity. He could not be secured, and after wounding a number of his men he was shot and killed by one of his own sergeants. With regard to the responsibility of these cases no general rule can be laid down. The responsibility cannot be affirmed in all cases, nor can it be denied. Most of the cases of amok are clearly irresponsible. Mention has been made, under Latah, of the occasional outbreaks of furor resembling amok that take place in that disease.

According to Kraepelin, who has studied this condition in Java very recently, amok is not an entity, but embraces a variety of conditions in which sudden, violent, impulsive acts are committed while consciousness is clouded. Some cases belong to the class of the "insanity of adolescence," some are epileptics, a few may be instances of "malarial psychosis," but there are rare cases of amok that Kraepelin cannot explain. Latàh is distinguished from it by the complete preservation of consciousness in that state.

ASTASIA-ABASIA.

Definition.—Astasia-abasia is a symptom of hysteria. It occasionally follows disturbance of the nervous system produced by injury, and in that sense might be considered a traumatic neurosis. Occasionally it has followed the acute infectious diseases.

Symptoms.—The symptoms consist in a *partial or complete inability to use the lower limbs* in standing or in walking, although if the patient lies upon her back in bed she can move her legs perfectly. Ex any alteration from the normal as to motion, co- When the condition simply interferes with walking, Patients who may be quite unable to walk can, neve

The prognosis is as favorable as that of ordi treatment is the same as that which is employ suffering from hysteria or neurasthenia.

NEURASTHENIA.

Definition.—Neurasthenia is a condition in whi suffers from various functional disorders due to ex vous stress and strain whereby the energies of the For this reason it is often called *nervous exhaustion*

Etiology.—The most common cause of neuras longed mental strain produced by business reverses of some important and difficult enterprise. The s by no means always in direct proportion to the s The condition is not met with in the lower classes is largely dependent upon the nervous temperamen the condition of his general health and surroundings tion is commonly met with as a result of excessive winter season devoted to late balls and receptions, o have passed through a long period of nervous st nursing of a sick husband, child, or some near relati only physical exhaustion but mental anxiety to exh is evident, therefore, that many causes may prod vided they result in a great expenditure of nervous e that rest cannot be obtained.

Every individual may be said to possess two fund energy. From one of these he takes daily that force the performance of his physiological functions and l is kept in reserve to meet the demands of extraor maintained, as is the reserve fund of a bank, to me abnormal. The patient who suffers from neurasth only expended his ordinary fund, but drawn so largel that he is a nervous bankrupt, and he suffers from or less serious symptoms because the various pa receive enough nervous energy to cause them to normal functions. If the strain has been very pr the patient is one whose nervous balance is no readily understood that a very serious state may de of the patient may be jeopardized if any intercurren

Symptoms.—The symptoms of neurasthenia a depending in many instances upon the organ, or or affected by the state of nervous exhaustion. In som dition of the patient suffers chiefly and the sympton irritability of temper to great mental depression and even to mental aberration, in the form of melancholia or even actual insanity. Sometimes persistent insomnia develops. In other instances the functions of the digestive tract suffer chiefly, and in others the heart displays the greatest evidence of disturbed nerve supply, so that attacks of palpitation ensue, or instead a lack of vasomotor control results in attacks of vertigo or syncope. In addition to these definite and specific symptoms the patient often complains of a host of subjective symptoms which are quite extraordinary in character. In spite of the variability of symptoms the following are constantly met with: sense of chronic fatigue, of exhaustion, and irritability. Hysteria is not infrequently associated with neurasthenia.

Diagnosis.—The physician should never reach a diagnosis of neurasthenia until by repeated examinations and study of the patient, and his secretions, he is convinced that no grave organic disease exists which may be responsible for the symptoms presented. If cardiac and renal disease are excluded, and no other organic malady can be found of sufficient gravity to produce the illness, and if the history of the patient reveals the existence of some cause capable of producing nervous exhaustion, the diagnosis of neurasthenia may be reached.

Prognosis.—The prognosis of neurasthenia depends upon the ability of the physician to remove the patient from exposure to the causes which have produced the condition, upon the ability or willingness of the patient to follow those methods of life which are conducive to the re-establishment of nervous balance and reserve energy, and upon the age and general physical state, for if the patient be one who is far advanced in years, or who by reason of disease or heredity is possessed of low recuperative power, it is manifest that complete recovery may be impossible. Given a case of neurasthenia in which all the conditions which are unfavorable may be excluded the prognosis is favorable, but the physician must be cautious in stating the duration of the period of recovery, for the progress toward health is governed not alone by the skill displayed in treatment, but by the recuperative power of the individual, a power which every physician of experience recognizes as a very variable quantity. Not rarely a seemingly frail person recovers speedily, whereas another patient of a more powerful build and physique makes progress so slowly as to cause great discouragement.

Treatment.—From what has been said of the causes of this condition it is manifest that the chief aim of the physician must be the re-establishment of the normal nervous energy or power. As first pointed out by Weir Mitchell, this can only be obtained by the accumulation of energy, and this accumulation of energy is to be had only by absolute mental and physical rest on the one hand and proper feeding on the other, the circulatory and other vital functions being maintained by passive exercises and electricity. The patient must so arrange his or her affairs that no business worries or family cares will be experienced. For this it is essential that the treatment shall be carried out in a health resort far removed from the home and office, or in a hospital or "rest-cure house," where the patient will be absolutely isolated from ordinary surroundings. An attempt to carry out the "cure" at home nearly always ends in failure, because the needed deg is not obtainable and the sounds made by the rest o patient or develop curiosity or worry as to their cau that a skilled trained nurse shall be in absolute contro any interference by members of the family. The pa sit up, but is required to remain in bed at perfect r kidneys, bowels, and skin is carefully looked after and once every day massage is given over the entire of passive exercise. In many cases it is well to give m and faradic electricity in the morning, the slowly int employed to exercise the muscles, and this in turn application of the rapidly interrupted current from t fifteen minutes. These measures combined with a early morning and an alcohol rub at bedtime, witl small quantities of food every three hours, will usua complain of being "too busy" instead of feeling, feel at the beginning of the "cure," that time hangs The patient must not receive or write letters nor this requires not only nervous but muscular strai nurse is permitted to read aloud to the patient for such a plan of treatment, in which all the nervous ene to conserve is secured, and in which every opportu addition of units of force by proper feeding, lasting is usually obtained.

TRAUMATIC NEUROSES.

Definition.—Under the term traumatic neuroses th dition in which an individual, after exposure to som or physical injury, develops a train of symptoms upon any demonstrable lesion of the nervous syst functional disorder of the nervous system in various lowing the accident, the patient presents symptoms jective, though they may be somewhat objective, a and completely incapacitated from performing the o a long period of time. Rarely the disability may these cases the question always arises as to whether addition to the functional disturbance an actual organ therefore, that cases of this character may, and do, p very difficult problems in differential diagnosis, for n disorders exist side by side with those due to true addition the functional disturbances may simulate or as to cause great confusion in symptomatology. difficulties are added the desire of the patient to from the individual or corporation responsible for becomes evident that malingering or unintentional duction of symptoms may be commonly met with.

Etiology.—The most common cause of traumatic neuroses are railroad accidents, trolley-car accidents, falls, and injuries received from falling bodies. As a result of exposure to one of these causes, with associated mental shock due to terror or horror, the nervous system develops the perversions about to be described.

Symptoms.—It is manifest from what has already been said that the symptoms may be most varied as to severity, distribution, and duration. Probably the most common statement of the patient is that he has lost power in one or more parts of his body, or he may suffer from disturbances of sensation, with or without loss of power. In males it is not infrequently claimed that the injury has resulted in a loss of sexual power, particularly if the back has received a blow or strain, even if the genital apparatus is itself entirely unaffected. In women the most common complaint is of pain or weakness in the back, of pelvic pain or displacement of the pelvic organs, and of vesical disorders. In other cases the chief claim is that more or less violent pain or tingling in the limbs is suffered. When loss of power is suffered from it appears usually as a hemiplegia or a brachial monoplegia, but if it be a hemiplegia the face nearly always escapes. Paraplegia is very rare and the sphincters of the bladder and rectum always escape.

Those paralyses which are not truly organic can be separated from those that are such by the facts that the reactions of degeneration do not develop in the paralyzed parts and the deep reflexes are usually preserved. Anæsthesia is practically always present if the paralysis of motion is complete, and it is of the type of hysterical paralysis in that it has often a sharp line of degeneration which is not coincident with the distribution of the sensory nerves of the part. Paraplegic cases do not suffer from anæsthesia of the genital organs. Again, it sometimes occurs that the symptoms complained of are not constantly in the same part or that positive suggestions may cause their development elsewhere. Not rarely an examination of the color fields of such a patient will reveal the reversals commonly found in hysteria. Disorders of all the special senses may also occur and total disappearance of these functions may take place—as complete deafness, blindness, or loss of taste or smell. Occasionally the patient may develop attacks which resemble to some degree ordinary epilepsy or catalepsy, but these attacks are separated from true epilepsy by the points already named when discussing that disease.

If we carefully exclude from any case of nervous disorder following an injury the presence of an actual organic lesion, we may unhesitatingly state that the patient is suffering from hysteria or neurasthenia due to injury, and we can treat him accordingly. On the other hand, it is not to be forgotten that the patient who suffers from the symptoms he describes is often a most miserable and unfortunate individual, as deserving of our pity as if we found him the victim of an incurable malady due to destruction of a part of his body. His functional disorders are as real to him and cause him as much suffering as if they depended upon organic causes, and a nervous system functionally perverted may be as useless as one actually grossly diseased, just as a watch which needs regulating may be as useless to its owner as one in which a spring is broken. While, therefore, it is our duty

to relieve such patients by every means in our pow
that their sufferings are often very real, we are for
condition may not be permanent, as it would be a
and so when the case has become one of medicol
not be possible to testify that the patient is incu
disabled. Not only is this true, but it is also a fact t
of litigation, and the frequent appearance of the
and experts for both sides and before a crowded
recovery impossible by still further exciting and dist
for aside from this form of excitement the descrip
accident impresses its terrors, over and over agai
horror-stricken by the original occurrence. Perfect
suffer all the symptoms they describe up to the peri
case is finished and then speedily improve.

Treatment.—The treatment varies, of course, wi
symptoms, but it may be said to be practically ide
advised in cases of hysteria and neurasthenia.

OCCUPATION NEUROSES

An occupation neurosis is a state in which th
becomes functionally disturbed by the exhaustion
supplying it, and in all probability by exhaustion
well. The causes of this exhaustion are exceedin
every pursuit in life which involves the continuou
hand and wrist may produce an occupation neuro
result we find spasm, cramp, or palsy developing
incapacitate the patient. The most common neur
is most common, and because small and accurate
is that due to writing, the so-called scriveners' p
Another form is telegraphers' cramp, and a thi
Less common forms are violinists' cramp, pianis
cramp, and "sewing spasm." Milkers and cigar
from neuroses of this character. In writers' cr
suffer chiefly, while in telegraphers' cramp the exte
involved. Various disorders of sensation in the ha
consist in sensations of tingling, tension, or numbn
or excessive dryness of the skin may be present. O
depends upon, or is associated with, a true neuriti
entire brachial plexus and cause pain in the upp
muscles of the neck and head on the affected side.
in many instances is that the patient first experience
of stiffness and lack of pliability in his fingers, w
panied by a certain lack of co-ordination in the mo
inability to move the fingers rapidly and accurate
the sufferer attempts to perform the movements wh
trouble, and almost all other motions can be gon

difficulty. If the patient now insists on keeping on with his duties, the stiffness is replaced by violent cramps, more or less painful, which come on suddenly and with considerable power. Co-ordination is still further disordered, and all attempts at a repetition of the offending act are resented by the affected centres and muscles in such a positive manner as to make all movements irregular and often jerking in character. Unless absolute rest and avoidance of former movements is permitted, the cramps, etc., are followed by loss of power, deepening into partial paralysis. Even when paralysis exists, however, it is surprising to see how many unoffending movements can be performed without discomfort and failure.

Some discussion has arisen as to whether the several symptoms which the disease presents are each in their turn an indication of a more advanced stage in the disorder or are merely more prominent in one case than another by chance or tendency on the part of the individual to any one of them. Thus, some observers have held that the first sign of the disorder was the feeling of distress or fatigue in the overworked extremity, and that the tremors followed because the warning given by the fatigue was not heeded. Finally, the disregard of this second symptom brought about the spasm or cramp, or, in other cases, the palsy. Other writers, especially those of the present day, have attempted to prove that there is no distinct onward march of the symptoms from fatigue to tremor and from tremor to palsy or cramp, but rather that the disorder is to be divided into four varieties, each one of which may assert itself without the development of another.

Thus, Lewis tells us that in some cases cramps come on, in others palsy, and in others tremors, while still another variety is separated from its fellows by the predominance of certain symptoms associated with disturbances of sensation. He states, however, that the disorder of sensation is always present in all forms of the trouble in some degree, and that it is only in cases where the trouble consists in a neuritis that the symptom rises to the importance of marking a separate variety.

Many very prominent writers on scriveners' and hammerers' palsy assert that predisposition is one of the prime factors in the causation of these maladies. While this is doubtless true to a certain extent, it is nevertheless a fact that all persons, be their temperaments nervous or otherwise, are affected, and in view of this fact the writer thinks that predisposition should not be accorded the leading position in the causation of the malady. It is, of course, probable that persons whose temperaments are nervous and excitable are naturally susceptible to nervous disorders, whereas the phlegmatic temperament is rather opposed to the conditions which are necessary for the presence of this disease.

Rosenthal calls attention to the fact that the loss of power is limited entirely to those centres which are the directors of the particular muscles involved, and states in substantiation of this assertion that the surrounding centres for other groups of muscles always escape, as is proved by the fact already mentioned, that other acts can be performed without difficulty. While it is true that the surrounding centres are not affected, it is also true that the centre governing like movements in the opposite hand is, by sympathy or other cause, affected with its fellow to a certain extent. This is proved by the fact that if the operator learns to send hand, that hand very soon follows the fate of its fel

Experiments performed by the late Dr. N. A. Ra on this subject that they may be quoted at this pc discover if exhaustion of one centre in the brain the corresponding centre on the opposite side of th he proceeded as follows: He attached a small lever the hand of the subject on the table, as when wri place the tip of his forefinger on the end of the le often as he could. Each depression was, of course Dr. Randolph found that, normally, the right for for 100 movements, the left forefinger for 75 move this primary point he proceeded to search after the n nation. He found that if the left forefinger was set forefinger had performed its 100 depressions, it b movements, and that if the right hand was set to finger had moved 75 times it could only move 75 t exhaustion of one centre produced exhaustion of tl on the opposite side of the brain.

Careful tests prove that in most instances e present, denoting a superexcitability of the spinal c evidences of neuritis of the nerve trunks have und In some cases of the disease a species of pseudomusc on, due, probably, to some centric nervous lesion, the congested condition which is nearly always pres cles. Thus we find the bellies of the muscles hard, devoid of power.

Treatment.—In the way of treatment rest is the possess for the cure of the affection; but although exciting cause is one of the essential factors for a affected arm should be used in every other motion wl so that it may not become useless from disuse. Ne therapeutic agent electricity, which is, however, cases where very slight or no inflammatory conditio the muscle, nerve, or nerve centre; and it should b use that current which causes the most contracti Galvanization of the affected muscles should be pe that the disordered nerve centres are not disturbed, a to gradually increase the exercise, so as not to exh muscles which are out of order. Movements whic with the affected parts are also useful, following the ing proposed by Fraenkel in the treatment of locomo

Finally, the administration of tonics, such as arsen is to be resorted to, and these measures combined best methods we have for effecting a cure.

RAYNAUD'S DISEASE.

Definition.—Raynaud's dsiease is a condition in which one or more of the fingers or toes, and rarely the nose and ears, suffer from a disorder of the local bloodvessels, with the result that these parts become bloodless and pallid or slate colored and mottled in appearance. The affected parts are cold and sometimes painful. The malady usually affects persons under thirty years of age, and females more commonly than males. The cause is unknown save that it seems to be of the nature of a paroxysmal neurosis involving the bloodvessels of the parts affected. Various conditions, all of them capable of causing a loss of normal nerve tone, have been considered as etiological factors, varying from diabetes and neurasthenia to fright and exposure to cold air or cold water.

Etiology.—The onset begins with a sense of tingling, or of heat or cold, in the parts which are to suffer from the well-developed state. The skin looks shrunken and ashen in hue and numbness is present to a more or less well-developed degree, but complete anæsthesia does not occur. The condition may last for a few hours or for weeks. When it disappears it nearly always returns in a short time.

When the disease occurs in its severe form local gangrene may ensue. The part becomes livid and dusky and small blebs develop on the fingers. These may dry up and recovery take place, only the skin being destroyed, or the process may become so deep that the entire part may be lost.

This condition is to be separated from senile gangrene by the youth of the patient, from frost-bite by the absence of a history of exposure to cold, and from chronic ergotism by the absence of any history of eating rye bread contaminated by ergot.

Treatment.—The treatment consists in the use of tonics and every possible measure designed to re-establish good general health. Hydrotherapy is often of value. Locally the nutrition of the affected part may be maintained to some extent by the use of dry or moist heat. Great care should be taken to protect those parts which are usually affected, from extremes of heat and cold.

ANGIONEUROTIC ŒDEMA.

Definition and Symptoms.—Angioneurotic œdema is a condition characterized by the sudden appearance, in a limited area in one or more parts of the body, of well-defined swelling due to some perversion of the normal functional activity of the vasomotor nerve supply, so that the bloodvessels of the part become dilated, and, in all probability, an extravasation of fluid takes place. The condition is to be clearly separated from that characteristic of inflammation. The temperature of the part is often lower, but sometimes it is higher than normal. The dimensions of the affected part vary greatly, but it is rarely more than a few inches in circumference. The hue of the area affected may be a deep red, as if suffering from intense congestion, or so pallid as to be cadaveric. It may be the seat of a sense of tingling, or heat, or itching, but actual pain does not occur, and pitting on pressure, to the extent that it appears in ordinary œdema, is absent.

Angioneurotic œdema occurs most commonly on the face or hands. It may affect the body and quite rarely the larynx and pharynx, when it may produce alarming symptoms by interfering with respiration. Instances of death due to this cause have been reported. The attacks last a few hours to several days, and are prone to occur at irregular intervals.

Angioneurotic œdema occurs more frequently during the third decade of life than at any other period, and in the United States affects females more frequently than males, although the reverse of this holds true in Europe. We do not know what the causative factor is, but it is known that exposure to cold and causes which diminish nervous tone bring on an attack in those who are susceptible.

In some cases the condition is induced by digestive disorders, or by the ingestion of some food which is toxic, as lobster, fish, or other animal food that is not fresh. In nearly all cases the patient is neurotic, and not rarely has a neurotic family history, or even a direct inheritance of the disorder from the parents.

Diagnosis.—Angioneurotic œdema must be separated from the local vasomotor disturbances of hysteria. This is done by the fact that in hysteria there are associated paralysis of motion or anæsthesia and, it may be, hysterical contractures. Again, the œdema of hysteria is often persistent, whereas this is temporary. From severe attacks of urticaria it is differentiated by the fact that "hives" are usually scattered widely over the body, and if they appear on the hands are characterized by multiple lesions. In most cases of hives, or urticaria, additional lesions can be produced by rubbing a part.

Prognosis. The prospect of complete cure in the sense of an escape from all future attacks is not encouraging. The general health is usually good between the attacks, and unless the part affected be the larynx the prospect of any serious result is unlikely.

Treatment.—The treatment can be directed only along those lines which will tend to improve the general health, of which the most useful are an out-door life, hydrotherapeutics, and the internal use of tonics, such as iron and arsenic if there is anæmia, and nux vomica and quinine if the nervous system is atonic. Phosphorus may also be useful. When lithæmic or gouty conditions are present, the iodides, salicylates, and colchicum may be of great value. It is needless to add that all causes known by the experience of the patient to be provocative of an attack should be sedulously avoided, for there can be little doubt that the occurrence of one attack predisposes to another.

ERYTHROMELALGIA.

Definition.—This condition was first described by Weir Mitchell in 1872. It consists in a hyperæmia of the foot and leg, rarely the hand, associated with pain which may vary in degree from a sense of weight and heaviness to exceedingly severe suffering. The malady first affects the neighborhood of the ball of the foot, and thence it spreads to the entire plantar surface.

In other cases the heel is first affected. Although exercise greatly increases the suffering, it is, as a rule, worse at night. The pain may be intermittent or continuous. The skin is often not only hyperæmic, but is often marbled or mottled in appearance. Elevation of the part, by decreasing the congestion, diminishes the pain.

Etiology.—The causes of the malady are several. In rare instances it seems to depend upon lesions in the spinal cord, in others it apparently depends upon diabetes mellitus, and in still others arteriocapillary fibrosis seems to be the underlying factor.

Diagnosis.—Before determining the diagnosis of erythromelalgia it is essential that gout and diseases of the soft and hard tissues of the foot be excluded. In the vast majority of cases the symptoms will probably be due to such causes, for true erythromelalgia is a very rare malady indeed, and but few cases have been recorded.

Treatment.—Treatment often fails to give much relief. The part should be kept in an elevated posture as much as possible, cool lotions may be applied to it, and if the patient be lithæmic the alkalies and salicylates should be given.

MIGRAINE.

Definition.—Much confusion exists as to the exact nature of the condition which is called migraine. By all authors it is used to describe a condition of severe pain, more or less limited to one side of the head, often accompanied by some disturbance of vision in one or both eyes, and by nausea and vomiting, which often do not develop until toward the end of the attack. Certain clinicians have expressed the belief that migraine is an hereditary affection, and even go so far as to regard it as a manifestation of nervous instability not far removed from epilepsy. This, however, is certainly incorrect in the vast majority of cases. It may be true that certain neurotic individuals who are subject to hysterical or epileptic manifestations often suffer from migraine. But, on the other hand, it cannot be denied that in the majority of instances the condition is a toxic neurosis due to the manufacture and retention in the body of abnormal products of metabolism. These products are chiefly the result of a disturbed action of the liver, either in the sense that the liver fails to destroy poisons which are absorbed from the intestines, or in the sense that it develops substances which it does not produce when in health. As a matter of fact migraine, as a toxic condition, is rarely the result of any disorder of function in a single organ, but is produced by several causes, an undue development of poison, a deficient action of the liver in destroying these poisons, and a torpid condition of the kidneys, whereby toxins are not speedily eliminated. It naturally follows that in high-strung, nervous individuals, and in those who have neurotic tendencies, these toxic products can readily disturb the functions of the sensory nerves of the head and so produce a seizure.

Among the active causes in provoking an attack of migraine, aside from the effects of autointoxication, there can be no doubt that nervous tire, or exhaustion, aids materially in causing an attack, particularly if in addition to such stress there is added undue sexual activity, All these factors diminish the nervous energy which and so tend to cause perversions of metabolism, an diminish the resistance of the nervous and vasculai such poisons.

The disease occurs most frequently in wome before the age of puberty. It is particularly prc have a gouty ancestry. Certain schools of ophtha urged the view that all cases of migraine are due There can be no doubt that in many instances exhaustion is a potent factor. The important po remember in studying this malady is that various ca for it; and if the patient is to be permanently reliev causes must be discovered and removed.

Symptoms.—The mode of onset of an attack varie state that for several days prior to a paroxysm they and anything but well. Often the chief sympto Other patients have no premonitory symptoms w morning in perfect health, they are seized at so with *blurring of the vision* in one or both eyes, so attack of *pain*, or pain may be the first and only may be so sudden and severe as to completely inca this form of migraine the terms "fulgurating" or applied. In most cases the pain exists chiefly in o involves the supraorbital region and the eyeball. W the entire head may suffer. The character of the p sensation in the head is tense. Not rarely *photop* some cases *vision* may be so much *interfered with* is described by the patient. In addition to the pa quently has some *vertigo*, is *mentally heavy* and dul *aphasic.*

After the attack has lasted from one to several frequently becomes *nauseated* and then *vomits.* As not contain undigested food; on the contrary, diges on with undue rapidity. The *material vomited* is and excessively *acrid* and *acid.* I am firmly convin result of an attempt on the part of the stomach materials, just as this organ eliminates oxydimorph ing. If the vomiting persists for any length of tin be brought up by reason of the drawing of bile th act of retching. It is a question whether this vom action of the poison which produces the symptoms, part due to the severity of the pain which, when it a resembles the sickening pain produced by an injury an attack the patient's *face* is usually *pallid* and having an anxious and *hunted expression* or one Sometimes the *radial pulse* is *small* and *hard,* and artery on the affected side stands out like a whipco come oftener than once a week, and sometimes much more rarely than this, unless the patient by errors in diet and by various excesses produces the provoking condition frequently. Sometimes patients state that the attack comes on in the midst of perfect health. Thus, I have heard a patient remark that she felt so well that she was sure she was going to be sick the next day, as it had been her experience that a sensation of well-being was not rarely followed by a nervous explosion. In rare cases a certain degree of *paralysis* of *the extraocular* muscles may be present during the attack. In still more rare instances the face is flushed instead of being pallid. Not rarely during the attack the *urine is scanty* and high colored, but as the attack subsides the *urine* is frequently passed in *large quantities* and is exceedingly *limpid*. Speedy recovery usually follows the vomiting of the acrid fluid already named.

Treatment.—The treatment depends upon the underlying cause of the malady. All excesses as to eating and sexual activity must be prevented. If the patient is run down and neurasthenic, a vacation or a rest cure is essential. If the kidneys fail to excrete a sufficient quantity of urinary solids per day, the various potassium salts, such as the acetate, citrate, or bitartrate of potassium, must be given in 5 or 10 grain doses three or four times a day, in copious draughts of water to increase urinary elimination. If there are any evidences that the liver is persistently or occasionally inactive, its function should be stimulated by the use of calomel, blue mass, or podophyllin. Many of these cases do very well if 5 to 10 grains of blue mass are taken every week or ten days, and then followed by a saline purge. In those instances in which the patient leads a sedentary life, active out-door exercise to ensure perfect oxidation processes in the body are essential. In those patients who suffer from gastrointestinal catarrh, a dose of Hunyadi or Apenta water, taken hot and in sips, before breakfast, will often be efficient not only in moving the bowels, but preventing the attacks. Often diluting one of these waters one-half with hot water makes it an efficient purgative. The use of salol in the dose of 5 to 10 grains a day as an intestinal antiseptic is often advantageous.

Of all forms of preventive treatment, that which is devoted to the increased activiy of the intestines, the liver, and the kidneys is of most importance. If the patient is gouty, or suffers from that condition commonly but erroneously called "uricacidæmia," not only should the treatment just recommended be employed, but the use of other more active salicylates, such as the salicylate of strontium in 5 or 10 grain doses three times a day, are advisable. If errors in refraction exist, carefully fitted glasses should be provided, and if the nasal mucous membrane is hypertrophied or other abnormalities exist in this region, they should be treated. For the relief of the attack many measures have been suggested. In those instances where the patient has prodromal symptoms, a brisk saline cathartic, such as Seidlitz powder, citrate of magnesia, or Rochelle salt, should be given, with the idea of sweeping out from the bowels poisonous material. This may be followed in half an hour to an hour by 2 grains of caffeine with 10 grains of bromide of sodium. The best way to give this is in granular effervescent salts. In some instances a small dose of phenacetin or acetanilid should be added. If high arterial tension is present, nitroglycerin is valuable. For the relief of the pain when it is very severe phenacetin, antipyrin, and acetanilid are useful, but it must always be remembered that the stomach is, as a rule, excreting rather than absorbing, and that the mere administration of a palliative at this time may be fruitless for this reason. Under these circumstances it may be necessary to give these drugs by the rectum, or to empty the stomach by vomiting or by the use of the stomach tube before they are administered. Sometimes the stomach can be stimulated to absorption by $\frac{1}{40}$ grain of strychnine. In certain cases the use of a full dose, 10, 15, or 20 drops, of the tincture of gelsemium with a grain of an active extract of cannabis indica gives the greatest relief. The use of cologne-water containing 5 to 10 grains of menthol to the ounce applied over the course of the painful nerve may give much relief. In many instances it is impossible for any of these remedies to do good unless the patient will lie down in a quiet and dark room for several hours.

SUNSTROKE.

Definition.—Sunstroke, more accurately called heatstroke, insolation, or thermic fever, and by the French *coup de soleil,* is a condition of the body produced by exposure to great heat. In rare instances the temperature of the patient does not rise, but falls, and to this condition is given the name heat exhaustion.

Etiology.—The chief factor in producing heatstroke is the presence of great heat associated, as a rule, with marked humidity of the atmosphere. It is important to bear in mind the fact that exposure to the rays of the sun is not necessary for the development of heatstroke. Cases are constantly met with in which the illness of the patient is due to artificial heat, and heatstroke may occur in the night as well as in the day if the atmosphere is hot and moist. Dry heat is better borne by all persons than is moist heat, probably because evaporation on the skin proceeds rapidly in dry air, and so the body is cooled by the function of perspiration, whereas in a moist atmosphere the imperfect evaporation results in an accumulation of heat in the body. For this reason heatstroke is very rare on the western plains of the United States, where the temperature in summer often reaches 105° in the shade, whereas in Philadelphia, where the air is humid, heatstroke is exceedingly common when the thermometer registers a temperature of 90°. In the one case evaporation is so rapid that the heat of the body is kept at a normal level, whereas in the latter case the perspiration lies on the skin in great beads. A second factor in producing heatstroke is the use of alcoholic drinks in any form. There can be no doubt that all such beverages greatly predispose to the development of this state. So, too, renal disease and a feeble heart may act as predisposing factors. Loss of sleep and torpidity of the bowels are also possessed of an evil influence.

Certain French clinicians have asserted, with notable facts in support of their views, that sunstroke is really a form of infection which develops under the atmospheric states already named.

Pathology and Morbid Anatomy.—While a very considerable number of clinicians in America and in the East Indies described sunstroke symptomatically in the early part of the last century, it was not till H. C. Wood collated our knowledge and enriched it by further experimentation that the profession began to fully grasp the facts concerning its production and the lesions which ensued.

The pathology of the disorder resides in the inability of the heat-regulating mechanism of the body to maintain a normal body temperature. The primary difficulty lies in a decreased power of the body to carry out an efficient heat dissipation, and this is followed by an unrestrained heat production, due, in Wood's opinion, to failure of the inhibitory heat centres, in the pons, to check oxidation processes. With diminished heat dissipation and increased heat production it is not difficult to perceive why the temperature of the body rises until a state of hyperpyrexia is reached.

The morbid anatomy consists in changes in the tissues which in turn permit decomposition to set in very rapidly, being preceded by well-marked rigor mortis. The veins of the brain and lungs are found distended with fluid blood, and everywhere the blood fails to clot as it does in the vessels of the ordinary cadaver. If an autopsy is made very soon after death, the left ventricle is found in firm systole, but the right ventricle is distended with blood. The liver and kidneys are also found to be intensely engorged.

Symptoms.—The symptoms of sunstroke consist, in the preliminary stage, in *oppression and dizziness.* If these evidences of heat are ignored, the stage of *sudden unconsciousness* develops, and is often ushered in by a *convulsion* which may be exceedingly violent. In other cases no convulsion develops, but *deep stupor* with stertorous breathing comes on. The *face* is at first *livid* and later *deeply cyanotic*, the great vessels of the neck and upper extremities being distended. The *temperature* of the patient speedily rises to a height never seen in any other disease, sometimes reaching 112° or more, the average being from 105° to 110°. The *pupils* may be *contracted* or *widely dilated.* If the fever cannot be reduced and the cardiac and pulmonary congestion are not relieved, death ensues within twelve to thirty-six hours. When improvement takes place, a relapse some hours later often ensues. A patient who has sunstroke may subsequently become very ill and die from a secondary meningitis. Persons who have had sunstroke are very susceptible to high temperatures, and when exposed in after years may be greatly distressed by an atmospheric temperature as low as 80°, if the air is moist.

Diagnosis.—There are only two other states that resemble heatstroke, namely, uræmia and apoplexy. The first can be excluded by the absence of hyperpyrexia and albumin in the urine. The second is excluded by the same lack of temperature, except in those cases in which the pons is involved, when the fever may be high, but pontile hemorrhage is usually speedily fatal and the paralysis severe. Sunstroke and uræmia may, however, exist simultaneously. The history of the patient will exclude epilepsy which is also excluded by the high fever.

Prognosis.—The prognosis depends on the height of the fever and the resistance which it offers to treatment. Do what we will a large number of these cases die.

Treatment.—The treatment of sunstroke, if it is to
factory results, must be bold and vigorous. In most
essential: First, that the temperature must be reduc
level, by the application of cold water or ice. This
stripping the patient, laying him upon a canvas cot,
stream of cold water upon his body from a hose, the
and vigorously rubbed at the same time by one or mo
object of producing reaction, of overcoming internal
the blood to the surface, whereby it may be cooled,
dissipation of heat, for frictions increase the dissipati
application of cold nearly fifty per cent. During thi
be applied to the head constantly. In other instanc
immersed in a tub of cold water, and if necessary piec
in this water. If the tub is used, active frictions are
case just stated. Care should be taken that the tem
begins to fall, does not drop too rapidly, so that the pa
thermia and collapse. If the patient is robust and
venous engorgement, free venesection should be practi
of large experience believe that venesection is of alm
with the use of cold. Venesection should be foll
clysis or by the intravenous injection of normal salt s
measures engorgement of the right side of the hea
toxæmia combated. If the circulation on the left si
failing, hypodermic injections of Hoffmann's anodyn
be administered. The use of alcohol should be avoid
confined, citrate of magnesia should be given in full p
them, and where the patient is unconscious and unab
a dose, $\frac{1}{6}$ of a grain of elaterium may be used not onl
but diminish cerebral congestion. The violent headac
sunstroke may, in some instances, yield to the ordi
combined with the use of bromide of sodium and ca
not do so, and there are any evidences of meningeal
free venesection should be practised, not only for the
order to prevent the development of secondary mening
of very great importance, but is often treated as of litt

In the after-treatment of the patient it is essential
should be carefully watched, as it nearly always has
second time. Such a tendency should be combated
cold to the head, and by cold bathing if actual hyperp
feet rest in bed for a number of days after the sunstr
upon, and the patient should be warned that any expo
days will be liable to produce another attack.

HEAT EXHAUSTION.

Heat exhaustion is a condition produced by the sam
but instead of hyperpyrexia developing the temperatur
the patient's skin may be bedewed with a cold sweat,

of severe collapse may be present. This condition is to be treated by immersing the patient in hot water, and by the application about his body, after the removal from the bath, of hot bottles or hot bricks to maintain body temperature. A failing circulation should be supported by hypodermic injections of Hoffmann's anodyne and atropine. Care should be taken that coldness of the extremities is not mistaken for true heat exhaustion, for it sometimes happens that the extremities are cold in thermic fever, although the temperature of the body may be far above normal. This point must be determined by taking the rectal temperature. If the rectal temperature is found to be very high, the treatment for heatstroke should be instituted and the circulation equalized by active rubbing. A hot bath in such a case is not advisable.

FACIAL HEMIATROPHY.

This is a condition in which one side of the face undergoes a slowly progressive wasting. As a rule it begins between the ages of ten and twenty years. The cause is unknown, although it is without doubt due to some localized degenerative change in the nervous system. In an autopsy upon a case of this character Mindel found degeneration of the trifacial nerve in its efferent fibres and atrophy of the substantia nigra.

When the malady first develops, the skin of the affected part begins to be thin and glossy and seems to be stretched. The fine hairs fall out and the sebaceous glands atrophy, so that the part is unduly dry. After that the subcutaneous tissues atrophy so that the natural fulness of the face is diminished, and, in the later stages of the affection, even the underlying bone may be atrophied or absorbed. The muscular tissues escape the atrophy to a greater extent, and do not undergo degenerative changes. The eye may become sunken from wasting of the orbital part, and the pupil may be in a state of mydriasis. Usually the condition is painless, but local spasm of the muscles of the part may occur. No treatment is of any avail in arresting the progress of the disease.

PERIODICAL PARALYSIS.

This term is applied to an extraordinary condition of paralysis involving widely distributed groups of muscles in the arms, legs, and trunk, which develops rapidly in apparently healthy individuals without any apparent exciting cause. Not rarely several members of a family are affected by the malady. The patient may go to bed in perfect health and wake to find himself paralyzed, or the paralysis develops after a preliminary sense of weakness in the affected parts. As a rule, the legs suffer chiefly. Very rarely the muscles of the neck are affected, but the cranial nerves always escape. The reflexes are minus, and the muscles and nerve trunks lose their reaction to faradic stimulation. The paralysis lasts from a few hours to a day, and speedy and perfect recovery ensues, but relapses frequently take place

The condition is apparently a form of autointoxication, and is said to be benefited by the use of alkaline diuretics.

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